2000 ANNUAL MEETING PROGRAM

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DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

SATURDAY, APRIL 29, 2000 12:00 NOON - 6:00 P.M.

METRO TORONTO CONVENTION CENTRE

ROOM 201

OBJECTIVE

The Academic Surgeon's Symposium is designed to help develop the Academic Cardiothoracic Surgeon. This is a continuing effort by the American Association for Thoracic Surgery to provide a specific educational conference for potential and active academic cardiothoracic surgeons. The present symposium will focus on several areas including building a clinical program, developing new technology, getting published, 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 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

PROGRAM

12:00 p.m. LUNCH Room 204

Introduction and Welcome

Chairs: Edward D. Verrier, M.D.

Irving L. Kron, M.D.

1:00 p.m. GETTING PUBLISHED

Andrew S. Wechsler, M.D.

Hahnemann University

Philadelphia, Pennsylvania

1:30 p.m. BUILDING A CLINICAL PROGRAM - MULTI-INSTITUTIONAL

Vaughn A. Starnes, M.D.

University of Southern California

Los Angeles, California

2:00 p.m. BUILDING A CLINICAL PROGRAM - IN A SINGLE INSTITUTION

William A. Baumgartner, M.D.

Johns Hopkins Hospital

Baltimore, Maryland

2:30 p.m. GETTING PROMOTED

Irving L. Kron, M.D.

University of Virginia Health Sciences Center

Charlottesville, Virginia

3:00 p.m. BREAK

3:15 p.m. BECOMING A DIVISION HEAD

Edward D. Verrier, M.D.

University of Washington

Seattle, Washington

3:45 p.m. MANAGING A MEDICAL CENTER

Floyd D. Loop, M.D.

Cleveland Clinic Foundation

Cleveland, Ohio

4:15 p.m. DEVELOPING NEW TECHNOLOGY

Delos M. Cosgrove, M.D.

Cleveland Clinic Foundation

Cleveland, Ohio

4:45 p.m. INFLUENCING THE POLITICAL PROCESS

Timothy J. Gardner, M.D.

Hospital of the University of Pennsylvania

Philadelphia, Pennsylvania

5:15 p.m. RECEPITON Room 204

CONGENITAL HEART DISEASE

SYMPOSIUM

SUNDAY, APRIL 30, 2000 8:00 A.M. - 5:00 P.M.

METRO TORONTO CONVENTION CENTRE

ROOM 201

OBJECTIVE

The 2000 AATS Congenital Heart Disease Symposium will be divided into four sessions, each one addressing different aspects of complex congenital heart surgery. The first session will address the operative options for management of patients with single ventricle requiring a Fontan procedure. The presenters will be describing their surgical techniques including a video presentation and summary of results. Management of specific problems with single ventricle patients including arrhythmias and the Fontan procedure in adult patients will also be covered.

The second session will be devoted entirely to "How I Do It" video presentations of corrective surgery for various complex anatomic defects. These will include tetralogy of Fallot, total anomalous pulmonary venous connection, aortic arch repair in conjunction with intra-cardiac procedures, and LV outflow reconstruction preserving the native aortic valve. Presentations will focus on technical aspects of the operations as well as surgical results.

Session three will discuss in-depth management options for patients with hypoplastic left heart syndrome variants where there is potential for a two-ventricle repair. Surgical techniques, decision making and management will be discussed as well as surgical results. A discussion session will be held at the end of the presentations that will cover controversial topics. The three presentations will be: Two-ventricle repair of hypoplastic left ventricle, Two-ventricle repair of unbalanced AV canal defects and Results with staged palliation for hypoplastic left heart syndrome variants. The final session will discuss new techniques including the use of continious bypass for repair of hypoplastic left heart syndrome and physiologic parameters utilized to monitor organ perfusion during regional cardiopulmonary bypass. The second presentation will cover the rapidly evolving area of tissue engineering with the creation of valved conduits from patient's autologous tissue to permit growth and prevent rejection. At the completion of the symposium the participants should have an enhanced understanding of management of patients with single ventricle and current surgical options as well as specific techniques for repair of complex congenital heart defects. An in-depth understanding of hypoplastic left heart syndrome variants and their management should also be gained from session three.

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 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

PROGRAM

7:00 a.m. REGISTRATION AND CONTINENTAL BREAKFAST

8:00 a.m. INTRODUCTION AND WELCOME

Pedro del Nido, M.D., Chairman

SESSION I OPERATIVE VARIATIONS OF FONTAN'S PROCEDURE (VIDEO Presentations)

Moderator: William G. Williams, M.D.

8:05 a.m. LATERAL TUNNEL CAVO-PULMONARY CONNECTION

John E. Mayer, M.D.

Children's Hospital

Boston, Massachusetts

8:35 a.m. INTRA-TO-EXTRA CARDIAC CONDUIT

Marc R. de Leval, M.D.

Great Ormond Hospital for Children

London, England

9:05 a.m. EXTRACARDIAC CONDUIT (WITHOUT BYPASS)

Frank L. Hanley M.D.

University of California at San Francisco

San Francisco, California

9:25 a.m. PANEL DISCUSSION

9:55 a.m. BREAK

10:20 a.m. ARRHYTHMIA SURGERY AND THE FONTAN CONVERSION

Constantine Mavroudis, M.D.

Northwestern University Medical School

Chicago, Illinois

10:50 a.m. FONTAN PROCEDURE IN THE ADULT

William G. Williams, M.D.

University of Toronto

Toronto, ON, Canada

SESSION II SURGICAL TECHNIQUES

(Video Presentations)

Moderator. Roger B.B. Mee, FRACS

11:20 a.m. TRANSATRIAL-TRANSPULMONARY REPAIR OF TETRALOGY OF FALLOT

Roger B. B. Mee, FRACS

Cleveland Clinic Foundation

Cleveland, Ohio

11:50 a.m. TOTAL ANOMALOUS PULMONARY VENOUS RETURN

Thomas L. Spray, M.D.

Children's Hospital of Philadelphia

Philadelphia, Pennsylvania

12:10 p.m. Panel Discussion

12:20 p.m. LUNCHEON- Exhibit Hall C

SESSION II SURGICAL TECHNIQUES (Cont.)

(Video Presentations)

1:30 p.m. AORTIC ARCH REPAIR IN CONJUNCTION WITH INTRA CARDIAC PROCEDURES

Edward L. Bove, M.D.

University of Michigan Hospital

Ann Arbor, Michigan

2:00 p.m. MODIFIED KONNO PROCEDURE FOR LV OUTFLOW OBSTRUCTION

Richard A. Jonas, M.D.

Children's Hospital

Boston, Massachusetts

2:20 p.m. Panel Discussion

SESSION III MANAGEMENT OF HLHS VARIANTS

*Moderator: Pedro J. del Nido,*M.D.

2:30 p.m. TWO-VENTRICLE REPAIR OF HYPOPLASTIC LEFT HEART COMPLEX

Christo I. Tchervenkov, M.D.

The Montreal Children's Hospital

Montreal, Quebec, Canada

2:45 p.m. INDUCTION OF LEFT VENTRICLE GROWTH AND TWO-VENTRICLE REPAIRS

John E. Foker, M.D.

University Hospitals

Minneapolis, Minnesota

3:00 p.m. STAGED PALLIATION OF HLHS VARIANTS

Pedro J. del Nido, M.D.

Children's Hospital

Boston, Massachusetts

3:15 p.m. DISCUSSION

3:30 p.m. BREAK

SESSION IV NEW TECHNIQUES

Moderator: Pedro J. del Nido, M.D.

3:50 p.m. NEO NATAL AORTIC ARCH RECONSTRUCTION: ALTERNATIVES TO CIRCULATORY ARREST

Frank A. Pigula,M.D.

Children's Hospital of Pittsburgh

Pittsburgh, Pennsylvania

4:10 p.m. TISSUE ENGINEERED OF VALVED CONDUIT

John E. Mayer, M.D.

Children's Hospital

Boston, Massachusetts

5:00 p.m. RECEPTION- EXHIBIT HALL

GENERAL THORACIC SURGERY

SYMPOSIUM

SPONSORED IN COOPERATION WITH THE GENERAL

THORACIC SURGICAL CLUB

SUNDAY, APRIL 30, 2000 8:00 A.M. - 5:45 P.M.

METRO TORONTO CONVENTION CENTRE

ROOM 205

OBJECTIVE

The 2000 General Thoracic Surgical Symposium will provide an in-depth review of four common problems: the solitary pulmonary nodule, stage I bronchogenic carcinoma, pleural collections and achalasia. These are familiar clinical entities and the management of these diseases is fundamental to the practice of general thoracic surgery. However, a better understanding of disease pathophysiology, the introduction of new screening and diagnostic technologies and the development of innovative treatment modalities have provided new options in detection, diagnosis and treatment.

The symposium consists of four moderated sessions. The format groups speakers to emphasize options and variations. The panel discussion allows debate which will provide a current consensus in the diagnosis and management of these common clinical problems. Audience participation is a vital component of the discussion period and the symposium.

ACCREDITATION

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PROGRAM

7:00 a.m REGISTRATION & CONTINENTAL BREAKFAST

8:00 a.m. INTRODUCTION

Chairman: Thomas W. Rice, M.D.

SESSION I PULMONARY NODULE

Moderator: Douglas J. Mathisen, M.D.

Boston, Massachusetts

8:10 a.m. SCREENING AND EVALUATION

Carolyn E. Reed, M.D.

Medical University of South Carolina

Charleston, South Carolina

8:40 a.m. INFECTIOUS AND INFLAMMATORY NODULES

Mark S. Allen, M.D.

Mayo Clinic

Rochester, Minnesota

9:10 a.m. NEOPLASTIC NODULES

G. Alexander Patterson, M.D.

Washington University

St. Louis, Missouri

9:40 a.m. DIAGNOSIS AND TREATMENT

Joel D. Cooper, M.D.

Washington University

St. Louis, Missouri

Malcolm DeCamp, M.D.

Cleveland Clinic Foundation

Cleveland, Ohio

10:10 a.m. PANEL DISCUSSION

10:30 a.m. BREAK

SESSION II ACHALASIA

Moderator: F. Griffith Pearson, M.D.

Toronto ON, Canada

11:00 a.m. THE PATHOLOGY OF ACHALASIA

John R. Goldblum, M.D.

Cleveland Clinic Foundation

Cleveland, Ohio

11:20 a.m. ACHALASIA: DIAGNOSIS AND THERAPY

Joel E. Fichter, M.D.

Cleveland Clinic Foundation

Cleveland, Ohio

11:40 a.m. LAPAROSCOPIC HELLER MYOTOMY

Claude Deschamps, M.D.

Mayo Clinic

Rochester, Minnesota

12:00 p.m. THE END-STAGE ESOPHAGUS: DEFINITION AND TREATMENT

Mark B. Orringer, M.D.

University of Michigan Medical Center

Arm Arbor, Michigan

Thomas W. Rice, M.D.

Cleveland Clinic Foundation

Cleveland, Ohio

12:30 p.m. PANEL DISCUSSION

1:00 p.m. LUNCH - Exhibit Hall C

SESSION III THE PLEURA

Moderator: Douglas E. Wood, M.D.

Seattle, Washington

2:00 p.m. MALIGNANT PLEURAL EFFUSION

Joe B. Putnam, Jr., M.D.*

University of Texas MD

Anderson Cancer Center

Houston, Texas

Mark J. Krasna, M.D.

University of Maryland

Baltimore, Maryland

2:20 p.m. ACUTE EMPYEMA

Stephen R. Hazelrigg, M.D.

Southern Illinois University School of Medicine

Springfield, Illinois

Michael Jaklitsch, M.D.

Brigham & Women's Hospital

Boston, Massachusetts

2:40 p.m. SPONTANEOUS PNEUMOTHORAX

Keith S. Naunheim M.D.

St. Louis University Medical Center

St. Louis, Missouri

Darroch W. O. Moores, M.D.

Albany Cardiothoracic Surgeons

Albany, New York

**Author has a relationship with Denver Biomaterials & Life Stream, Inc.*

3:00 p.m. CHYLOTHORAX

Daniel Miller, M.D.

Mayo Clinic

Rochester, Minnesota

3:15 p.m. PANEL DISCUSSION

4:00 p.m. BREAK

SESSION IV STAGE I NON-SMALL CELL LUNG CANCER

Moderator: Richard Feines, M.D.

Rochester, New York

4:30 p.m. OPEN RESECTION

L. Penfield Faber, M.D.

Rush - Presbytarian - St. Luke's Medical Center

Chicago, Illinois

4:45 p.m. VATS RESECTION

Scott J. Swanson, M.D.

Brigham & Women's Hospital

Boston, Massachusetts

5:00 p.m. PREOPERATIVE THERAPY

John Roberts, M.D.**

Vanderbilt University Hospital

Nashville, Tennessee

5:15 p.m. POSTOPERATIVE THERAPY

Robert J. Ginsberg

Memorial-Sloan Kettering Cancer Center

New York, New York

5:30 p.m. PANEL DISCUSSION

5:45 p.m. ADJOURN - RECEPTION EXHIBIT HALL

***Author has a relationship with Bristol-Myers*

ADULT CARDIAC SURGERY

SYMPOSIUM

SUNDAY, APRIL 30, 2000 8:00 A.M. - 5:00 P.M.

METRO TORONTO CONVENTION CENTRE CONSTITUTION HALL

OBJECTIVE

The 2000 Adult Cardiac Surgical Symposium will focus in depth on two problems that are fundamental to cardiac surgery: aortic valve replacement and myocardial revascularization. The morning session is video-based and is entirely devoted to the technical aspects of various types of aortic valve replacement and strategies including standard mechanical and bioprostheses, stentless valves, homografts, and pulmonic valve autotransplantation. This approach represents a departure from previous years but emphasizes the importance of technical surgery and will provide participants with a concentrated exposure to aortic valve replacement by multiple experienced surgeons.
The afternoon session focuses on myocardial revascularization and includes both technical and data-related segments. The emphasis is on arterial grafting and off-pump surgery, as well as new concepts of myocardial revascularization including percutaneous intervention, robotic bypass grafting, laser revascularization, and gene therapy.

At the end of the symposium the participants should understand the technical aspects of all commonly performed operations for aortic valve replacement. They will understand the fundamental principles we have learned about myocardial revascularization during the bypass surgery era, technical aspects of recent innovations in bypass surgery and alternative invasive therapies.

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 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

PROGRAM

7:00 am. REGISTRATION AND CONTINENTAL BREAKEFAST

8:00 a.m. INTRODUCTION

Bruce W. Lytle, M.D., Chairman

SESSION I TECHNICAL SYMPOSIUM:

AORTIC VALVE REPLACEMENT

8:05 a.m. AVR WITH HOMOGRAFT TECHNIQUE OF ROOT INCISION

Tirone E. David, M.D.

Toronto General Hospital

Toronto, Ontario, Canada

8:20 a.m. AVR WITH TILTING DISC PROSTHESIS

Gary W. Akins, M.D.**

Massachusetts General Hospital

Boston, Massachusetts

8:35 a.m. AVR WITH BILEAFLET PROSTHESIS

Joseph M. Graver, M.D.*

Emory University School of Medicine.

Atlanta, Georgia

8:50 a.m. MINIMALLY INVASIVE AVR

Michael K. Banbury, M.D.

The Cleveland Clinic Foundation

Cleveland, Ohio

9:05 a.m. AVR WITH BIOPROSTHESIS AND ROOT ENLARGEMENT

Hartzell V. Schaff, M.D.

Mayo Clinic

Rochester, Minnesota

9:20 a.m. PARTIAL AORTIC ROOT AVR WITH FREESTYLE STENTLESS PORCINE VALVE

Sary F. Aranki, M.D.

Brigham & Women's Hospital

Boston, Massachsuetts

9:35 a.m. PANEL DISCUSSION

10:00 a.m. BREAK

10:20 a.m. THE TORONTO SPV™ IMPLANTATION TECHNIQUE

Christopher M. Feindel, M.D.

The Toronto Hospital

Toronto, Ontario, Canada

**Author has a relationship with Silver Carbomedics, Inc.*

***Author has a relationship with Medtronics, Inc.*

10:35 a.m. SUBCORONARY INSERTION OF AORTIC HOMOGRAFT

Donald B. Doty, M.D.*

LDS Hospital

Salt Lake City, Utah

10:50 a.m. HOMOGRAFT AORTIC ROOT REPLACEMENT FOR ADVANCED AORTIC VALVE ENDOCARDITIS: A VIDEO PRESENTATION OF SURGICAL TECHNIQUE

Gosta B. Pettersson, M.D.

The Cleveland Clinic Foundation

Cleveland, Ohio

11:05 a.m. AVR WITH A PULMONARY AUTOGRAFT

Nicholas T. Kouchoukos, M.D.
Missouri Baptist Medical

Center St. Louis, Missouri

11:20 a.m. PANEL DISCUSSION

11:45 a.m. LUNCH - Exhibit Hall C

1:00 p.m. ADVANCES IN INTERVENTIONAL CARDIOLOGY

Stephen Ellis, M.D.**

The Cleveland Clinic Foundation

Cleveland, Ohio

1:20 p.m. RADIAL ARTERY BYPASS GRAFTING

Richard F. Brodman, M.D.

New York Hospital Cornell Medical Center

New York, New York

1:35 p.m. PATENCY RATES OF ARTERIAL BYPASS GRAFTS

Hendrick B. Earner, M.D.

Washington University School of Medicine

St. Louis, Missouri

1:55 p.m. COMPOSITE ITA GRAFTING

Alfred J. lector, M.D.

Midwest Heart Surgery Institute

Milwaukee, Wisconsin

2:15 p.m. WHAT WE KNOW ABOUT CORONARY BYPASS GRAFTING
Bruce W. Lytle, M.D.

The Cleveland Clinic Foundation

Cleveland, Ohio

2:40 p.m. PANEL DISCUSSION

3:00 p.m. BREAK

**Author has a relationship with Cryolife, Inc.*

**Author has a relationship with Cardio/Johnson &Johnson, Boston Scientific/Scimed, Centeon & Eli Lilly*

3:15 p.m. OPERATIVE TECHNIQUES FOR BEATING HEART

CORONARY ARTERY BYPASS SURGERY WITH SUCTION STABILIZATION

Michael J. Mack, M.D.

Medical City Dallas Hospital

Dallas, Texas

3:30 p.m. EXPOSURE OF POSTERIOR CIRCULATION WITH PRESSURE STABILIZATION: SKELETONIZATION OF ITA GRAFTS

Antonio M. Calafiore M.D.

University G. D'Annunzio

Chieti, Italy

3:50 P.M. ROBOTICALLY ASSISTED CORONARY ARTERY BYPASS GRAFtTNG

Ralph J. Damiano, Jr., M.D.*

Hershey Medical Center

Hershey, Pennsylvania

4:05 p.m. TRANSMYOCARDIAL REVASCULARIZATION

Craig Richey Smith, M.D.**

Columbia Presbyterian Medical Center

New York, New York

4:20 p.m. GENE THERAPY FOR CORONARY ARTERY DISEASE

Todd K. Rosengart, M.D.***

Evanston Hospital, Northwestern Univ. Medical School

Evanston, Illinois

4:35 p.m. PANEL DISCUSSION

5:00 p.m. ADJOURN - RECEPTION EXHIBIT HALL

**Author has a relationship with ComputerMotion.*

***Author has a relationship with Eclipse, Inc.*

****Author has a relationship with Genvec.*

MONDAY MORNING, MAY 1, 2000

Back to Annual Meeting Program

80TH ANNUAL MEETING

Metro Toronto Convention Centre
Toronto, Ontario, Canada, April 30-May 3, 2000

PROGRAM OUTLINE

MONDAY, MAY 1, 2000

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

8:15 a.m. SCIENTIFIC SESSION

(10 *minute presentation, 10 minutes discussion)*

Constitution Hall, Metro Toronto Convention Centre

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

Tirone E. David, M.D.

1. Perioperative Complications After Living Donor Lobectomy

Richard J. Battafarano*, Richard C. Anderson*, Brian Meyers*, Tracey J. Guthrie*, Dan Schuller*, Joel D. Cooper and G. Alexander Patterson, St. Louis, Missouri

Discussant: Vaughn A. Starnes, M.D.

OBJECTIVE: Clinical lung transplantation has been limited by the availability of suitable cadaveric lungs. Living donor lobectomy provides right and left lower lobes from a pair of living donors for each recipient. We reviewed our experience with living donor lobectomy from July 1994 to August 1999.

METHODS: Fifty-four donor lobectomies were performed. The hospital records of these 54 donors were retrospectively analyzed to examine the incidence of perioperative complications.

RESULTS: Median hospital stay for all donors was 5.0 days (range 2.7-12.4). Twenty-one of the 54 donors (38.9%) had no perioperative complications. Thirty-three of the 54 donors (61.1%) experienced postoperative complications. Nine major complications occurred in 8 patients and included bronchial stump fistula (3), pleural effusion requiring drainage (2), bilobectomy (1), hemorrhage requiring red cell transfusion (1), permanent phrenic nerve injury (1), and atrial flutter ultimately requiring electrophysiologic ablation (1). These 33 donors experienced 46 minor complications including pneumonia (8), pericarditis (7), dysrhythmia (6), persistent air leak (6), transient hypotension requiring fluid resuscitation (4), atelectasis (3), subcutaneous emphysema (3), urinary tract infection (2), loculated pleural effusion (2), ileus (2), C.difficile colitis (1), rupture

of saline breast implant (1), and severe contact dermatitis secondary to adhesive tape (1). There were no post-operative deaths and only 1 donor required surgical re-exploration.

CONCLUSIONS: Donor lobectomy can be performed with low mortality and remains an important alternative for potential recipients unable to wait for cadaveric lung allografts. However, morbidity is high and must be considered when counseling potential living donors.

**By Invitation*

2. The Relationship of Hospital Size and Case Volume to the Cost of Coronary Artery Bypass Surgery

David M. Shahian, Gerald J. Heatley* and George A. Westcott*, Burlington, Massachusetts

Discussant: Timothy J. Gardner, M.D.

OBJECTIVE: This study challenges the concept that higher volume heart surgery programs are inherently more cost effective.

METHODS: Retrospective administrative and cost data were obtained for all 12,774 patients who underwent isolated CABG at 12 Massachusetts hospitals during 1995 and 1996. Hospital acute care beds ranged from 220 to 862 (mean 434)) and total (DRG 106 + 107) annual CABG cases per hospital varied from 271 - 913 (mean = 532). Bivariate and multivariate analyses were employed to study the relationship between the DRG-spe-cific direct cost and a number of patient (age, gender, acuity class, payer) and hospital (number of beds, annual DRG-specific case volume, cardiothoracic residency) predictor variables. For each hospital, we also studied the relationship between changes in CABG case volume and the corresponding changes in average cost from 1995 to 1996.

RESULTS: Scatterplots revealed a broad range of mean direct CABG cost among hospitals with comparable case volumes. When hospital beds and annual cases were analyzed as disaggregate continuous variables, there was no linear relationship with CABG direct costs ($r = -0.05$ to $+0.07$). When hospital size was grouped into strata and analyzed by ANOVA, the smallest hospitals had the lowest costs ($p = 0.0001$). The relationship between case volume strata and costs showed no consistent pattern. In multivariate analysis, higher patient acuity class was the most important predictor of cost for each DRG and year (partial $R^2 = 0.15 - 0.21$). Beds and case volume met inclusion criteria for each model but added little to the "explanation" of variability R^2, often less than 1%. Finally, there were substantial inter-hospital differences in the magnitude and direction (direct versus inverse) of their 1995-1996 A volume versus A cost.

CONCLUSIONS: Within the range of hospital size and case volume represented in this study, there is no evidence that either variable is related to the cost of performing CABG. Massachusetts hospitals appear to function on different segments of different average cost curves, probably related to variations in quality, patient flow, process efficiency, standardization, and capacity.

**By Invitation*

3. Independent Factors Associated with Longevity of Prosthetic Pulmonar Valves and Valved Conduits

Christopher A. Caldarone*, Brian W. McCrindle*, Glen S. Van Arsdell*, John G. Coles, Gary Webb*, Robert M. Freedom* and William G. Williams, Iowa City, Iowa; Toronto, ON, Canada

Discussant: Richard A. Jonas, M.D.

OBJECTIVE: Because most studies identifying predictors of pulmonary valve prosthesis failure (i.e. reoperation) are limited to a single valve type and vary according to patient age, inter-study comparison requires an assumption that differences in age are not important. To evaluate the age-dependence of variables predictive of prosthesis replacement, the following analysis was conducted:

METHODS: Retrospective analysis of 945 operations in 727 patients undergoing placement of pulmonary valve prostheses was performed. After age was identified as a strong independent predictor of valve failure, the database was stratified into age-based subsets. Predictors of valve replacement were identified in each subset.

RESULTS: Freedom from valve replacement at 5 years was 81%. For the entire cohort, significant independent factors associated with decreased time to valve replacement included: younger age (Hazard ratio: 0.71/log-years), diagnosis (Hazard ratios: Tetralogy=reference, Pulmonary atresia/ VSD 2.19, Truncus 1.76, D-transposition 2.60, L-transposition 2.33, tetralogy w/ absent pulmonary valve 1.74, Double outlet right ventricle 3.57, Pulmonary stenosis 1.03, Pulmonary atresia/intact septum 1.90), type of prosthesis (Hazard ratios: Pulmonary homograft conduit=reference, aortic homograft conduit 1.82, pulmonary or aortic homograft implant 2.21, porcine valve conduit 1.80, porcine valve implant 1.59, Polystan conduit 3.39, Pericardial valve implant 1.90), and time-dependent requirement for pulmonary valve stent placement. Important predictors of valve failure varied among age groups: Age less than 3 months: valve type; Age 3 months to two years: smaller normalized valve prosthesis size; Age 2 years to 13 years: gender, smaller normalized valve prosthesis size, placement of endovascular stents, and valve type; Age 13 years to 65 years: smaller normalized valve prosthesis size, placement of endovascular stents, and increased number of previous valve placements.

CONCLUSIONS: There is a significant interaction between age and the effects of diagnosis, valve type, and size on prosthetic pulmonary valve longevity.

**By Invitation*

4. The Batista Procedure Is Not an Alternative to Cardiac Transplantation

†Anders Franco-Cereceda, Patrick M. McCarthy, Eugene H. Blackstone, Katherine J. Hoercher*, Jennifer A. White*, James B. Young* and Randall C. Starling*, Cleveland, Ohio

Discussant: Gianni Angelini, M.D.

OBJECTIVE: We prospectively investigated partial left ventriculectomy (PLV; Batista procedure) to assess suitability as an alternative to cardiac transplantation (Tx).

METHODS: From May 1996 until December 1998, 62 patients (pts) had PLV, with mitral valve repair (MVR) in 95% (mean mitral regurgitation [MR] 3.0±1.0; only 26% had 4+ MR).

RESULTS: Survival and freedom from failure (Class IV CHF) are shown in the table. Despite extensive interrogation of preoperative variables, including MR, only higher peak O_2 consumption was predictive of 3-year freedom from failure.

CONCLUSIONS: PLV is associated with a high risk of early failure which was largely unpredictable and not related to preoperative MR. Early and late failures preclude use of PLV as an alternative to Tx. Less traumatic methods to reduce LV wall stress in more selected pts may improve upon these results.

	30 days	3 mos	12 mos	24 mos	36 mos
Survival	99%	94%	76%	64%	53%
Freedom from failure	81%	72%	57%	48%	42%
Class IV risk/month	8.4%	4.3%	1.9%	1.2%	1.0%

9:35 a.m. Andrew G. Morrow Research Scholar Presentation

Stephen C. Yang

Johns Hopkins University School of Medicine

9:40 a.m. 47th Evarts A. Graham Memorial Traveling Fellowship Presentation

Anders Franco-Cereceda

Stockholm, Sweden

9:45 a.m. INTERMISSION - VISIT EXHIBITS

†1999-2000 *AATS Graham Fellow*

*By *Invitation*

10:30 a.m. SCIENTIFIC SESSION

Constitution Hall, Metro Toronto Convention Centre

Moderators: James L. Cox, M.D.

Tirone E. David, M.D.

5. Persistent Left Ventricular Hypertrophy Influences Survival Following AYR in Patients with the Medtronic Freestyle Stentless Bioprosthesis

Dario F. Del RizzcA Ahmed Abdoh*, Paul Cartier*§, Donald D. Doty§ and Stephen Westaby[5], Winnipeg, MB, and Quebec City, PQ, Canada; Salt Lake City, Utah; Oxford, United Kingdom

Discussant: David J. Wheatley, M.D.

OBJECTIVE: Small non-randomized cohort series have suggested that the superior hemodynamic performance of Stentless valves confers a survival advantage to patients undergoing AVR, when compared to conventional stented and mechanical devices. It has been suggested that this difference may be related to better regression of LVH. We hypothesize that persistent LVH is associated with decreased survival following AVR.

METHODS: We examined 1173 patients who underwent AVR with the Medtronic Freestyle Stentless valve from 1992-1997 by subcoronary (846),miniroot(103), or full root(224) techniques. The series had 54.9% males, 73.5% with NYHAIII/IV symptoms, and 35.1% of patients had concomitant CABG surgery. As was the dominant lesion in 40.7%.

RESULTS: Cox's proportional hazards model identified age and post-op LV mass index (LVMI), expressed as continuous variables, as important determinants of long-term survival. There was an incremental increased risk of death of 8% for each year of age(HR 1.08, 95% CI 1.03-1.12, $p < 0.001$) and for every increase in LVMI of 1 gm/m^2 the risk of death increased by 1% (HR 1.01, 95% CI 1.004-1.012, $p < 0.001$). Multiple linear regression analysis revealed that LV mass regression was influenced by LVMI at surgery ($p < 0.001$), proir MI ($p = 0.04$), history of carotid stenosis ($p = .02$), and systemic hypertension ($p = 0.01$). Indexed EOA (EOA)was also an important predictor of LVMI. If EOA, remained < 0.8 cm^2/nr at 3-years post-op, LVMI remained at 95.5 ± 26.5% of baseline (i.e. < 5% reduction). In sharp contrast, if EOA, was > 0.8 cm^2/m^2 there was nearly a 25% reduction in LVMI ($p < 0.001$) as compared to LVMI immediately post-op.

CONCLUSIONS: The data demonstrate that persistent LVH despite surgical correction places patients at increased risk of mortality following AVR. Persistent LVH is strongly influenced by baseline LVMI, hypertension, and the hemodynamic performance of the prosthesis. The data argue that earlier intervention, treatment with antihypertensive drugs, and careful attention to patient-prosthetic mismatch may have important prognostic implications to the patient.

§Authors have a relationship with Medtronic

*By *Invitation*

6. Prosthesis Size and Mortality After Aortic Valve Replacement: a Multi-Institutional Meta-Analysis

Eugene H. Blackstone, Eric G. Butchart*, Delos M. Cosgrove, W.R. Eric Jamieson, John H. Lemmer, D. Craig Miller and Akiko Chai*, Cleveland, Ohio; Cardiff, Wales, United Kingdom; Vancouver, BC, Canada; Portland, Oregon; Stanford and Irvine, California

Discussant: David H. Adams, M.D.

OBJECTIVE: It has been suggested that larger aortic prostheses, with lower trans-prosthesis gradients, are associated with superior survival. This large multi-institutional study was undertaken as a definitive investigation of the relation between prosthesis size and survival.

METHODS: Pooled data for 6610 AVRs from 6 institutions provided 40,415 patient-years of follow-up for analysis, mean 6.5±4.5 years, maximum 20 years, with 25% followed >10 years. 3561 prostheses were porcine xe-nograft, 1222 bovine pericardium, 1730 mechanical, and 97 allograft. 491 were manufacturer's labeled size 19. Prosthesis size was expressed as labeled size (mm), indexed orifice area (IOA, cm^2/m^2 BSA), and standardized size (Z-value, number of standard deviations [SDs] from mean normal AV size based on BSA). 12.5% of patients received a prosthesis with IOA <1.25 cm^2/m^2. 18% received a prosthesis between -2 and -5 SDs (Z-value) below normal. Multivariable analyses identified factors associated with use of smaller prostheses, and hazard function analysis quantified the influence of prosthesis size on survival, adjusted for valve type, clinical and operative variables.

RESULTS: Smaller prostheses were placed in women and smaller patients, the elderly, and patients with aortic stenosis (all P<.0001). 30-day mortality was 4.4%. Risk factors included higher

NYHA class P<.0001, previous AVR P<.0001, and concomitant CABG P=.002. Labeled prosthesis size (P=.6), IOA (P=.6), and Z-value (P=.9) were not risk factors. 10-year survivals for IOA <1.5, 1.5-2, and >2 cm^2/m^2 were 60±2.1%, 54±1.6%, and 56±1.6%. 10-year survivals for Z-value <-2, -2 to 0, and >0 SDs were 64±2.6%, 53±1.5%, and 56±1.5%. Risk factors for late mortality included older age, men, aortic regurgitation, previous AVR, and concomitant CABG (all P<.0001); however, prosthesis size expressed in any fashion had no demonstrable influence.

CONCLUSIONS: 1) Small prosthesis size does not influence early or late survival, down to an IOA of 0.8 cm^2/m^2 or 4 SDs below normal. 2) Therefore, neither oversizing the prosthesis nor aortic root enlargement appear necessary for managing most small aortic roots.

11:15 a.m. PRESIDENTIAL ADDRESS

The Innovation Imperative
Delos M. Cosgrove, M.D.

Cleveland, Ohio

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

**By Invitation*

MONDAY AFTERNOON, MAY 1, 2000

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

Constitution Hall, Metro Toronto Convention Centre

Moderators: Verdi DiSesa, M.D.

Marko I. Turina, M.D.

7. The Porcelain Aorta at Aortic Valve Replacement: Surgical Strategies and Results

Bruce W. Lytle, A. Marc Gillinov*, Vu Hoang*, Delos M. Cosgrove, Michael K. Banbury*, Patrick M. McCarthy, Gosta B. Pettersson*, Joseph F. Sabik*, Nicholas G. Smedira* and Eugene H. Blackstone, Cleveland, Ohio

Discussant: Nicholas T. Kouchoukos, M.D.

OBJECTIVE: Aortic valve replacement (AYR) in patients (pts) with severe ascending aortic atherosclerosis poses technical challenges. A "no-touch" technique including AVR under deep hypothermic circulatory arrest (HCA) has been advocated when dealing with the porcelain or unclampable aorta. The purpose of this study was to determine operative strategies and results of AVR in pts with a severely atherosclerotic ascending aorta that could not be safely cross-clamped.

METHODS: From 1/90 to 12/98, 4983 pts had aortic valve surgery; of these, 62 pts (1.2%) had a severely atherosclerotic ascending aorta and required HCA to facilitate AVR, and they form the study group. 40% had previous cardiac surgery and 13% had history of chest irradiation. Severe aortic atherosclerosis was recognized preoperatively in 50%.

RESULTS: All pts had HCA, but several different strategies were used to manage the ascending aorta (table). Overall mortality was 14%, and 10% of pts suffered strokes. Increasing NYHA functional class and impaired left ventricular function were risk factors for hospital mortality. Choice of operative technique did not influence pt outcome; however, no pt having ascending aortic replacement suffered a stroke.

CONCLUSIONS: AVR in pts with severe ascending aortic atherosclerosis is associated with increased operative morbidity and mortality. Complete AVR under HCA requires a prolonged period of HCA. Ascending aortic replacement is a preferred technique as it requires a short period of HCA and results in comparable mortality with a low risk of stroke.

	AVR under HCA	Aortic Endarterectomy Replacement	Ascending Aortic	Inspect and Cross-Clamp	Balloon Occlusion
Number	24	16	12	6	4
HCA (min)	53±20*	13±6	17±6	4±5	5±4
Stroke	17%	12%	0	0	0
Mortality	12%	25%	17%	0	0

**P<.001* vs. other techniques

**By Invitation*

8. Valvular Heart Surgery in Patients with Previous Mediastinal Radiation Therapy

Nobohiro Handa*, Christopher G. A. McGregor*, Gordon K. Danielson, Richard C. Daly*, Joseph A. Dearani*, Charles J. Mullany, Thomas A. Orszulak, Hartzell V. Schaff, Kenton J. Zehr*, Paula J. Schomberg*, Betty J. Anderson* and Francisco J. Puga, Rochester, Minnesota

Discussant: R. Scott Mitchell, AID.

OBJECTIVE: To characterize the outcome of valvular heart surgery for patients with previous mediastinal radiation therapy from January 1976 through December 1998.

METHODS: The study consists of 60 patients (37 females, 23 males) with a mean age of 62±15 years (28 to 88 years). Valvular heart surgery performed included aortic valve replacement (n=26), mitral valve procedure (n=16), tricuspid valve procedure (n=6), and multiple valve procedure (n=12). Associated procedures included coronary bypass surgery (48%), pericardiectomy (12%), myectomy (5%), chest wall reconstruction (5%) and permanent pacemaker placement (2%).

RESULTS: Early mortality was 7 cases (12%). Early mortality in patients with constrictive pericarditis was 40%(4/10) compared with 6%(3/50) in patients without constrictive pericarditis. By univariate analysis, early mortality was associated with constrictive pericarditis (P=0.011), reduced preoperative ejection fraction (P=0.015) and longer cardiopulmonary bypass times (P=0.037). A total of 14 patients (23%) required PPM before (n=7), during(n=l), or early(n=6) after valvular heart surgery. Total follow-up was 199 patient-years. There were 19 late deaths (malignancy 7, heart failure 5, other cardiac 4, other non-cardiac 3). Survival rates free of all causes of death, late cardiac death and cardiac reoperation at 5 years for hospital survivors were 66±8%, 82±7% and 93±4%, respectively. By univariate analysis, late cardiac death was associated with low ejection fraction (P=0.002), NYHA class IV(P=0.004), preoperative congestive heart

failure(P=0.02), and preoperative atrial fibrillation(P=0.038). Eighty-five percent of the discharged patients were in NYHA class I or II at follow-up.
CONCLUSIONS: Early results of valve replacement after mediastinal radiation therapy were good except in the presence of constrictive pericarditis. Long-term outcome was limited by malignancy and heart failure. Early surgical intervention is recommended before the development of risk factors for late death, namely, severe symptoms, left ventricular dysfunction and atrial fibrillation.

*By *Invitation*

9. Late Results of Heart Valve Replacement with the Hancock II Bioprosthesis

Gideon Cohen*, Tirone E. David, Susan Armstrong* and Joan Ivanov*,

Toronto, ON, Canada

Discussant: W.R. Eric Jamieson, M.D.

OBJECTIVE: The Hancock II bioprosthesis was recently approved by FDA for clinical use in the USA. This report describes the late clinical outcomes of patients who had AVR and MVR with this bioprosthesis.

METHODS: From 1982 to 1994, 670 pts had AVR and 310 had MVR with Hancock II bioprosthesis. Patients' mean age was 65±12 years for both groups. Most patients were in NYHA class III and TV and 41% of AVR group and 45% of MVR had coronary artery disease. Patients were followed prospectively at annual intervals. The mean follow-up was 87±45 months for AVR and 75+48 months for MVR, and it was 99% complete for both groups.

RESULTS: Table 1 shows the freedom from morbid events at 10 and 15 years. Patient's age and valve position were independent predictors of primary tissue failure. The freedom from primary tissue failure after AVR at 15 years was 72%±7% for patients <65 years of age and 99.6%±0.4% for pts >65 years of age whereas after MVR was 60%+9% for pts <65 years and 74%±9% for >65 years.

CONCLUSIONS: The Hancock II bioprosthesis has provided good clinical outcomes and it is a durable valve in older patients, particularly in the aortic position.

Table 1: Freedom from morbid events at 10 and 15 years

	AVR		MVR	
Freedom from:	10 yr.	15 yr.	10 yr.	15 yr.
Death	61%±2%	47%±3%	52%±3%	30%±5%
Valve-related death	95%±1%	92%±2%	89%±1%	86%±3%
Cardiac-related death	80%±2%	72%±3%	73%±3%	47%±7%
Thromboembolism	87%±2%	83%±3%	89%±2%	87%±3%
Endocarditis	97%±1%	96%±1%	96%±1%	91%±4%
Primary tissue failure	97%±1%	81%±5%	86%±3%	66%±6%
Reoperation	94%±1%	77%±5%	85%±3%	69%±6%

**By Invitation*

10. Mitral Valve Repair and Aortic Valve Replacement Is Superior to Double Valve Replacement

A. Marc Gillinov*, Eugene H. Blackstone, Delos M. Cosgrove, Paul Kerr*, Antonino Marullo*, Patrick M. McCarthy, Nicholas G. Smedira* and Bruce W. Lytle, Cleveland, Ohio
Discussant: Cary W. Akins, M.D.

OBJECTIVE: Double valve replacement has been advocated for patients with concomitant aortic and mitral valve disease. The purpose of this study was to determine if mitral valve repair is superior to mitral replacement.

METHODS: From 1975 to 1998,984 patients underwent double valve surgery. Of these, 819 had aortic valve replacement with either mitral valve replacement (n=518) or repair (n=301). Mitral valve pathology was rheumatic in 70% and degenerative in 20%. Mitral valve repair included commissurotomy in 131 (44% of repairs), ring annuloplasty in 170 (56%), leaflet resection in 27 (9%) and chordal procedures in 14 (5%). The prevalence of mitral valve repair increased from 25% in the 1970s to 50% in the 1990s. Mitral valve replacement was more common in pts with severe mitral stenosis P<.0001, atrial fibrillation P=.0009, and patients receiving a mechanical aortic prosthesis P=.0005. Mitral valve repair was more common in patients with annular dilatation P<.0001. These differences were used for propensity-matched multivariable comparisons. Follow-up extended to 22 years, mean 6.9+5.9 years, with 5199 patient-years of follow-up available for analysis.

RESULTS: Hospital mortality was 6.4%. It was similar for mitral valve repair (5.3%) and mitral valve replacement (7.0%) P=.4. Survival at 5,10, 15, and 20 years was 80%, 63%, 46%, and 31% after mitral valve repair vs. only 72%, 52%, 34%, and 21% after mitral valve replacement P=.006. Late mortality was increased by older age P<.0001, atrial fibrillation P=.009, and mitral valve replacement rather than repair P<.0001. After repair of non-rheumatic mitral valves, 5, 10, and 15-year freedom from valve replacement was 94%, 92%, and 90%. In contrast, after repair of rheumatic mitral valves, freedom from valve replacement at these intervals was 97%, 89%, and 73%.

CONCLUSIONS: Mitral valve repair is 1) feasible in a large proportion of patients with double valve disease, 2) improves late survival in patients with double valve disease, and 3) should be considered in all patients with double valve disease, including those with rheumatic mitral stenosis.

**By Invitation*

11. Cardiac Surgery Combined with the Maze-Ill Procedure

James L. Cox, Niv Ad* and Terry Palazzo*, Washington, District of Columbia

Discussant: Hartzell V. Schaff, M.D.

OBJECTIVE: This study was designed to determine the efficacy of combining the Maze procedure with other types of cardiac surgical procedures.

METHODS: Between April 1992 and October 1999, we performed 301 Maze-Hi procedures. 180 patients underwent the Maze only, and 121 patients had the Maze plus other cardiac surgery,

including valve surgery in 75 patients and non-valve cardiac surgery in 46 patients. Events within the first 3 months of surgery were considered perioperative . 263 patients were followed from 3 months to 7.5 years (Mean: 3.9 + 2.7 years) (Late).

RESULTS: See Table. The operative mortality rate for Maze plus mitral valve surgery was 2.5 % and the arrhythmia control was 98 % (n=40).

CONCLUSIONS: Perioperative mortality and morbidity are related directly to age > 65 years but not to cardiac surgery performed concomi-tantly with the Maze procedure. Atrial fibrillation is controlled in 98-99% of patients whether or not concomitant cardiac surgery is required.

Table 1

	Maze Only	**Maze + Other**	**P**
Perioperative Stroke Rate	0.4%	0%	NS
Overall Operative Mortality Rate	1.3%	5.0%	*
Operative Mortality Rate <65	0%	0.8%	NS
Operative Mortality Rate >65	10.3%	10.6%	NS
Late Stroke Rate	0.6%	0%	NS
Late Mortality Rate	1.3%	3.0%	NS
Arrhythmia Control	99%	98%	NS

*p<0.05 by univariant analysis; p=0.06 by multivariant analysis

3:15 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

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

Constitution Hall, Metro Toronto Convention Centre

Moderators: Verdi DiSesa, M.D.

Marko I. Turina, M.D.

12. Optimal Surgical Management of Mitral Regurgitation from Anterior Leaflet Prolapse

Ian A. Nicholson*, Lawrence H. Cohn, Gregory S. Couper and David H. Adams, Boston, Massachusetts

Discussant: Ottavio Alfieri, M.D.

OBJECTIVE: Anterior leaflet prolapse of the mitral valve remains a challenge in mitral valve repair for myxomatous degeneration. We reviewed 173 patients undergoing MV repair for anterior leaflet prolapse to determine the most durable operative method.

METHODS: One hundred and seventy three patients (114 males , 59 females) underwent mitral valve repair between 1984 and 1999. Mean age at operation was 59 years. Patients underwent either chordal shortening and/or anterior leaflet resection (Group 1, N= 100) or Gortex chordoplasty {2-4 mattress sutures of C5 Gortex to anterior leaflet}(Group 2 , N= 73). The mean follow-up was 3.1 years in Group 1 and 2.25 years in group 2 .

RESULTS: Cardiopulmonary bypass and aortic cross clamp times were similar in the two groups. The incidence of concomitant CAD requiring C ABG was 26% in Group 1 and 33% in Group 2 . Operative death rate was 3% for Group 1 vs 1.4% for Group 2 [P = N.S.]. Late deaths were 4 (4%) in Groupl and 3 (4.1%) in Group 2 [P= N.S] Re-operation for structural valve degeneration occurred in 15/100 (15%) in Group 1 and only 4/73 (5.4%) in Group 2 [P< 0.04].
CONCLUSIONS: Gortex chordoplasty is a more reproducible technique for anterior leaflet prolapse repair with a much lower reoperation rate for failed repair.

*By *Invitation*

13. Increased Mortality of Aortic Valve Re-Replacement Is Not Due to Aortic Valve Reoperation

Terrence M. Yau*, Joan Ivanov* and Tirone E. David, Toronto, ON, Canada

Discussant: Thomas Orszulak, M.D.

OBJECTIVE: We quantified the contribution of redo AV surgery itself to the mortality of AV re-replacement.

METHODS: Predictors of early outcomes and the effect of reoperation were determined by logistic regression in 1881 patients undergoing AV surgery from 1990-1998.

RESULTS: Patients undergoing redo AV surgery (N=205, 11%) were younger, more likely to require urgent surgery, to have heart failure, endocarditis, and AI or mixed AS/AI than primary patients (all p=0.001), but less likely to have diabetes (p=0.003) or coronary disease (p=0.001). NYHA class, LV function, BSA, valve size and crossclamp times were not different. Annular enlargements were more common in redo procedures (23% vs. 34%, p=0.0002). Mortality (2.3% vs. 4.4%, p=0.07) and stroke (2.2% vs. 4.9%, p=0.02) were greater in redos, but MI, low output syndrome and IABP use were similar. Redo AV surgery itself carried only a slightly increased odds ratio for mortality (Table) compared to other risk factors; the mortality of elective re-replacement (1.7%) was similar to that of primary surgery (1.5%) (p=0.8).

CONCLUSIONS: The risk of AV re-replacement is due mostly to endocarditis or shock, annular enlargement, and comorbidity, rather than the requirement for AV re-replacement itself. This data supports primary implantation of bioprosthetic AVs in young patients to avoid anticoagulation and its complications, as elective reoperation for primary tissue failure is associated with low risk.

Independent Predictors of Mortality	**Odds Ratio**	**95% Cl**
Age	1.04	1.01-1.07
PVD	3.76	1.77-7.99
Shock	4.37	1.71-11.2
Active endocarditis	4.87	1.67-14.2
Annular enlargement	2.18	1.17-4.05
Redo AV surgery	***1.55***	***1.02-2.33***

**By Invitation*

14. Heparinless Cardiopulmonary Bypass for Repair of Aortic Trauma

Stephen W. Downing*, Marcelo G. Cardarelli*, Safuh Attar, Douglas C. Wallace*, Aurelio Rodriguez*, Joseph S. McLaughlin, Jamie Brown* and Glenn J. R. Whitman, Baltimore, Maryland

Discussant: Irving L. Kron, M.D.

OBJECTIVE: Distal circulatory support for the repair of traumatic rupture (TR) of the aorta reduces paraplegia. However, standard cardiopul-monary bypass (CPB) requires heparin and may increase bleeding and death. Left atrial to aortic bypass eliminates heparin, but cannot heat, cool, oxygenate or rapidly add volume. We hypothesized that a heparin-bonded CPB system would be simple, effective, and free of these shortcomings.

METHODS: A retrospective review over a 5 year period at a regional level I trauma center. A heparin-bonded bypass system was utilized consisting of a 19 or 21 French femoral vein (right atrial) line, an oxygenator-heater/ cooler and a centrifugal pump flowing at 3-5 L per minute. Arterial return was to the femoral artery or distal aorta. No systemic heparin was given.

RESULTS: From 7/6/94 to 9/8/99, 54 patients underwent repair of a TR. Two patients repaired with simple clamping, 2 patients already on ECMO and 1 patient who exsanguinated at thoracotomy were excluded. The mean age was 43 ± 17 years. 14% were hypotensive, 16% had intracranial injuries, 37% had pelvic injuries, 63% had abdominal injuries and 24% had pulmonary contusions. The cross clamp time was 32 ± 11 minutes and bypass time was 64 ± 44 minutes. In the first 15 patients the femoral artery and vein were cannulated in radiology after angiography. There was one femoral artery and one femoral vein injury with one limb loss and this procedure was discontinued. The subsequent 34 patients had percutaneous femoral vein and direct distal aortic cannulation without event. The mortality rate was 10%. One death was intraoperarive due to arrythmia, the remainder were due to other injuries. There was no new paraplegia and no worsening of neurologic or pulmonary injuries.

CONCLUSIONS: This approach has advantages over standard CPB and left atrial to aortic bypass including simple cannulation without intrapericardial or hilar dissection, avoidance of anticoagulation; and the ability to easily treat hypothermia, hypoxia and hypovolemia. The mortality rate was below published averages and paraplegia effectively prevented.

**By Invitation*

15. Extracorporeal Membrane Oxygenation in 242 Adults: Survival at 1 Year

Nader Moazami*, Nicholas G. Smedira*, Patrick M. McCarthy, Camille M. Golding*, Bruce W. Lytle, Eugene H. Blackstone and Delos M. Cosgrove, Cleveland, Ohio

Discussant: Robert H. Bartlett, M.D.

OBJECTIVE: To define the survival and the changing role of ECMO in a diverse population of patients in the modern era of LVAD support and thoracic transplantation.

METHODS: Retrospective review of 242 adult patients with a mean age of 53±14 years who were placed on ECMO support from 1992-1999. Indications were post-cardiotomy (119), myocardial infarction (35), ARDS (23), cardiac arrest (11), decompensated heart failure (31), deterioration during cardiac cath-eterization (6), and after cardiac (10) and lung (7) transplantation.

RESULTS: Veno-arterial support was employed in 209 patients. In this group, 54 (26%) were bridged to LVAD or heart transplantation, 80 (38%) were weaned, and 75 (36%) died on ECMO. Overall, 68 (33%) were discharged; 30 (55%) in the LVAD versus 38 (48%) in the weaned group (p£0.2). At 1 year, 51 (75%) patients were alive. Veno-venous support was used in 33 patients, 18 with ARDS, 5 post-lung transplantation, 8 post-cardiotomy, and 1 after heart transplantation and acute MI. Overall, 17 patients died on ECMO, 16 (48%) were weaned and 11 (69%) were discharged home; 8 (72%) were alive at 1 year. In specific subgroups, survival to discharge varied from 66% in the post-catheterization to £20% in the heart transplant and cardiac arrest groups (p£0.001). Mortality was associated with severe neurologic deficit in 19 (11%), irreversible myocardial damage in 63 (39%) non-transplant candidates, and multi-system organ failure in 81 patients (50%).
CONCLUSIONS: ECMO can be used in a large and diversified setting of cardiopulmonary collapse. Although overall mortality remains high, 1-year survival after discharge is excellent. Use of veno-arterial ECMO support as bridge to LVAD implantation allows survival of a large number of patients who would otherwise die.

**By Invitation*

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B

GENERAL THORACIC SURGERYRoom 205

Moderators: Thomas W. Rice, M.D.

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

16 Detection of Early Lung Cancer. CT Scan or Chest X-Ray? Survival Implications

Nasser K. Altorki, Michael S. Kent*, David Yankelevitz*, Claudia Henschke*,

Daniel Libby*, Mark Pasmantier* and James P. Smith, New York, New York

Discussant: Joe Friedberg, M.D.

OBJECTIVE: It has been recently proposed that chest CT scans may be a useful method for early detection of lung cancer. In this study we determined the stage distribution of lung cancers detected by a screening CT scan. This was compared with the stage distribution of patients whose lung cancers were detected by a routine chest X-ray (CXR).

METHODS: Two groups of patients were reviewed. Twenty patients had biopsy-proven non-small cell lung cancer detected through a CT scan screening program. A second group of patients (n=103)had their lung cancers detected on routine CXR. Patients with pulmonary symptoms or prior history of cancer were excluded.

RESULTS: There was no difference in age, gender or cell-type distribution between the two groups. Stage distribution is shown in the following table. There was no difference between the groups in the overall prevalence of Stage I disease versus more advanced disease. However, a significantly greater number of patients were stage IA in the CT group compared to the CXR group (p=.004). Of 15 patients with Stage I disease in the CT group, 7 had tumors 1 cm. or less versus 8 out of 74 stage I patients detected by CXR.

CONCLUSIONS: As a screening modality for lung cancer CT scan yields a higher incidence of Stage LA disease than that achievable by a CXR. This may result in significant imporoval of survival in patients with Stage I disease.

TNM stage	CXR(n=103)	CT scan (n=20)
IA	41 (40%)	15(75%)
IB	33 (32%)	1 (6%)
IIA	6 (6%)	1 (6%)
IIB	15(15%)	0
IIIA	1 (1%)	2(12%)
IIIB		1 (6%)

**By Invitation*

17. Subcentimeter Non-Small Cell Lung Cancer a Program for Detection and Resection Is Warranted

Scott J. Swanson*, Raphael Bueno*, Michael T. Jaklitsch*, Steven J. Mentzer, Jeanne M. Lukanich* and David J. Sugarbaker, Boston, Massachusetts

Discussant: Joel D. Cooper, M.D.

OBJECTIVE: For lung cancer screening to have a favorable impact, survival of patients whose tumor is detected when relatively small should be superior to that of patients with larger tumors. To look at this, we examined the survival of patients who had a resection of non-small cell lung cancer that was less than or equal to 1 centimeter.

METHODS: From 1990-1998, 182 patients had malignant solitary lung nodules less than or equal to 1 centimeter resected at the Brigham and Women s Hospital. Of these, 40 patients had primary non-small cell lung cancer (node-negative or indeterminate). Preoperative, perioperative and follow up data were recorded in our prospective thoracic database. Survival was performed by Kaplan-Meier lifetable analysis.

RESULTS: 27 women and 13 men (37/40 with smoking history), median age 64 years (46-86) underwent 9 anatomical (lobes/segments), 8 wedge resection with node sampling and 23 wedge resection without node sampling. Median tumor size = 0.8 cm (0.2-1.0). Histologically, there were 34 adenocarcinomas, 5 squamous cell and 1 undifferentiated carcinoma. There was no perioperative mortality. Median length of stay was 4 days (1-15). Follow up is complete. Five-year survival is 88%. Median survival has not been reached at a median follow up of 3.3 years. Type of resection was not statistically significant (logrank, $p = 0.43$) although there were no recurrences or late deaths in the anatomic resection subgroup. This may reflect more accurate staging.

CONCLUSIONS: Long-term survival following resection for subcentimeter non-small lung cancers appears better than that for overall stage I lung cancer. These data support aggressive screening and surgical strategies for small non-small cell lung cancers. The use of helical CT scanning for early lung cancer, as recently reported, may be the screening method of choice.

*By *Invitation*

18. CALGB 9335: a Multi-Center Phase-II Prospective Study of Video-Assisted Wedge Resection Followed by Radiotherapy for T1NO NSCLC in High-Risk Patients; Preliminary Analysis of Technical Outcome

Hani Shennib, Leslie Kohman, James E. Herndon*, Jeffrey Bogart*, David J. Sugarbaker, Mark Green* and Robert Keenan, Montreal, Canada; Syracuse, New York; Durham, North Carolina; Boston, Massachusetts; Charleston, South Carolina; Pittsburgh, Pennsylvania

Discussant: Robert J. Ginsberg, M.D.

OBJECTIVE: Video-assisted technology may offer advantages, not yet proven, in cardiothoracic surgery. The objective of this NIH sponsored phase-II prospective multicenter trial was to determine the feasibility of treating patients with cardiopulmonary dysfunction and T1 peripheral non-small cell lung cancer by video-assisted wedge resection and local (56Gy)radiotherapy. High-risk patients had one or more of the following: FEV_1<40%predicted, DLCO <50% Vo_2max<15ml/Kg/min, use of Supplemental oxygen,and Pa Co_2 >45mmHg.

METHODS: Between September 1995 and September 1999, 65 patients were accrued of which 60 were eligible [50%male, median age 69YJ. Technical failure occured in 15 patients [25%]. These included conversion to open thoracotomy in 9 patients, abortion of the operation in 2 patients, 1 postoperative death and 3 patients with postoperative positive resection margins. Postoperative staging was raised to T2 in 6 patients [10%] and benign in another 6 patients [10%]. Other complciations included prolonged air leak 10%, pneumonia 6%,respiratory failure 4% arrhythmia 6%. Resection was by staplers except 6 patients by cautery and 1 patient by laser. Adhesions were absent in 48%, minimal in 21% and moderated to extensive in 28%. 39/48 patients had VATS accessible intrathoracic lymph nodes. Margins were >lcm in [45%] and l<="" span=""> patients had microscopic positive resection margins. Minimal intraoperative bleeding occured in 22 patients and moderate in 1 patient. Median duration of the procedure was 160 min [40-255min]. Only 22 patients proceeded to radiotherapy.

CONCLUSIONS: We conculde that VATS wedge resection is feasible and relatively safe in the majority of patients with poor cardiopulmonary status but there is a substantial incidence of conversion to thoracotomy and positive resection margins. Long-term local control with this method is as yet unknown, but the low incidence of successful completion of radiotherapy indicates that this approach may not be feasible.

**By Invitation*

19. Factors Affecting Early Morbidity and Mortality After Pneumonectomy for Malignant Disease

Alain Bernard*, Claude Deschamps, Mark S. Allen, Daniel L. Miller*, Victor F. Trastek, Greg D. Jenkins*, and Peter C. Pairolero, Rochester, Minnesota

Discussant: Malcolm M. DeCamp, Jr., M.D.

OBJECTIVE: Pneumonectomy may be associated with significant morbidity and mortality with little information existing as to the factors involved.

METHODS: From January 1985 to September 1998, 639 consecutive pts (469 males and 170 females) underwent pneumonectomy for malignancy. Median age was 64 years (range, 20 to 86 yrs). Indication for resection was primary malignancy in 607 pts (95.0%) and metastatic disease in

32 (5.0%). Forty-nine pts (7.7%) underwent completion pneumonectomy. Factors affecting in-hospital morbidity and mortality were analyzed using univariate and multivariate analysis.
RESULTS: Cardiopulmonary complications occurred in 245 pts (morbidity, 38.3%; 95% CI, 34.6 to 42.2%). Univariate analysis demonstrated that factors adversely affecting morbidity included increasing age ($p < 0.01$), male gender ($p = 0.04$), associated respiratory ($p = 0.02$) or cardiovascular disease ($p < 0.01$), amount of cigarette smoking ($p = 0.02$), preoperative radiation ($p = 0.02$), muscle reinforcement of bronchial stump ($p < 0.001$), and amount of blood transfused ($p = 0.01$). Factors adversely affecting morbidity with multivariate analysis included increasing age ($p < 0.001$), associated cardiovascular disease ($p = 0.001$) and muscle reinforcement of bronchial stump ($p < 0.001$). There were 43 deaths (mortality, 6.7%; 95% CI, 4.9 to 9.0%). Mortality was 6.6% ($n = 40$) for primary malignancy and 9.4% ($n = 3$) for metastatic disease. Factors adversely affecting mortality with univariate analysis included associated cardiovascular ($p = 0.05$) or hematologic disease ($p = 0.03$), preoperative chemotherapy ($p = 0.01$) or radiation ($p = 0.04$), muscle reinforcement of bronchial stump ($p = 0.03$), extended resection, ($p = 0.02$), and decreased DLCO ($p < 0.01$, $N = 388$). Factors affecting mortality with multivariate analysis included associated cardiovascular ($p = 0.04$) hematologic disease ($p = 0.01$), and lower body mass index ($p = 0.01$).

CONCLUSIONS: Multiple factors adversely affect morbidity and mortality after pneumonectomy. Appropriate selection and meticulous perioperative care are paramount to minimize risks in pts who require pneumonectomy for cure.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:50 p.m. SIMULTANEOUS SCIENTIFIC SESSION B

GENERAL THORACIC SURGERYRoom 205

Moderators: Thomas W. *Rice, M.D.*

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

20. Experience with Pulmonary Resection from Gynecologic Malignancies

John J. McMahon*, Chukwumere E. Nwogu*, Mathew W. Pombo*, M. Steven Fiver*, Shashikant B. Lele*, Deborah L. Driscoll* and Timothy M. Anderson*, Buffalo, New York

Discussant: Michael Maddaus, M.D.

OBJECTIVE: Pulmonary metastases from cervical and uterine primaries are uncommon. Although thoracotomy for removal of isolated pulmonary metastasis is well documented in a wide variety of solid rumors, there is a paucity of data regarding the optimal managment of patients with gynecologic malignancies metastatic to lung. We have analyzed a single institution experience in an attempt to clarify the role of metastasectomy for uterine and cervical cancers.

METHODS: We retrospectively reviewed the Roswell Park Cancer Institute experience between 1982 and 1999 of eighty-two patients with gynecologic tumors metastatic to lung, including 25 who underwent pulmonary resection.

RESULTS: Among 82 patients there were 60 uterine and 22 cervical primaries. Nineteen patients with uterine and 6 with cervical origin underwent pulmonary resection for lung metastases. Median survival for the combined surgery group (n=25) was 65 months compared to 32 months for the

combined non-surgical group (n=57, p=0.04). Median time from lung metastasis until death or last follow-up was 30 months in the surgical group compared to 10 months in the non-surgical group (p=0.01). Among patients with uterine primaries undergoing metastasectomy (n=19) median survival was 67 months compared to 37 months for the non-surgical uterine group. Median time from lung metastases until death or last follow-up was 26 months in the uterine surgical group compared to 13 months in the non-surgical group. Uterine leiomyosarcomas tended to have a worse prognosis than other uterine pathologies. Among patients with cervical primaries undergoing surgery (n=6), median survival was 65 months compared to 23 months in the non-surgical group (n=16, p=0.03). Median time from lung metastases until death or last follow-up in the surgical group was 36 months, compared to 6 months in the non-surgical group (p=0.003).

CONCLUSIONS: Pulmonary resection provides a survival advantage in patients with uterine and cervical metastases isolated to lung. Furthermore, there appears to be a greater disease-specific survival advantage in patients undergoing lung resections for metastases from cervical origin compared to those of uterine derivation.

**By Invitatio*

21. Early Results of Isolated Single Lung Perfusion for Treatment of Unresectable Sarcomatous Metastases

Joe B. Putnam, Jr., Robert S. Benjamin*, Soo J. Rha*, Garrett L. Walsh*, Stephen G. Swisher*, Ara A. Vaporciyan*, W. Roy Smythe* and Jack A. Roth, Houston, Texas

Discussant: Robert Downey, M.D.

OBJECTIVE: Despite resection and chemotherapy, patients with sarcoma-tous pulmonary metastases (PM) frequently progress to respiratory insufficiency and death. We examined the role of isolated single lung perfusion (ISLP) with adriamycin for patients with (1) unresectable sarcomatous PM, (2) absence of other more effective chemotherapy, and FEV1 of 0.8 liters in the con-tralateral lung.

METHODS: 15 patients, who were entered onto a Phase I study, were treated with ISLP between January, 1995 and April, 1999. Ipsilateral pulmonary artery and veins were isolated, clamped, cannulated, and perfused. ISLP was performed over 20 minutes with adriamycin in a buffered crystalloid solution at 60mg/m^2,200 mg/1 (Group 1, n=7); 75 mg/$m^{2'}$ 250 mg/1 (Group 2, n=4), or 60mg/m2,100 mg/1 (Group 3, n=4). Adriamycin levels were determined for lung, tumor, and serum. Actuarial survival was calculated.

RESULTS: No intraoperative complications occurred. Higher drug levels were obtained in lung tissues (median 125 mcg/g tissue, range 9.4 -193 mcg/g) compared to tumor (median 58 mcg/g tissue, range 9.5 -117 meg/ g). Serum drug levels were negligible. Two patients developed Grade IV pulmonary toxicity (Group II). Operative mortality was 20% (3/15): 1, paradoxical tumor embolus (Group 1); 1, acute lung injury (Group II), and 1, pneumonia 3 weeks postop (Group 3). Late toxicity included 40% decrease in ventilation and perfusion to the treated lung. Two international patients were lost to follow-up. Five of ten evaluable patients had regression or stabilization of PM compared to PM in the untreated lung. All other patients had continuous growth of PM. Actuarial median survival was 19.1 months. Four patients remain alive greater than 2 years after ISLP.

CONCLUSIONS: ISLP may be performed safely at a dose of 60 mg/m2 (200mg/l or 100 mg/1). ISLP minimizes systemic chemotherapy toxicity, achieves high drug levels in tissue, and is associated with prolonged survival in patients with isolated unresectable sarcomatous PM.

**By Invitation*

22. Is There Ever a Role for Salvage Operation in Malignant Pleural Mesothelioma

Tarek M. Aziz*, Scott Queen*, Hosney Yosef* and Dhurv Prakash*, Glasgow, United Kingdom

Discussant: L. Penfield Faber, M.D.

OBJECTIVE: We analyzed our experience in the period January 1989-December 1998 aiming to confirm the role of surgery in the multimodality treatment of malignant pleural mesothelioma

METHODS: 109 patients were diagnosed as malignant pleural mesothelioma. The median age was 62 years (range 46-73). Apre-operative tissuse diagnosis was confirmed in all patients by open-pleural biopsy. The surgical procedures included palliative pleurectomy in 18 patients, radical pleuropneumonectomies in 63 patients. Radical surgical treatment was only considered if the patient is generally fit, and the tumour was confined to the hemithorax and there was no mediastinal invasion. Post operative chemotherapy (carboplatin + epirubicin) was used the majority of patients who underwent radical surgery (except the first 13 patients)

RESULTS: The operative mortality was 8.9%. The median follow up is 42 months (range 2-87). The median survival for palliative therapy was 8 months compared to a median survival of 38 months for patients who underwent radical surgery + post-operative chemotherapy (p=0.02). However, the median survival for those who did not have post operative chemotherapy following their radical surgery was poor (13 months).Thirty four patients were still alive at 30 months following their radical surgery + chemotherapy and 21 of them being disease-free. The main factors affecting the results is the number and development of metastasis following surgery.

CONCLUSIONS: Radical surgery and adjuvant chemotherapy might represent an effective form of treatment in selected malignant pleural mesothelioma. We advocate general radical pleuro-pneumopnectomy for malignant mesothelioma if it is part of multi-modal theraputic protocol.

**By Invitation*

23. A Single-Institution, Multidisciplinary Approach to Primary Sarcomas Involving the Chest Wall Requiring Full Thickness Resections

Garrett L. Walsh*, Bryan M. Davis*, Stephen G. Swisher*, Ara A. Vaporciyan*, W. Roy Smythe*, Jack A. Roth and Joe B. Putnam, Jr.*, Houston, Texas

Discussant: Mark S. Allen, M.D.

OBJECTIVE: Primary sarcomas involving the chest wall (PSCW) requiring full thickness excision are rare. We reviewed our experience with these lesions in a tertiary referral cancer center using multidisciplinary approaches.

METHODS: A 10 year retrospective study identified 51 patients referred with PSCW; 38 for initial treatment (I) and 13 after previous failed surgical excisions elsewhere (Recur). Presenting symptoms were pain alone 23/51 (45%), pain with an associated mass 8/51 (16%) and an asymptomatic mass alone 12/51 (24%). Median symptom duration was 258 days in the primary group and 184 days in the recurrent group. Tumor locations were sternal (n=11), rib alone (n=36) and posterior rib with extension into vertebral bodies (n=4). Histologies included: chondrosarcomas (15), malignant fibrous histiocytomas (7), osteosarcoma (4), Swing's (1), desmoids (7) and other histologies (17). The median tumor volume of those presenting initially were 509 cm^3 compared to 131 cm^3 in patients with recurrent lesions.

RESULTS: 24/51 patients (47%) received treatment prior to resection including: chemotherapy alone (20), radiation alone (3) and combined chemo/XRT (1). The complete sternum was removed in 6/11 and the average rib resections required was 3.9. Four patients had vertebral body resections. Prosthetic meshes were required in 16/51 and mesh with methylmethacrylate in 18/51. Muscle flap reconstructions by plastic surgery were required in 24 patients. Negative margins were obtained in 47/51. There were no perioperative deaths with morbidities occurring in 12/51 (24%) [wound (3), prolonged air leak (1), prolonged ventilator requirement (1), arrhythmias (2), Adriamycin induced cardiomyopathy (1) and other (4)]. Post-operative treatment was administered to 13 patients [chemo alone (9) and chemo/XRT (4)]. The cumulative five-year survival of all patients was 65% [67.4% (I) and 55.4% (Recur)]. The average follow-up is 35.3 months.
CONCLUSIONS: A combined aggressive multidisciplinary approach to PSCW resulted in no treatment-related deaths and a prolonged survival in both (I) and (Recur) patient subsets.

**By Invitation*

24. Chest Wall Invasion in Non-Small Cell Lung Carcinoma.

Microscopically Negative Margins Represent the Rationale for En-Bloc Resection.

Francesco Facciolo*, Giuseppe Cardillo*, Michele Lopergolo*, Guido Pallone*, and Massimo Martelli*, Rome, Italy
Discussant: Valerie W. *Rusch, M.D.*

OBJECTIVE: Intraoperative assessment of chest wall invasion represents a challenge either for thoracic surgeon or for pathologist. Most surgeons do extrapleural dissection until they do no find clear evidence of chest wall invasion. According to such criteria the number of incomplete resection is high and the prognosis of these patients is very poor. The aim of the present study is to evaluate the need for en-bloc resection in NSCLC invading the chest wall.

METHODS: Beteween January 1990 and December 1998, out of 1621 major pulmonary resections for lung carcinoma performed at our Institution, 97 (6%) patients with NSCLC invading parietal pleura or chest wall underwent en-bloc resection of the chest wall and lung plus radical mediastinal lymphadenectomy. Indications for chest wall resection were: CT or MRI evidence of chest wall invasion intraoperatively confirmed by parietal pleura attachment. No attempt to extrapleural dissection has been performed in our series. Five of our patients underwent preoperative induction therapy because of an N2 status. Seventy-nine patients underwent adjuvant therapy .

RESULTS: All patients underwent RO radical resections with microscopically negative margins. The pathologic depth of invasion was into the pleura alone in 28 (28.9%), into the pleura and soft tissue in 31 (32%), and into the pleura, soft tissue and bone in 38(39.1%). No 30-day mortality was reported. Major complications occurred in 12 (12.4%)patients. Eighty-nine of our 97 patients were

included in the follow-up program (median: 27 months; range:9-96 months). The overall 5-year Kaplan-Meier estimated survival was 52%. The 5-year survival of patients with T3N0M0 disease was 46.8%(71 cases), T3N1M0 disease 100%(6 cases), and T3N1M0 disease 18.8%(12 cases).No locoregional recurrence was reported.

CONCLUSIONS: In patients with NSCLC invading chest wall, a complete (R0) resection can only be achieved with en-bloc resection of the chest wall and lung. The impressive 0% of locoregional recurrence justifies our aggressive approach.The long-term survival appears to be very appealing in T3N0 and T3N1 patients.

**By Invitation*

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C

CONGENITAL HEART DISEASE Room 201

Moderators: John E. Mayer, M.D.

Roger B. B. Mee, M.D.

25. Is Modified Ultrafiltration Truly Superior to Conventional Ultrafiltration for Hemoconcentration After Pediatric Cardiac Surgery?

LeNardo D. Thompson*, Doff B. McElhinney*, Pauline Findlay*, Wanda Miller-Hance*, Mark J. Chen*, Maiko Minami*, V. Mohan Reddy*, Andrew J. Parry* and Frank L. Hanley, San Francisco, California

Discussant: William Gaynor, M.D.

OBJECTIVE: Although several studies have shown MUF to be better than CUF for minimizing the consequences of hemodilution after cardiac surgery with cardiopulmonary bypass (CPB) in children, any such benefit may be due to the volume of fluid removed. We conducted a randomized study to test the hypothesis that MUF and CUF have similar efficacy when a standardized volume of fluid is removed.

METHODS: From 10/98-9/99,110 children £15 kg were randomized to MUF (43) or CUF (67) for hemoconcentration after cardiac surgery with CPB. MUF was administered after CPB and CUF during rewarming, using a Hemocor HPH 400 filtration system. UF flow and suction rates were equal, and the volume of fluid removed was standardized as a percentage of effective volume (EV) added (the sum of prime volume and volume added during and after CPB, less urine output). In pts <10 kg, 50% of EV was removed, while 60% of EV was removed in pts between 10-15 kg. Hemoglobin, hemodynamics, and shortening fraction were measured before CPB, and 10 min and 1 hr after UF.

RESULTS: Median age was 6 mo (1 d-5 yr) and median weight was 6 kg (2-15 kg). Median duration of CPB was 109 min (32-313 min). Median pre-and postoperative hematocrit levels were 35% (20-49%) and 36% (25-53%), respectively. There were no significant differences between pts assigned to MUF or CUF in age, weight, or duration of CPB. Median UF duration was 10 min (3-25 min) and did not differ between groups. Median volume of UF effluent was greater in pts receiving CUF than MUF (95163 vs 68128 mL/kg, p=0.01). The total volume of blood products received during and after CPB was greater in CUF pts (129178 vs 102148 mL/kg, p=0.05). By repeated measures ANOVA, pts receiving MUF and CUF did not differ with respect to hematocrit

(p=0.87), mean arterial pressure (p=0.85), heart rate (p=0.43), or left ventricular shortening fraction (p=0.21) from pre-CPB to 10 min and 1 hr post-UF.

CONCLUSIONS: When a standardized volume of fluid is removed based on weight and EV added, hematocrit, hemodynamics, and ventricular function do not differ between pediatric pts receiving MUF and CUF for hemofilrration after cardiac surgery.

**By Invitation*

26. Hypothennic Cardiopulmonary Bypass Alters Oxygen/Glucose Uptake by the Pediatric Brain.

Frank A. Pigula*, Edwin M. Nemoto*, Ira S. Landsman* and Ralph D. Siewers, Pittsburgh, Pennsylvania
Discussant: Richard A. Jonas, M.D.

OBJECTIVE: The effects of hypothermic cardiopulmonary bypass (CPB) on the pediatric brain remain ill defined and may contribute to brain injury. Uptake of oxygen and glucose by the brain is a critical, tightly coupled process that may be expressed as the oxygen-glucose index(OGI). We hypothesize that CPB alters OGI in the pediatric brain.

METHODS: Cerebral arteriovenous (A-V) oxygen, glucose, and lactate differences were compared in 11 children during CPB. Five paired arterial and jugular bulb samples were obtained (preCPB, $CPB_{cooling'}$, $CPB_{nadir'}$, CPB_{rewarm}, postCPB). OGI was calculated: OGI(%)=[A-$V_{O2(Å^3mol/ml)}$/6 * A-$V_{glu(Å^3mol/ml)}$ * 100 Dissolved O_2 was included.

RESULTS: On CPB_{coolmg} and CPB_{nadir}, OGI decreased significantly as A-V_{GLUC}remained stable with lower A-V_{02} At CPB_{rewarm} both A-V_{O2} and A-V_{GLUC} increased, and OGI remained depressed. A-V_{LACT} increased at re-warming.

CONCLUSIONS: We conclude that CPB alters oxygen and glucose uptake by the pediatric brain. On CPB, OGI decreased as a result of excessive cerebral glucose uptake relative to oxygen. The resulting substrate imbalance (excess glucose) may lead to osmotic cerebral edema. Also, excess glucose availability at rewarming may induce anaerobic metabolism, reflected by increased lactate production. Thus, this phenomenon may contribute to CPB related brain injury in children.

	TEMP(^{0}c)	A-V_{02}	A-V_{GLUC}	OGI(%)	A-V_{LACT}
preCPB	35±.4	2.5±.9	.4±.9	117±70	-1.3±.2
$CPB_{cooling}$	28±1	1.1 ±.5*	.4±.4	53±19†	-2.5±1.7
CPB_{nadir}	24±4	1.4±.6*	.6±.8	54±25†	-2.8±1.9
CPB_{rewarm}	26±6	2.8±.9	.8±.4	62±16†	-3.7±3.2‡
postCPB	36±.5	2.6±.8	.4±.2	149+83	-3.4±3.1‡

values are mean±SD. *p<.01 compared to preCPB, CPB_{rewarm}™, postCPB. †p<-01compared to preCPB, postCPB. ‡p<.05 compared to preCPB, ANOVA.

**By Invitation*

27. Percutaneous Arteriovenous Carbon Dioxide Removal Improves Survival in Acute Respiratory Distress Syndrome: a Prospective Randomized Outcomes Study in Adult Sheep

Joseph B. Zwischenberger, Scott K. Alpard*, Weike Tao*, Donald J. Deyo* and Akhil Bidani*, Galveston, Texas
Discussant: Robert H. Bartlett, M.D.

OBJECTIVE: $AVCO_2R$ is a simple arteriovenous shunt for CO_2 removal to minimize baro/volutrauma secondary to mechanical ventilation. We performed a prospective randomized outcomes study in our clinically relevant model of ARDS.

METHODS: Our LD40 model of ARDS requires smoke inhalation (36 breaths) and a 40% TBSA 3rd degree burn followed by protocol driven volume-controlled mechanical ventilation. All animals developed ARDS ($PaO_2/FiO_2 < 200$) 48-52 hours after injury. 18 animals randomized to $AVCO_2R$ (n=9) or SHAM (n=9). One in each group died of technical complications (statistics based on 8 per group). $AVCO_2R$ animals were anesthetized, systemically heparinized, then the common carotid artery and jugular vein cannulated with percutaneous 10F arterial and 14F venous cannulas connected to a commercially available 2.5 cm2 low resistance gas exchanger. SHAM received identical operative exposure without can-nulation. Both groups received identical, algorithm-directed pressure-controlled ventilation to normal blood gases.

RESULTS: The study involved 2,946 hours of cage-side critical care and 696 hours of $AVCO_2R$ without significant complications. 8/8 $AVCO_2R$ and only 3/8 SHAM survived the 7 day study. $AVCO_2R$ survivors averaged 2.4 days of mechanical ventilation versus 6.2 days for SHAM. The circuit pressure gradient was less than 10 mmHg and CO_2 removal averaged 103 mL/min (97% of total CO_2 production). $AVCO_2R$ blood flow ranged from 820 to 968 mL/min (11-14% of cardiac output). Cardiac output, heart rate, mean arterial pressure, and pulmonary artery wedge pressure did not significantly change despite AVCO2R. At 48 hours of ARDS, $AVCO_2R$ achieved significant reductions compared to SHAM in TV (420 to 270 mL/ min), PIP (25 to 14 cmH_2O), MV (13 to 5 L/min), RR (25 to 16 breaths/ min), and FiO_2 (.88 to .35).

CONCLUSIONS: Percutaneous $AVCO_2R$ is a simple arteriovenous shunt capable of near-total CO2 removal, which, in this model of ARDS, allowed a significant reduction in minute ventilation, significantly decreased ventilator dependent days, and significantly improved survival.

**By Invitation*

28. Surgically Created Double Orifice Left Atrioventricular Valve: a Valve-Sparing Repair in Selected Atrioventricular Septal Defects.

Loc Mac*, Patrice Dervanian*, Virginie Lambert*, Jean Losay* and Jean-Yves Neveux*, Paris, France

Discussant: Constantine Mavroudis, M.D.

OBJECTIVE: Reconstruction of a competent left atrioventricular valve (LAW) is the cornerstone of the repair of atrioventricular septal defects (AVSD). Regardless of used techniques, some structural features of LAW (large mural leaflet, dysplastic tissue valve) represent a challenge for repair without a postoperative regurgitation. A retrospective study was conducted to evaluate the results of a surgically created double orifice LAW performed in such circumstances.

METHODS: Among 157 patients operated on for AVSD since October 1989, 10 patients, selected on an individual intraoperative basis, underwent primary repair (8 pts) or reoperation (2 pts) using this additional procedure. Median age at repair was 3.3 years (range 5 weeks to 33 years). Down's syndrome was present in 4 pts. Anatomical types were complete (3), intermediate (5), and partial (2). Preoperative moderate to severe LAW regurgitation was present in 6 pts. After the standard repair (two-patch technique in cases with a common orifice, cleft closed in each case), these patients were found to have moderate to severe residual LAW valve regurgitation not amenable to repair using an annuloplasry. Thereby, the top edge of the mural leaflet was anchored to the facing free edge of the cleft using interrupted sutures.

RESULTS: No hospital death or morbidity was observed. LAW regurgitation was none or trivial (8 pts), and mild (2 pts). The repair did not result in LAW stenosis as shown by color coded echocardiography and mean LAW diastolic pressure gradient was 3.2±1.1 mm Hg (range 1.4 to 4.5 mm Hg). At a median follow-up of 69 months (range 2 to 86 months), there was 1 late death, unrelated to LAW malfunction, due to advanced pulmonary vascular disease. LAW regurgitation did not increase with time. At rest, mean LAW diastolic pressure gradient was 3.9±2.7 mm Hg (range 1.5 to 9.7 mm Hg). One child developed a moderate LAW stenosis without pulmonary hypertension.

CONCLUSIONS: Surgical creation of a double orifice LAW is an effective additional procedure for repair of atypical cases of AVSD which may decrease the need of reoperation and/or LAW replacement.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION C

CONGENITAL HEART DISEASE Room 201

Moderators: John E. Mayer, M.D.

Roger B. B. Mee, M.D.

29. Orthotopic Concordant Cardiac Xenotransplant Baboons Surviving More than 300 Days: Effect of Immunosuppressive Regimens

Miki Asano*, Steven R. Gundry, Hironori Izutani*, Sandra Nehlsen-Cannarella*, Omar Fagoaga* and Leonard L. Bailey, Loma Linda, California

Discussant: Robert E. Michler, M.D.

OBJECTIVE: We reviewed long-term survival in three consecutive series of rhesus monkey-baboon orthotopic cardiac xenotransplants(XTx)to detect lymphocyte subsets (LS), xenoantibody (XAb) to rhesus RBC and quality of life (QOL).

METHODS: Six juvenile baboons have survived more than 300 days after XTx. The immunosuppressive regimens were as follows: (A) splenectomy, FK506, methotrexate (MTX) and anti-lymphocyte globulin (G), (B)pre-trans-plant and chronic cyclosporin A (CsA), MTX and anti-thymocyte G, (C) same as (B)+ pre-transplant total lymphoid irradiation (TLI (SOcGyXlO) and intraop donor bone marrow infusion. Rejections (Rj) were detected by echocardiography. LS were monitored using CD2, CD4, CDS, CD25 and CD20. QOL was evaluated by body weight (BW)(corrected by Z-value), the number of rejections, the number of days using antibiotics (AB).

RESULTS: Group C had the least number of rejections and days on AB. BW gain was observed in all except 1 in Group B. During Rj, CD2 and CD20 increased in all groups ($p<0.05$). CD25 in group C(Rj/Rj.free 0.08±0.03/0.03±0.02mm3)was significantly lower vs A($p=0.007$/01.016)or B($p=0.0023$/$p<0.0001$). No XAb was detected in group C, whereas low titers were detected after 6 mo in group B.

CONCLUSIONS: Pre-transplant TLI combined with CsA, MTX and ATG leads to long-term survival with better QOL probably by intensive suppression of both CD25 T cell activation and Xab production.

Regime	Animal	surv(days)	Rj(#)	1st RJ(POD)	AB(days)	BW(Z-value)
A	1	504	6	55	192	-1.70
B	2	515	6	11	87	+0.21
	3	413	11	9	39	-4.02
	4	371	8	8	38	-1.81
C	5	486(alive)	3	20	3	-0.67
	6	332(alive)	2	135	4	-0.41

**By Invitation*

30. Pediatric Heart Transplantation: Improving Results in High-Risk Patients

John G. Coles, Jin Lee*, Glen Van Arsdell*, Lori West*, Lee Benson*, Anne Dipchand*, Goran Dellgren*, Carl Cordelia*, Brian W. McCrindle* and William G. Williams, Toronto, ON, Canada

Discussant: Thomas L. Spray, M.D.

OBJECTIVE: Our institutional experience with 68 pediatric patients (pts) undergoing cardiac transplantation (1990 - Oct. 1999) was reviewed to determine the impact of unconventional donor and recipient management protocols implemented to extend the availability of this therapy.

METHODS AND RESULTS: The introduction of donor blood insulin cardioplegia (IBCP) was associated with a significant improvement in patient and graft survival: among 63 ABO- matched transplant procedures, both the patient and graft loss rate were significantly (by multivariable analysis) lower with the use of the IBCP (mortality rate: 1/26; 3.8%) vs. conventional cardioplegia [11/37; 29.7%; p(Wilcoxon) <0.05], despite significantly longer ischemic times in the former group (up to 9 hr; $p<.05$). Twenty-three (33.8%) pts were deemed at ultra-high risk: 8 of 11 patients with cardiomyopathy transplanted following ECMO support survived without major sequelae; 3 of 4 additional pts survived early retransplantation. Ten pts underwent intentional ABO-incompatible (ABO-I) transplantation under a protocol of plasma exchange on bypass. There were 2 early deaths due to non-specific graft failure (n=1) and respiratory complications with mild vascular rejection (n=1), and 1 late death due to lymphoma. Among 7 surviving ABO-I pts followed up to 31 mo. there have been no episodes of humoral rejection despite development of anti-donor blood group antibodies in A to O, but not B to O, mismatches.

CONCLUSIONS: The results with pediatric cardiac transplantation continue to improve as a result of changes in both surgical and medical protocols permitting salvage of patients conventionally considered at high risk or non-transplantable.

**By Invitation*

31. Interrupted Aortic Arch and Ventricular Septal Defect: Significance of Subaortic Narrowing

Mark S. Bleiweis*, Adel K. Younoszai*, V. Mohan Reddy*, Olaf Reinhartz*, Antonio Laudito*, Leonardo D. Thompson*, Michael M. Brook*, and Frank L. Hanley, San Francisco, California
Discussant: Ralph Mosca, M.D.

OBJECTIVE: Left ventricular outflow tract (LVOT) management in patients with interrupted aortic arch (IAA) continues to be challenging and controversial. Intervention for enlargement of LVOT during primary repair of IAA was a risk factor for death in the Congenital Heart Surgeons Society study. We sought to determine the impact of LVOT narrowing on postoperative mortality, hemodynamic performance, and need for reintervention.

METHODS: Since 7/92, twenty-seven patients with IAA underwent repair at our institution. We retrospectively reviewed pre- and post-operative echocardiograms, operative variables, and followup data with emphasis on LVOT dimensions and anomalous subclavian artery (ASA) from the descending aorta. LVOT dimensions were indexed to body weight and surface area. Followup echocardiograms were reviewed for LVOT dimensions and morphology. Statistical analyses were performed to determine any significant correlations between LVOT dimensions and postoperative hemodynamics.

RESULTS: Twenty-five had Type B and 2 had Type A, and DiGeorge's syndrome was present in 20 patients. Twenty-four of 25 patients with Type B IAA underwent single-stage complete repair, and only two had concomitant subaortic muscle resection. In 9 recent patients, repair was done without circulatory arrest. Early mortality was 1/27 (3.7%). Thirteen of 26 survivors (50%) had LVOT gradients >20 mm Hg by doppler at a mean follow-up of 23 months (range from 1 to 57 months). Neither absolute subaortic diameter, subaortic diameter indices, nor presence of an ASA correlated significantly with followup LVOT gradient. Six patients required surgical reintervention for LVOT obstruction (4) and coarctation (2) with one death in a patient who required LVOT resection at primary repair, also.

CONCLUSIONS: IAA and VSD can be repaired with low operative mortality, even without circulatory arrest. Since absolute and indexed measures of the LVOT do not correlate with postoperative gradient, we advocate not performing any concomitant procedures to enlarge the LVOT at the initial operation, especially if subvalvar diameter is greater than 3 mm or greater than body weight in kilograms.

*By *Invitation*

32. Extra-Anatomic Aortic Bypass Via Sternotomy for Complex Aortic Arch Stenosis in Children

Kirk R. Kanter, Eldad Erez*, Willis H. Williams, and Vincent K. H. Tarn*, Atlanta, Georgia
Discussant: John J. Lamberti, M.D.

OBJECTIVE: Recurrent aortic narrowing following repair of aortic coarc-tation (CoA) or interrupted aortic arch (IAA) as well as diffuse, long-segment aortic hypoplasia can be very difficult to manage. Extra-anatomic ascending to descending aortic bypass grafting (EABG) through a Sternotomy is an alternative approach for this problem.

METHODS: Since 1985, 19 patients aged 2 months to 18 years (mean 10.7 years) underwent EABG using 10-30mm Dacron grafts. Initial diagnosis was CoA with hypoplastic arch in 14, IAA in 4, and diffuse long-segment aortic hypoplasia in 1. There were 20 previous operations in 17 children: transthoracic interposition graft (7), end-to-end anastomosis (6), subclavian arterioplasty (4), and synthetic patch (3). The mean time from initial repair was 7.5 years (range 0.6-18 years). Three children had previous ster-notomies. Cardiopulmonary bypass was avoided in all but 5 patients (3 with simultaneous intracardiac repairs).

RESULTS: There were no hospital or late deaths. On follow-up from 4 months to 14.7 years (mean 8.1 years) there were no reoperations for recurrent stenosis. One patient has mild systolic hypertension, two patients have arm to leg gradients: 20mmHg at rest in one and a 60mmHg systolic exercise gradient with no gradient at rest in the other. One patient required exclusion of an aortic aneurysm at the old CoA repair site 13 years after EABG. Three children had subsequent successful cardiac operations.

CONCLUSIONS: EABG is an effective and relatively easy approach for selected cases of complex or reoperative aortic arch obstruction in children with satisfactory results. EABG should be considered when complex arch reconstruction is necessary if collaterals may be inadequate or when an associated cardiac operation is necessary.

**By Invitation*

TUESDAY MORNING, MAY 2, 2000

33. A Case for Anatomic Correction in Atrioventricular Discordance? Effects of Surgery on Tricuspid Valve Function

Marjan Jahangiri*, Andrew N. Redington*, Martin J. Elliott*, Jaroslav Stark, Victor T. Tsang*, Marc R. de Leval†, London, United Kingdom.

Discussant: Tom R. Karl, M.D.

OBJECTIVE: To assess tricuspid valve function in atrioventricular discordance (AVD) following palliative procedures (pulmonary artery banding and Blalock-Taussig shunt) and corrective procedures (anatomic correction, AC; physiologic correction, PC).

METHODS: Tricuspid valve dysfunction was assessed by transthoracic echocardiography and graded into no regurgitation (0), mild (1), moderate (2)and severe (TR) before and after 150 operations performed in 99 patients with AVD who underwent surgery between 1988 and 1999. The ventricular arterial connection was discordant in 92% and double outlet right ventricle in 8%. 66% had a VSD and 28% had pulmonary stenosis. Twenty six patients underwent pulmonary artery banding and 25 had a modified Blalock-Taussig shunt performed. Eighty patients underwent PC and nineteen underwent AC (atrial-arterial switch, n=15; atrial-Rastelli, n=4).

RESULTS: Table I summarises patients with TR and the effect of surgery on this (3 in the PC group had tricuspid repair and 4 had replacement). The operative mortality in patients who underwent PC was 7% as compared to no death in the AC group (p=0.59).The median follow-up was 3.2 years (range; 3 months-10.2 years).

CONCLUSIONS: Volume loading (shunt) or right to left septal shift (PC) worsens TR whereas volume reduction (banding) or left to right septal shift (AC) has beneficial effects on tricuspid valve

function. Anatomic correction can be performed with a low morbidity and mortality in selected patients with AVD and provides superior functional result.

Operation, N	Preop TR Score mean±SD	Postop TR Score mean±SD	p-value
Pulmonary Artery Banding, 20	1.83±0.72	0.86±0.55	<0.001
Blalock-Taussig Shunt, 16	1.59±0.80	2.42±0.61	<0.001
PC, 27	1.50±0.69	1.05±0.94	NS
AC, 15	1.59±0.87	0	<0.001

†Graham Fellow, 1973-1974
**By Invitation*

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

Supported by an unrestricted educational grant from St. Jude Medical, Inc.
Constitution Hall, Metro Toronto Convention Centre
(8 minute presentation, 7 minutes discussion)
Moderators: Eric A. Rose, M.D.
Edward D. Verrier, M.D.

L1. Gene Transfer of Bcl-2 Does Not Affect Myocardial Stunning But Ameliorates the Deletorious Effects of Chronic Remodeling

Allan S. Stewart*, Henry L. Zhu*, Derek R. Brinster*, Hugh L. Sweeney*, and Timothy J. Gardner, Philadelphia, Pennsylvania

OBJECTIVE: Numerous studies implicate apoptosis as an important consequence of ischemia/reperfusion injury. However, no study reproducibly associates the reduction of apoptosis with an improvement in post-ischemic myocardial function. Data is lacking to determine if apoptosis is advantageous to organ survival or deleterious to myocardial function. This experiment employs gene transfer of bcl-2 to significantly reduce apoptosis and correlate that reduction with acute and chronic measurements of contractility.
METHODS: An adenovirus encoding for bcl-2 was constructed and injected into the lateral wall of 20 New Zealand white rabbits. 20 rabbits recieved adenolac-Z as a control. Five days post-injection, the rabbits were subjected to 30 min or proximal circumflex occlusion followed by reperfusion. 10 rabbits in each group underwent four hours reperfusion, while the remaining underwent 6 weeks of reperfusion. Functional measurements were obtained with echocardiography, aortic flow probe measurements, and sonomicroscopy. Infarct percentage was assessed with TTC staining. Histological analysis was performed with HandE, trichrome staining, and TUNEL assay. Gene expression was assessed with Western blot and RT-PCR.
RESULTS: Bcl-2 reduced the percentage of apoptotic cells from 19.2±3.5% to 4.1±1.7% ($p<.05$). However, this decrease did not result in a significant improvement in contractility, ventricular stroke work, or ejection fraction in the acute group. In contrast, bcl-2 was found to significantly improve regional wall motion, ejection fraction, stroke work, and enhanced diastolic relaxation in the chronic group. A decrease in fibrosis, cell-cell slippage, and ventricular wall thickness was seen in the bcl-2 chronic group, but not seen in the acute group.
CONCLUSIONS: Apoptosis was demonstrated after reperfusion injury but had no influence on post-ischemic myocardial stunning. However, apoptosis was found to adversely influence chronic remodeling and ventricular function. Gene transfer of bcl-2 may be a useful strategy to protect the heart from the deletorious consequences of apoptosis induced remodeling.
**By Invitaiton*

L2. Epidermal Growth Factor Augments Post-Pneumonectomy Lung Growth

Aditya K. Kaza*, John A. Kern*, Stewart M. Long*, Victor E. Laubach*, Joshua A. Tepper*, Kimberly S. Shockey*, Curtis G. Tribble, and Irving L. Kron, Charlottesville, Virginia

OBJECTIVE: We hypothesized that post-pneumonectomy compensatory lung growth can be augmented by the administration of exogenous epidermal growth factor (EGF).
METHODS: Adult Sprague-Dawley rats were divided into three groups. Sham left thoracotomy was performed in the first group (C), left pneu-monectomy in the second group (P), and left

pneumonectomy with administration of EGF (0.2ug/g, at 72 hour intervals) in the third group (P¢). The right lung growth was studied in each group at 1, 3, 5, 10 and 21 days after surgery. Wet lung weights were measured. Volumetric analysis was performed using saline displacement technique after intra-tracheal instillation of 2% glutaraldehyde to a pressure of 25cm. Lung weights (g) and volumes (cc) were expressed as a ratio to the total body weight (kg) (lung weight and volume indices).

RESULTS: Using ANOVA, we noted a significant increase in lung weight index between the P and P' group at 21 days (3.61 vs 4.62, p=0.006). Contrast analysis also revealed a significant increase in lung weight index between P and P' at 3 days (2.75 vs 3.08, p=0.034). Lung volume index was evaluated using ANOVA, which showed significant increase in right lung volume between the P and P' groups at 5 (15.09 vs 16.98), 10 (18.81 vs 24.48) and 21 (21.01 vs 28.54) day intervals ($p<0.001$).

CONCLUSIONS: This study demonstrates that administration of exogenous epidermal growth factor has a significant impact on post-pneumonectomy lung growth. This process may be mediated by an up-regulation of growth factor receptor expression in the contra-lateral lung after pneumonectomy. We believe that this is the first demonstration that adult lung growth can be exogenously enhanced.

**By Invitation*

L3. Early Sustained Reduction of Pulmonary Angiotensin-Converting Enzyme Activity Following Superior Cavopulmonary Anastomosis in the Lamb

Sunil P. Malhotra*, V. Mohan Reddy*, Frank L. Hartley and Kirk Riemet, San Francisco, California

OBJECTIVE: The Glenn shunt is a superior cavopulmonary anastomosis (SCPA) widely used for palliation of various forms of congenital heart defects. However, pulmonary arteriovenous malformations (PAVMs) of varying clinical significance develop in 15-60% of patients following surgery. Histological analysis of these PAVMs reveals the proliferation of numerous dilated, thin-walled vessels. To assess alterations in regulators of vascular tone following SCPA, changes in angiotensin-converting enzyme (ACE) activity and plasma angiotensin II (AT-II) levels were examined.

METHODS: Lambs, aged 30-40 days, underwent an end-to-end anastomosis of the superior vena cava to the right pulmonary artery. In age matched controls, a sham operation was performed. PAVMs developed in all SCPA lambs by 6 weeks after surgery, as demonstrated by contrast echocardiography. Animals (n=16) were then studied at various time points following surgery. ACE activity was measured in lung homogenates. Levels of AT-II in the right pulmonary vein were measured by ELISA.

RESULTS: Compared to controls, ACE activity in the right lung of Glenn animals was reduced 86% at 4 and 14 days, 52% at 50 days, and 13% at 133 days following surgery. This correlated with a 70% reduction in AT-II levels in SCPA animals studied at 4-14 days following surgery, while levels at 50 and 133 days approached control levels.

CONCLUSIONS: ACE activity is an indicator of endothelial integrity. Diminished activity following SCPA suggests pulmonary endothelial damage. Moreover, the resulting decrease in AT-II production, a pulmonary vasoconstrictor, may promote chronic dilatation of the right pulmonary vascular bed. The role of these perturbations of the affected vasculature in PAVM formation remains to be determined.

**By Invitation*

L4. Should Mediastinal Drainage Be Autotransfused Postoperatively in the Cardiac Surgery Patient?

John S. Thurber*, Edward R. Zech*, Loretta Aiken*, and Gary H. Meyers*, Bethesda, Maryland

OBJECTIVE: Autotransfusion (AT) of mediastinal drainage in the post-operative cardiac surgery patient has been a method of perioperative blood conservation for over 20 years. The risks and

benefits of this practice have been reviewed in a number of reports, with conflicting results. This study prospectively evaluates the risks/benefits of the use of AT.

METHODS: A prospective, randomized study of 128 patients undergoing elective cardiac surgery was performed. Patients were randomized into one of two groups: the experimental group received autotransfused mediastinal drainage (AT) for 6 hours after surgery (n=62), and the control group was treated with standard chest drainage with no AT (n=66). Both groups received homologous blood transfusion when the hemoglobin fell to less than 8.0 g/dl. Pre- and post-operative variables recorded included: hematocrit, platelets, PT/PTT, fibrinogen and d-dimer levels, and homologous blood products infused.

RESULTS: Packed red blood cells were required in 9 of 62 (15%) patients in the AT group, and in 14 of 66 (22%) patients in the non-AT group (*p*=ns). Total homologous blood product exposure was slightly higher in the non-AT group (25% vs. 19% AT, *p*=ns). Pre- and post-operative hematologic parameters were similar between the two groups. D-dimers were elevated in the serum of 11% of AT patients, compared with 3% of non-AT patients (p=0.03). There was an increased cost for nursing effort and equipment involved with the AT patients.

CONCLUSIONS: The use of AT in the postoperative cardiac surgery patient did not result in significant reduction in the use of homologous blood products, and did result in increased cost. Therefore, in the setting of routine blood conservation practices, the postoperative use of AT in the cardiac surgery patient is not recommended.

**By Invitation*

L5. Subdiaphragmatic Venous Hemodynamics in the Fontan Circulation

Tain-Yen Hsia*, Sachin Khambadkone*, Francesco Migliavacca*, and †Marc R. de Leval, London, United Kingdom

OBJECTIVE: To investigate Subdiaphragmatic venous physiology in Fontan patients (FP) in order to understand some of the early and late problems and to improve their management.

METHODS: Doppler flow were evaluated in subhepatic inferior vena cava (IVC), hepatic vein (HV) and portal vein (PV) with respiratory monitoring and a tilt table to assess effects of respiration and gravity. 19 controls (group A) and 44 FP, 29 in functional class 1 (group B)and 15 in class 3 (group C), were studied. IVC, HV and wedged-HV (WHV) pressures were measured during catheterization in 11 controls and 8 class 3 FP. Difference between HV and WHV is the transhepatic venous pressure gradient(TVPG).

RESULTS: Shown below, ratio of inspiratory/expiratory antegrade flows (R)represented effect of respiration. Gravity effect was evaluated by a ratio of flow rates in supine/upright positions (G). * denotes p-values <0.05, † <0.0001, # =0.07.

CONCLUSIONS: This is the first time hydrostatic force have been evaluated in FP. Gravity reduced class 1 FP's IVC and HV flow; progression to class 3 did not exacerbate this. In the PV, while FP have lost normal expiratory augmentation to flow, gravity more adversely influenced class 3 than class 1 FP. This poorer flow dynamics is coupled to higher splanchnic pressures and a lower gradient. Reduced TVPG in class 3 FP further suggests the hepatic sinusoidal reserve is impaired, creating an open tube phenomenon. These observations may account for some late gastrointestinal problems in FP.

FlowRatio	R (IVC)	R(HV)	R(PV)	G (IVC)	G(HV)	G(PV)
Avs.B	1.2 v 1.6	1.7v 2.9*	0.8 v 1.0*	1.2 v 1.8*	1.7 v 2.3#	1.9 v 2.1
Bvs.C	1.6 v 1.5	2.9 v 3.1	1.0 v 1.1	1.8 v 1.7	2.3 v 2.4	2.1 v 3.1*
Pressures mm Hg	IVC	HV	WHV	TVPG		
Control vs Fontan	6.2±2.0 v 13.7±3.7†	5.9±1.9v 15.7±5.0†	8.4±2.9 v 15.0±4.0†	2.5±2.4 v 0.5±0.7 *		

†1973-74 AATS Graham Fellow

**By Invitation*

L6. Laparoscopic Gastric Ischemic Preconditioning Prior to Transhiatal Esophagectomy

Sandra M. Jones*, Daniel Gagne*, Mary Beth Malay*, Dennis L. Fowler*, Robin S. Macherey* and Rodney J. Landreneau, Pittsburgh, Pennsylvania

OBJECTIVE: Cervical esophagogastric anastomotic disruption following THE is a significant problem. Ischemia of the "proximal gastric tip" is a primary cause of anastomotic failure. We sought to determine if gastric blood flow could be improved with the preoperative performance of LAP ischemic preconditioning, by selectively ligating the short gastric (SG) or the left and short gastric (LG/SG) blood supply to the stomach 3 weeks prior to THE.

METHODS: Fifteen 25 kg mongrel dogs underwent a 2 stage experiment of LAP followed 3 weeks later by THE. Prior to each stage, hemodynam-ics were stabilized. Blood flow was assessed using the fluorescent microspheres method. Three groups were separated into 5 dogs each. *Group 1* LAP alone, *group 2* LAP/ligation of SG only, *group 3* LAP/ligation of LG/SG. Microsphere injection occurred prior to pneumoperitoneum and at completion of LAP. All 15 dogs underwent THE 3 weeks later. Microsphere blood flow injection was made after anesthesia and after esophago-gastic anastomosis. All animals were euthanized and gastric perfusion (near anastomosis) was analyzed. Differences in blood flow were evaluated using Student's T test.

RESULTS: The mean baseline blood flow was 0.58ml/mg tissue. After THE, proximal gastric blood flow fell to 19% of baseline(0.11ml/mg) in the control *(group 2),* to 31%(0.18ml/mg) in SG *(group 3),* and to 59% (0.34ml/ mg)in LG/SG *(growp 3)*. This relative preservation of blood flow among the LG/SG group was significant compared to the control *group* 1(0.11ml/ mg vs 0.34ml/mg, p=0.02)Preoperative ligation of SG vessels alone *(group2)* did not provide significant "ischemic conditioning" improvement in proximal gastric blood flow following THE.

CONCLUSIONS: Ischemic preconditioning of the proximal stomach during preoperative LAP can significantly improve blood flow to the "gastric tip" prior to THE. Future consideration of this procedure during LAP staging of esophageal carcinoma may be considered to reduce anastomotic complications following THE.

**By Invitation*

§L7. Assisted Venous Drainage Presents Risk of Undetected Air Microembolism

Angelo LaPietra*, Eugene A. Grossi, Bradley A. Pua*, Rick A. Esposito*, Aubrey C. Galloway, Christopher C. Derivaux*, Lawrence Classman* and Stephen B. Colvin, New York, New York

OBJECTIVE: Methods for minimally invasive cardiac surgery rely on augmented venous return (AGVR) techniques (kinetic or vacuum) for cardiopulmonary bypass. Such techniques can introduce venous air emboli (AE) which can pass to patients. We examined this potential with different AGVR techniques.

METHODS: An in vitro bypass system was created using kinetic (Biomedicus Pump) (K-AGVR) or vacuum (hardshell reservoir) (V-AGVR) systems. Roller or centrifugal pumps were used on the arterial side with a fiber oxygenator and a 37m arterial filter. Air was introduced into the venous line via an open 25g needle. Test conditions included varying the amount of venous negative pressure (-15 to 75mmHg), AGVR type, and arterial pump (AP).

RESULTS: Changes in negative venous pressure did not affect the number of microbubbles introduced into the system. K-AGVR filled quickly with micro and macro bubbles requiring manual clearing. Microbubbles/ min (mean±SD) are shown below for the venous inlet, pre-oxygenator, and patient side of the arterial filter.

CONCLUSIONS: Some AGVR configurations permit a significant quantity of microbubbles to reach the patient despite filters. Centrifugal pump has air handling disadvantages when used for K-AGVR, but aids in clearing AE when used as the arterial pump. The surgeon using these techniques should be aware of the potential risks and how to minimize them.

	Venous Inlet	Pre-Oxygenator	Patient Side
AP: Roller			
V-AGVR	9754±2898	16±21	010
K-AGVR	10004±1258	14218±905	817
AP: Centrifugal			

V-AGVR	8639±2987	249±690*	010
K-AGVR	9806±2758	1317±3311*	0+0

[§]Authors have a relationship with Heartport, Baxter Healthcare, St. Jude Medical & Medtronic
**By Invitation*

L8. Hemodynamic Changes During Beating-Heart CABG Surgery

Quoc-Bao Do*, Olivier Chavanon*, Pierre Couture*, Andre Denault*, Raymond Carrier*, Montreal, PQ, Canada.

OBJECTIVE: To study the effect of different manipulations during beating-heart CABG, we monitored the systemic arterial pressure (SAP), the pulmonary arterial pressure (PAP), the mixed venous oxygen saturation (SvO_2) and the cardiac output (COI)on 54 patients who underwent complete revascularization. Five patients also had a transoesophageal echocardiography (TEO) to assess mitral valve dynamics and ventricular function. Mean patient's age was 66.4±9.2 years, and 3.3±0.8 distal anastomosis were performed per patient.

METHODS: Stabilization of the heart were done using a "fork-type" stabilizator, and the target coronaries were clamped proximally and distally to the anastomosis site without preconditioning.

RESULTS: Changes in SAP, PAP, SvO_2 and COI, as shown in this table, occurred during the stabilization period preceding coronary anastomosis.

CONCLUSIONS: The mobilization and stabilization of the heart rather than clamping the coronaries, were responsible for some minor hemody-namic changes during beating-heart CABG surgery. The marked elevation of PAP during LAD and DG revascularization suggests that compression of left ventricle outflow tract may be the cause.

	LAD	DG	MG	RC(PDA)
Clamp (min)	9.5±0.4	7.4±0.3	8.3±0.4	8.6±0.6
Î”SAP (%)	-8.6±2.9	-14.0±4.9	-16.3±2.0	-15.1±2.5
Î”PAP (%)	23.9±4.6	37.7±14.4	12.5±5.3	12.7±3.8
Î”Sv0$_2$(%)	-8.5±1.2	-8.1±0.7	-6.5±1.4	-9.6±0.6
Î”COI (%)	-4.2±2.9	4.5±2.8	-6.3±1.9	-2.6±1.7

No correlation between $Sv0_2$, COI, SAP, PAP and clamping time were found. They were no significant mitral regurgilation on TEO, although some diastolic and systolic regional dysfunction were found during left anterior (LAD) and the diagonal (DG) coronary clamping.

**By Invitation*

9:00 a.m. SCIENTIFIC SESSION

Constitution Hall, Metro Toronto Convention Centre

Moderators: Delos M. Cosgrove, M.D.
Tirone E. David, M.D.

34. Late Results of Aortic Valve Sparing Operations

Tirone E. David, Susan Armstrong*, Joan Ivanov*, Christopher M. Feindel, and Gary Webb*, Toronto, ON, Canada

Discussant: Magdi H.Yacoub, M.D.

OBJECTIVE: To determine the late results of aortic valve sparing operations in patients with aortic root and/or ascending aortic aneurysms.

METHODS: All patients with aortic root and/or ascending aortic aneurysms who had aortic valve repair have been followed prospectively at annual intervals. The mean age of 161 consecutive patients operated on from July 1987 to June 1999 was 54+17 years, range 16 to 84 years. Forty-two patients had the Marfan syndrome according to Gent criteria. Thirty-one patients had type A aortic dissection, 15 had mega-aorta syndrome, 34 had coronary artery disease and 9 had mitral regurgitation. The technique of reimplantation of the aortic valve was performed in 48 patients and remodeling of the aortic root in 113. The follow-up was complete and ranged from 0 to 134 months, mean of 34±28.

RESULTS: There were 3(2%) operative and 16(10%) late deaths. Cardiovascular events were the cause of death in 15 of 19 patients. Actuarial survival at 10 years was 80%±5% for all patients and 100% for those with the Marfan syndrome. Aortic dissection and mega-aorta syndrome were independent predictors of death. The most recent Doppler echocardiogram showed trace or no

aortic insuffiency (AI) in 77 patients, mild in 70, moderate in 8, and severe in 3. The 3 patients with severe AI were reoperated on uneventfully. The freedom from moderate or severe AI was 85%±5% at 10 years. Aortic root remodeling and the need for repair of cusp prolapse were associated with a higher risk of AI (p=0.03). The freedom from reoperation was 98%±1% at 10 years.
CONCLUSIONS: Aortic valve sparing operations to treat aortic and/or ascending aortic aneurysms provide excellent and lasting functional results in patients with normal aortic cusps including those with the Marfan syndrome.
**By Invitation*

35. Repair Is Preferable to Replacement for Ischemic Mitral Regurgitation

Per Nils Wierup*, A. Marc Gillinov*, Eugene H. Blackstone, Delos M. Cosgrove, Ehab S. Bishay* and Patrick M. McCarthy, Cleveland, Ohio
Discussant: D. Craig Miller, M.D.

OBJECTIVE: To determine whether mitral valve (MV) repair is preferable to MV replacement for ischemic mitral regurgitation (MR).
METHODS: From 1985 to 1997, 402 patients (pts) with ischemic MR underwent either MV repair (n=339) or MV replacement (n=63), Myocardial infarction (MI) was acute in 24% and remote in 76%. By multivariable logistic regression, pts were more likely to receive a repair if they were male *(P=.04),* had chronic ischemic MR *(P=.001),* underwent non-emergency operation *(P=.002),* had restricted leaflet motion *(P<.0001),* or underwent ITA grafting *(P=.002).* These factors were used for propensity matching. Factors associated with early and late mortality were identified by multivariable, multi-phase hazard function analysis.
RESULTS: Hospital mortality was 11%. The timing of MI and operative strategy influenced hospital mortality (table). In propensity-matched pts, survival after MV replacement was 76%, 60%, and 43% after 30 days, 1 year (yr), and 5 yrs. In contrast, survival after MV repair was 91%, 80%, and 60% at these same time intervals *(P=.003).* Risk factors for death within the first yr of operation included older age *(P=.002),* greater wall motion abnormality *(P=.006),* and replacement rather than repair *(P=.0005).* All pts were predicted to benefit from repair; however, the benefit became more pronounced with advancing pt age and less apparent in pts with more severe heart failure *(P=.001).* Freedom from repair failure was 93% at 5 yrs.
CONCLUSIONS: For surgical management of ischemic MV regurgitation, MV repair is the treatment of choice.

		Hospital Mortality (%)			
Time of Infraction	**Repaired (%)**	**Total**	**Repair**	**Replace**	**P value (repair vs. replace)**
Acute	71	22	17	35	.1
Remote	87	8	5	30	<.001
P value	<001	.002	.009	.7	

**By Invitation*

36. Induction Chemoradiation Plus Surgical Resection Is Feasible and Highly Effective Treatment for Pancoast Tumors: Initial Results of SWOG 9416 (Intergroup 0160) Trial

Valerie W. Rusch, John J. Crowley*, Michael J. Kraut* and David R. Gandara*, New York, New York; Seattle, Washington; Detroit, Michigan; Sacramento, California
Discussant: Douglas J. Mathisen, M.D.

OBJECTIVE: Rates of complete resection (50%) and 5 year survival (30%) for Pancoast tumors have not changed for 30 years. However, combined modality therapy has improved outcome in other Stage HI non-small cell lung cancers. This prospective intergroup trial tested the feasibility of concurrent inducton chemoradiation and surgical resection in mediastinoscopy negative Pancoast tumors with the ultimate objective of improving resectability and overall survival.
METHODS: Patients with pathologically proven T3-4 N0-1 Pancoast tumors received 2 cycles of cisplatin and etoposide chemotherapy concurrent with 45 Gy radiation. In patients with no evidence

of disease progression thoracotomy was performed 3-5 weeks later. Two cycles of chemotherapy were given postoperatively.
RESULTS: From 4/95-9/99, 116 patients were entered on study. This analysis includes 101 eligible patients, 71 men and 30 women with a median age of 56 yrs. Induction therapy was completed as planned in 93% patients with 2 Grade 5 and 17 Grade 4 toxicities (predominantly cytopenia). To date, 81 patients have undergone thoracotomy with the most common procedure being lobectomy + chest wall resection. 1 patient died postoperatively. A pathologic complete response (pCR) occurred in 57.5% patients and 63% rumors were downstaged. At 1 year overall survival was 77% for T3, 80% for T4 tumors; at 3 years 50% for both T3 and T4 tumors. Most common site of relapse was the brain.
CONCLUSIONS: 1) This combined modality approach was highly feasible in a multi-institutional setting; 2) pCR rates were unexpectedly high; 3) resectability and overall survival are improved compared to historical controls; 4) improved outcome was especially notable for T4 tumors which usually have a grim prognosis.
**By Invitation*

TUESDAY AFTERNOON, MAY 2, 2000

37. Critical Aortic Stenosis in the Neonate: a Multi-Institutional Study of Management, Outcomes and Risk Factors

Gary K. Lofland*, Brian W. McCrindle*, William G. Williams, Eugene H. Blackstone, Christo Tchervenkov, Richard A. Jonas, and the members of the CHSS*, Kansas City, Missouri; Toronto, ON, Canada; Cleveland, Ohio; Montreal, PQ, Canada; Boston, Massachusetts

Discussant: Frank L. Hanley, M.D.

OBJECTIVE: To determine outcomes and associated risk factors of different management strategies in the treatment of critical aortic stenosis (CAS) in neonates.

METHODS: Data regarding 285 cases (22 institutions) diagnosed within 30 days of birth from 1994 to 1999 were submitted to the CHSS Data Centre, and analyzed with parametric time-dependent event analysis in the hazard domain. Patients with aortic valve atresia, abnormal atrio-ventricular and ventriculo-arterial connections, or isolated aortic arch lesions were excluded.

RESULTS: Nineteen of 20 patients with no CAS-related intervention died (95%). Initial CAS-related procedure indicated a biventricular route (BVR) consisting of aortic valvotomy in 102 cases (31 died; 30%) or a "single ventricle" route (SVR)in 163 cases (59 died; 36%)(initial heart transplantation in 7, Norwood operation in 156). Anatomic characteristics completely discriminated BVR from SVR cases. Overall, survival in those having initial CAS-related intervention was 83% at 1 month after entry, 69% at 6 months, 66% at 1 year and 64% at 5 years. There was no significant difference in time-related death between BVR and SVR (survival at 5 years, 68% vs. 61%, respectively; p=0.36). Independent incremental risk factors for mortality included younger age at entry, non-Caucasian race, higher grade of MV hypoplasia, presence of LV endocardial thickening, and the presence of aortic coarctation (CoA). In SVR, incremental risk factors for mortality included non-Caucasian race, MV stenosis, and presence of VSD or CoA. In BVR, surgical valvotomy was associated with higher mortality than balloon valvotomy, but this was not significant after adjustment for anatomic complexity. Incremental risk factors for mortality

in BVR included non-Caucasian race and higher grade of MV and LV hypoplasia. Freedom from CAS-related reintervention in BVR was 67% at 6 months and 44% after 5 years from initial valvotomy, with no difference between surgical vs. balloon valvotomy.

CONCLUSIONS: Neonatal CAS continues to be associated with a high mortality with both BVR and SVR. Better patient selection for both approaches may improve outcomes.

PRESENTATION OF SCIENTIFIC ACHIEVEMENT AWARD
Denton A. Cooley Houston, Texas

10:20 a.m. INTERMISSION - VISIT EXHIBITS

11:00 a.m. BASIC SCIENCE LECTURE:

Decoding the Human Genome

J. Craig Venter, Ph.D.

11:30 a.m. ADDRESS BY HONORED SPEAKER:

Effects of the Net Economy
James L. Barksdale

12:15 p.m. ADJOURN FOR LUNCH - VISIT EXHIBITS

12:30 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

Metro Toronto Convention Centre, Summit Room

**By Invitation*

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - 2 ADULT CARDIAC SURGERY

Constitution Hall, Metro Toronto Convention Centre

Moderators: D. Craig Miller, M.D.

Timothy J. Gardner, M.D.

38. Early Discharge Following Coronary Artery Bypass Graft Surgery: Cost Savings or Cost Shifting?

Harold L. Lazar, Carmel A. Fitzgerald*, Tazeen Ahmad*, Yusheng Bao*, Oz M. Shapira*, and Richard J. Shemin, Boston, Massachusetts

Discussant: Richard D.Weisel, M.D.

OBJECTIVE: Changes in reimbursement policies and rising health care costs have resulted in shorter hospital length of stay(LOS) after Coronary Artery Bypass Graft(CABG) surgery. This study was undertaken to determine whether early discharge following CABG surgery resulted in cost savings or merely cost shifting by increasing the utilization of outpatient nursing and inpatient rehabilitation services.

METHODS: Patterns of discharge were analyzed in 330 patients undergoing CABG in 1990 (Group I) when there were no early extubation or fast track protocols and compared to 334 CABG patients in 1998(Group II) when these protocols were utilized.

RESULTS: Age, gender, angina class, ejection fraction, the incidence of diabetes, the number of vessels bypassed and the need for inotropic or mechanical support were similar between the groups.

There was no difference in 30 day mortality, periop MI, infection, strokes or pneumonia. Group II patients spent less time on the ventilator(17.2 ±16.9 SD hours VS 10.219.5, P<0.001), had a lower incidence of reoperation for bleeding(0.5% VS 2.7%; P< 0.03), and had a shorter LOS (5.512.5 days vs 8.613.1; P<0.0001). However, fewer Group II patients were discharged home(55.3% VS 95.1%; P<0.000001). Furthermore more Group II patients went home with VNA services(79.6% VS 7.5%; P<0.0001). A higher incidence of GROUP II patients (44.3% VS 2.4%; P<0.0001) were discharged to rehab facilities where their average LOS was 11.5±15.9 days. The need for readmission to acute care facilities was also increased in Group II patients(5.3% VS 0.6%; P<0.0001).

CONCLUSIONS: Although early extubation and fast track protocols result in earlier discharge from acute care facilities, the anticipated savings may be offset by cost shifting due to increased (1) utilization of outpatient nursing services (2) discharges to rehab facilities and (3) hospital readmissions.

**By Invitation*

39. Selection of a Cardiac Surgery Provider in the Managed Care Era

Winnie Yip*, David M. Shahian, Jerilynn Jacobson* and George A. Westcott*, Burlington and Boston, Massachusetts

Discussant: David F. Torchiana, M.D.

OBJECTIVE: Health planners predict that managed competition and the availability of outcome data should lead to more "rational" provider selection. Using a standard econometric model, we examine this hypothesis in the context of cardiac surgery, where there are profound implications for national health expenditures and quality.

METHODS: McFadden's conditional logit model was used to study the determinants of cardiac surgery provider selection among 6952 patients in the metropolitan Boston market (8 hospitals within a 15 mile radius) during fiscal year 1997. Hospital variables included beds, annual cardiac case volume, acuity-adjusted clinical and financial performance for the 3 preceding years (mean mortality, length of stay[LOS], charges, and cost), reputation markers (percent out-of-state referrals, cardiac residency program), and distance from the center of each hospital's zip code to the center of each patient's zip code. Patient variables included DRG, age, acuity level, and payer.

RESULTS: In all models, proximity of patient to hospital was the most stable and consistent predictor of choice (OR 0.89, p = 0.000). A cardiac surgery residency program significantly enhanced the probability of selection (OR 3.60, p = 0.000) as did percent out-of- state referrals (OR 1.06, p = 0.124). Higher adjusted mortality rates led to decreased probability of selection (OR 0.566, p = 0.113) but higher LOS was paradoxically associated with greater probability (OR 1.15, p = 0.193). Neither average hospital charges nor costs had any relationship to the probability of selection (OR 1.000, p = 0.989). Subgroup analysis by payer type revealed a striking increase in the preference for a teaching hospital among patients with commercial insurance (OR 19.08, p = 0.001). Non-Medicare managed care patients were the only subgroup in which higher mortality rate hospitals were more likely to be chosen (OR 3.34, p = 0.088).

CONCLUSIONS: Even within a competitive metropolitan market with advanced managed care penetration, the major observable determinants of cardiac surgery provider selection are hospital "reputation" and proximity to the patient's home, not objective clinical or financial performance.

**By Invitation*

40. Reoperative Coronary Bypass Surgery: Effect of Patent Grafts on Perioperative Outcomes

Michael A. Borger*, Vivek Rao*, Richard D. Weisel, Alex Floh*, Gideon Cohen*, Christopher M. Feindel and Terrence M. Yau*, Toronto, ON, Canada

Discussant: Hendrick B. Earner, M.D.

OBJECTIVE: Patent grafts may increase the risk of reoperative coronary bypass surgery, an effect which may be mitigated by the use of retrograde cardioplegia. We attempted to determine the effects of patent grafts and retrograde cardioplegia on operative mortality.

METHODS: Systematic review of all redo coronary bypass patients (REDO, n = 744) at our institution from 1990-97. Independent predictors of operative mortality (OM) were determined with stepwise logistic regression analysis.

RESULTS: OM occurred in 42 patients (5.7%). Fifty percent of REDO patients had one or more patent grafts to the LAD, 33% to the RCA territory, and 27% to the circumflex territory. The previous LAD graft conduit was a saphenous vein in 82% and a LIMA in 18%. Patent grafts were injured in 14 patients (1.9%). Patent LAD grafts at the time of REDO did not result in a significant increase in the risk of OM (see Table), nor did patent grafts to the RCA or circumflex territory. Independent predictors of OM were age, NYHA class, LV grade, peripheral vascular disease, and failure to use retrograde cardioplegia (RETRO). RETRO was used in 40% of patients, and resulted in a significant decrease in OM (p = 0.02). Patients with stenosed LAD grafts seemed to receive the largest benefit from RETRO (OM 4% vs 10% without RETRO, p = 0.05).

CONCLUSIONS: We were unable to demonstrate an increased risk of operative mortality in redo patients with patent grafts. We strongly recommend the use of retrograde cardioplegia in reoperative coronary bypass surgery, particularly in patients with diseased LAD grafts.

Graft to LAD	Operative Mortality
None	10.7%
Stenosed	7.3%
Patent	4.7%
Occluded	3.8%

41. Long-Term Angiographic Follow-up of Complementary Saphenous Vein Grafting

Robert A. Dion*, David Glineur*, David Derouck*, Robert Verhelst*, Philippe Noirhomme*, Gebrine El Khoury* and Claude Hanet*, Brussels, Belgium

Discussant: Stephen E. Fremes, M.D.

OBJECTIVE: In order to achieve complete myocardial revascularisation, saphenous vein grafting (SVG) is still frequently used in addition to arterial grafting. We wanted to know the angiographic patency rates of complementary SVG after 10 years or more.

METHODS: Five hundred patients having received sequential internal thoracic artery grafting and complementary SVG between 1985 and 1991 were recently reviewed. Age averaged 61 years, 53 had a LVEF < 40%, 117 were operated in emergency, there were 35 reoperations. In total 2,156 distal anastomoses were constructed (4.3/patient), of whom 1,367 arterial (2.7/pt) and 789 venous (1.6/pt). Only 10.7% of the later were constructed on the LAD. The follow-up is 97.4% complete and averages 9.6 years. One hundred sixty-one patients consented to a late angiographic restudy after a mean interval of 7.4 y (max 12.2 y)

RESULTS: At 5 and 10 years, 94% and 77% of the patients remained free of cardiac events. Only 15 pts required an iterative revascularisation (CABG 4, PTCA 11), that is 0.3%/pt/year. Overall 428/448 arterial anastomoses (95.5%) and 153/211 venous anastomoses (72.5%) were patent, p <0.001. The sequential venous anastomoses remained strikingly more patent than the single grafts: 126/166 (76%) versus 27/45 (60%), p= 0.04. There was no difference in patency between the latero-lateral (diamond-shaped or not) and the termino-lateral (T or not) sequential anastomoses. There was no significant difference in patency between the anastomoses sequential or not directed to the LAD, Circumflex and right coronary artery areas: 16/19 (84.2%), 55/83 (66.3%) and 82/109 (75.2%), Pearson p = 0.2. Diabetes had no influence on patency rates neither overall (27/39, 69.2% versus 126/172, 73.2%: p = 0.8) nor for the sequential anastomoses (104/135, 77% versus 22/31, 71%: p = 0.5).

CONCLUSIONS: Complejmentary *sequential* venous grafting yields surprisingly high long term patency rates (76%). We could not find any influence of diabetes on the patency rates.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:25 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - 2 ADULT CARDIAC SURGERY

Constitution Hall, Metro Toronto Convention Centre

Moderators: D. Craig Miller, M.D.

Timothy J. Gardner, M.D.

42. Myocardial Revascularization on the Beating Heart After Recent Onset of Acute Myocardial Infarction.

Giuseppe D'Ancona*, Hratch L. Karamanoukian*, Marco Ricci*, Reginald Abraham*, Jacob Bergsland* and Tomas A. Salerno, Buffalo, New York

Discussant: Gerald D. Buckberg, M.D.

OBJECTIVE: Coronary artery bypass grafting (CABG) after the recent onset of acute myocardial infarction (AMI), is associated with high morbidity and mortality. Revascularization without cardiopulmonary byapss (CPB), has been used to treat such patients (pts).

METHODS: From January 1995 to June 1999, 518 pts underwent CABG after recent AMI (1-20 days): CPB was used in 421 pts (Group A) and 97 pts (Group B)were operated without CPB. Preoperative risk factors were significantly (P<0.05) higher in Group B (redo, CHF, stroke, extensively calcified aorta,dialysis, evidence of left ventricular hypertrophy). Preoperative use of intra-aortic balloon pump (IABP) (5.2 vs. 2.4% P=NS) and emergent operations (5.2vs.2.6% P=NS) were similar in both groups. Mean number of grafts per pt was 3.46 in Group A vs. 1.82 in Group B (P=NS).

RESULTS: Crude mortality was 2.9% in Group A vs. 6.2% in Group B (P=NS). Major complications were comparable in the groups. Using univariate analysis mortality was found to be correlated to preopertive CHF, preoperative use of IABP, ventricular hypertrophy, advanced age, postoperative sepsis, and sternal infection. Multivariate analysis showed that only advanced age, postoperative sternal infection and sepsis, preoperative hemodynamic instability and evidence of left ventricular hypertrophy were positively related to death. Use or avoidance of CPB were not correlated to mortality when univariate or multivariate analysis was performed. Postoperative transmural AMI was positively related in univariate analysis to global ischemic time, preoperative HTN, female sex, use of warm cardioplegia and postoperative sepsis. Using logistic regerssion, global ischemic time was still correlated to postoperative trans-mural myocardial infarction together with preoperative HTN. The number of grafts was not correlated to postoperative AMI.

CONCLUSIONS: Multivariate analysis of a cohort of pts with recent AMI demonstrates that CABG can be performed with equal efficacy with oru without CPB. While CPB is not correlated to mortality, myocardial ischemic time (ie. cross-clamp time) is an independent risk factor for postoperative AMI.

**By Invitation*

43. Bilateral Internal Mammary Artery Grafting: in Situ vs Y Graft. Long Term Clinical and Angiographic Results.

Antonio M. Calafiore, Marco Contini*, Giuseppe Vitolla*, Michele Di Mauro*, Valerio Mazzei*, Giovanni Teodori* and Gabriele Di Giammarco*, Chieti, Italy.

Discussant: Alfred J. Lector, M.D.

OBJECTIVE: To evaluate if the use of BIMA as in situ or Y graft provides the same long term results.

METHODS: From September 1991 to August 1999,1359 pts had BIMA in situ (n=1104, group A) or as Y (n=255, group B) graft.

RESULTS: Anastomoses/pt and BIMA anastomoses/pt were higher in group B (3.2±0.9 and 2.7±1.1) than in group A (2.8±0.8 and 2.3±0.8), $p<0.001$. Thirty day mortality was 1.9% in group A vs 3.1% in group B, p=ns. There was no difference in postoperative course. Eight years survival was 96.9+0.6 in group A vs 96.2±2.2 in group B, p=ns, and event free survival was 95.8±0.8 in group A vs 95.4±2.1 in group B, p=ns. Early angiographies were obtained in 281 pts (894 anastomoses, 828 distal and 66 proximal Y) 215 (591) in group A and 66 (303) in group B. Patency rate was 98.9% in group A and 96.7% in group B, p=ns. Late angiographies were obtained in 68 pts (18 in goup A and 50 in group B)at mean of 17.5±18.4 months: patency rate was 100% in group A and 98.9% in group B, p=ns, while grade A patency rate was 98.0% in group A and 98.4% in group B, p=ns. No Y anastomosis was occluded or stenosed.

CONCLUSIONS: The use of BIMA in situ or as a Y graft have similar survival, cardiac events incidence and angiographic patency in early and late phase.

**By Invitation*

4:35 p.m. EXECUTIVE SESSION (Members Only)

Constitution Hall, Metro Toronto Convention Centre

6:15 p.m. Reception at Royal York Hotel

Followed By "The Lion King"

Princess of Wales Theatre

(Separate Subscription Required)

By Invitation

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - 2

GENERAL THORACIC SURGERYRoom 205
Metro Toronto Convention Centre

Moderators: Mark J. Krasna, M.D.

David J. Sugarbaker, M.D.

45. Superficial Adenocarcinoma of the Esophagus

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

Discussant: Nasser K. Altorki, M.D.

OBJECTIVE: Superficial adenocarcinoma of the esophagus (SAE), invading no deeper than the submucosa, was uncommon before the epidemic of Barrett's adenocarcinoma. SAE is now identified more frequently with regular endoscopic surveillance (ES); however, there is limited experience with treatment and outcome. The purpose of the study was to evaluate the results of surgical management of SAE and identify predictors of survival.

METHODS: Between 9/85 and 9/99, 111 patients (pts) underwent resection of SAE. 89% were men, median age was 64 years (yrs)(range 35-83). 50% were in ES programs. The table shows pathologic staging. Follow-up extended to 14 yrs, mean 47±40 months. Risk factors for mortality were identified by Cox multivariable regression.

RESULTS: There were 3 (2.7%) operative deaths. 5-yr survival decreased as depth of tumor invasion (T) increased: 96±4% Tis, 84±6% T1 intramucosal, and 47±12% T1 submucosal, *P=.004*. 5-yr survival was worse for N1 vs. No pts (17±16% vs. 82±5%, *P<.001*). 5-yr survival was worse in SAE discovered at first diagnostic esophagoscopy than in SAE discovered in ES programs (69±8% vs. 84±6%, *P=.005*). By multivariable analysis, N1 disease (*P=.006*), increasing T *(P=.05),* and older age (*P=.007*) decreased survival; participation in an ES program (*P=.009*) improved survival. Need for postoperative reinrubation led to worse early survival (47%±15 vs. 95%±2 at 1 yr, *P=.02*).

CONCLUSIONS: 1) Resection of SAE offers excellent survival with minimal operative mortality. 2) Survival is improved by ES and early resection before SAE invades the submucosa or metastasizes to regional lymph nodes. 3) Early survival may be improved by careful preoperative respiratory evaluation and aggressive perioperative respiratory care.

	Total n,(% total)	NO n,(% T subgroup)	N1 n,(% T subgroup)
Tis (high-grade dysplasia)	35 (32%)	35(100%)	0 (0%)
T1 intramucosal	47 (42%)	46 (98%)	1 (2%)
T1 submucosal	29 (26%)	23(79%)	6(21%)

**By Invitation*

46. Histology and Stage Are Independent Prognostic Factors in Thymomas

Cameron D. Wright, Abeel A. Mangi*, John C. Wain, Dean M. Donahue*, James S. Allan*, Ashby C. Moncure, Earle W. Wilkins, Hermes C. Grille and Douglas J. Mathisen, Boston, Massachusetts

Discussant: Antoon E.M.R. Lerut, M.D.

OBJECTIVE(s): The Masaoka Staging system is currently used to stratify patients with thymomas. Histologic classification by the Muller-Hermelink Scheme has also been shown to correlate with prognosis. We reviewed patients with thymomas to evaluate the Masaoka staging system and histolgy as prognostic factors.

METHODS: Single institution restrospective review.

RESULTS: From 1972 to 1999, 155 patients underwent resection of a thy-moma. Overall 15 year survival was 55% whereas 15 year disease-specific survival (DSS) was 89%. Univariate analysis revealed that Masaoka stage (p<.0001), histology (p<.0001), and complete resection (p<.0001) predicted survival. Multivariate analysis revealed that Masaoka stage (p=.005) and histology (p=.02) independently predicted survival. There was no difference in disease-free survival (DPS) or DSS between Masaoka stage 1 or 2 or between 2a and 2b (p=ns). Classification of patients into two risk groups based on Masoka stage and histology clearly separated patients who had no relapses from those who did not (p=.0001) and was an independent predictor of survival (p=.002).

CONCLUSIONS: Histology (by the Muller-Hermelink system) is an independent predictor of survival in thymoma. The early stages of the Masaoka classification system are not distinct and do not accurately predict recurrences. A dichotomous classification system which takes into account both histology and stage better separates thymoma patients into clinically important prognostic groups and could help guide adjuvant treatment.

Risk of Recurrence Based on Stage and Histology

	LOW RISK			HIGH RISK	
HISTOLOGY	Medullary	Cortical	WDTC	Cortical	WDTC
STAGE	1,2a,2b	1,2a		2b,3,4	
RECURRENCE	0/88			15/67	
DSS(15y)	98%			75%	

**By Invitation*

47. Neoadjuvant Chemotherapy Increases the Length of Stay and Health-Provider Effort in Patients Undergoing Pulmonary Resection for NSCLC

John R. Roberts*, Chad wick Eustis*, Elaine M. Eustis* and Walter Merrill,

Nashville, Tennessee

Discussant: Keith S. Naunheim, M.D.

OBJECTIVE: Surgical effort has been the topic of HCFA and Medicare actions in recent months. However, little data about need for increasing surgical effort due to advances or changes in treatment exist. One such change is the use of neoadjuvant chemotherapy, which has become the standard for stage IIIA NSCLC in many institutions. Further, neoadjuvant therapy and may be used for earlier stages in the future. We have previously shown that neoadjuvant chemotherapy increases life-threatening complications in patients undergoing surgery and postulated that these patients would require greater surgical effort than other patients.

METHODS: All patients undergoing resection (lobectomy or greater) after neoadjuvant chemotherapy were compared to patients undergoing similar resections without preoperative chemotherapy. The resections were all done at a single institution in one year. Data collected were length of stay, ICU days, intubated days, chest tube duration, operative time, EBL, and health care provider visits. Two-tailed Student's t test was used to analyze differences in means and chi-square to determine differences in proportions. Differences <0.05 were considered significant.

RESULTS: Thirty-four patients underwent resection after neoadjuvant chemotherapy and 67 patients were resected without preoperative therapy. No differences between the groups in age, pulmonary function, or comorbid diseases were found. The patients receiving chemotherapy did have a more advanced stage (2.52 versus 1.55, $p<0.0001$). There was no hospital mortality. Patients receiving preoperative chemotherapy had a greater length of stay (13.9 vs. 8.0 days, $p=0.032$), greater blood loss (462 cc vs 304 cc, $p=0.03$), and required twice as many health provider visits (66.1 vs 33.4, $p=0.03$)

CONCLUSIONS: Neoadjuvant carboplatin and taxol increased the length of stay, EBL, and number of physician visits in this cohort of patients compared to a similar cohort undergoing surgery in the same institution. These data demonstrate that neoadjuvant chemotherapy increases the physician effort necessary to care for these patients.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - 2

GENERAL THORACIC SURGERY Room 205
Metro Toronto Convention Centre

Moderators: Mark J.Krasrw,M.D.

David J. Sugarbaker, M.D.

48. Outcome of Lung Volume Reduction Surgery in Emphysema Patients Eligible for Lung Transplant

Bryan F. Meyers*, Stephen S. Lefrak*, Mary S. Pohl*, Tracey J. Guthrie*, Roger D. Yusen*, G. Alexander Patterson and Joel D. Cooper, St. Louis, Missouri

Discussant: Douglas E. Wood, M.D.

OBJECTIVE: Between March 1993 and May 1998, we performed 200 consecutive bilateral lung volume reduction (LVRS) operations for patients with emphysema. Ninety-nine of these patients were considered eligible for either LVRS or lung transplant (TX) based on age, impairment, and absence of contraindications. The clinical outcomes of these 99 patients were reviewed to assess the consequences of LVRS on patients eligible for lung transplant.

METHODS: A retrospective chart review was performed using a prospectively assembled computer database.

RESULTS: The 61 men and 38 women had a mean age of 55 ± 7 years at the time of LVRS. Mean values for first second expired volume, total lung capacity and residual volume were 24 ± 7, 141 ± 19 and 294 ± 54 percent predicted, respectively. These values are identical to those observed in our overall LVRS experience. There were 4 perioperative deaths and 14 late deaths. Two-year and 5-year survival after LVRS were 92% and 77%. The 31 patients who have been listed for TX after LVRS include 13 who have been transplanted, 14 who remain on the list, and 4 who have been removed from the list. All 13 transplanted patients survived TX and one has subsequently died of chronic rejection. Twelve surviving recipients have a median post-transplant follow-up of 1.3 years. The mean age of the TX recipients was 57.5 ± 5.1 years with TX occurring 3.6 ± 1.0 years after LVRS. The fourteen patients still on the TX waiting list have a mean interval since LVRS of 4.6 ± 1.0 years. Sixteen of the 99 patients underwent lower lobe LVRS and 10 of these patients have either been transplanted (6) or listed (4). No significant differences were found between patients listed for TX and patients not listed when compared according to age, lung function, or response to LVRS.

CONCLUSIONS: Lung transplant following LVRS is feasible and associated with no apparent increased mortality. Patients undergoing LVRS for lower lobe disease are more likely to progress to transplant.

**By Invitation*

49. A Novel Approach Using Magnetic Resonance Technique for the Detection of Lung Allograft Rejection

Shinichi Kanno*, Paul C. Lee*, Stephen Dodd*, Mangay Williams*, Timothy R. Billiar*, Bartley P. Griffith, Chien Ho*, Pittsburgh, Pennsylvania

Discussant: Steven J. Mentzer, M.D.

OBJECTIVE(s): Although various techniques have been explored for the detection and quantification of allograft rejection, a practical and reliable method that is non-invasive is still elusive.

METHODS: For our magnetic resonance (MR) experiments, we have developed a new rat model of heterotopic lung transplantation to the inguinal region. Allogeneic transplants (DA†'BN) were performed with and without cyclosporin-A (CsA) treatment, with syngeneic transplants (BN†'BN) serving as controls (n=6 per group). MR images were obtained with a gradient echo method before and after injection of ultra-small superparamagnetic iron oxide (USPIO).

RESULTS: At day 5, allogeneic transplants without CsA treatment developed a grade 4 rejection pathologically. A significantly lower MR signal was seen 24 hours after USPIO injection (346±7.6 vs 839±43.4, arbitrary unit, $p<0.05$). Syngeneic transplants showed no evidence of rejection pathologically and no differences in MR signal between injections (863±18.8 vs 880±22.5). Allotransplants treated with CsA showed a grade 2 rejection pathologically. The change in MR signals in that group was small, but significant enough to show a decrease in signal intensity after injection (646±10.5 vs 889123.5, $p<0.05$). Immunohistochemistry and iron staining in the allografts revealed that USPIO was taken up by the infiltrating macrophages that accumulated at the rejecting site.

CONCLUSIONS: We demonstrated a novel approach for detection of acute lung rejection with USPIO injection. This method might have tremendous clinical application.

**By Invitation*

50. Laryngotracheal Resection and Reconstruction for Postintubation Tracheal Stenosis Extending to the Subglottic Region

Paolo Macchiarini *, Jean-Philippe Verroye *, Alain Chapelier *, Elie Fadel * and Philippe Dartevelle, Hannover, Germany; Paris, France
Discussant: F. Griffith Pearson, M.D.

OBJECTIVE: Analyze the characteristics and results of laryngotracheal resection and reconstruction for postintubation tracheal stenosis extending to the subglottic region.

METHODS: Fourty-rwo patients (31 males and 11 females, mean age 41±18 years) underwent resection of the anterior cricoid cartilage and primary thyrotracheal reconstruction for subglotic stenosis. Five of them had also a tracheo-esophageal fistula, repaired simultaneously *via* a primary two-layers esophageal closure. Twenty-patients (52%) were referred to us after initial unsuccessful endoscopic (n=22) or surgical *(n=5)* management. The stenosis appeared 30±43 days from endotracheal intubation (n=19) or tra-cheostomy (n=23). There were 24 cuff lesions, 7 stomal lesions, and 11 at both levels; all but one stenosis were circumferential. The upper limit of the stenosis lied 1.9±.7 cm below the vocal cords. Stenoses measured 2.8±.9 cm in length and the esophageal defects 2.5±1 cm. The subglottic diameter was reduced by 60% in 36 or 86% of patients. All but one operations were performed through a cervical incision only. The length of resection ranged from 2 cm to 6.5 cm (mean 4.6±1). Eighteen thyrohyoid and 4 supralaryngeal releases were employed to reduce anastomotic tension.

RESULTS: Four patients (9%) required post-operative tracheostomy, and 38 (91%) were extubated within 24 hours. Early complications occurred in 11 patients (27%) and were most frequent in patients requiring laryngeal release and extended resections; one patient died (2%). Among the remaining 41 patients, 39 or 95% had excellent or good anatomical and functional long-term results. Two failures required definitive tracheostomies.

CONCLUSIONS: Resection of the anterior cricoid cartilage and primary thyrotracheal reconstruction is the best treatment for post-intubation sub-glottic stenosis.

**By Invitation*

51. Timed Barium Esophagram: a Simple Physiologic Assessment for Achalasia

Srodjan Kostic*, Thomas W. Rice, Joel E. Richter*, Mark E. Baker*, Malcolm M. DeCamp*, Lisa A. Rybicki* and Eugene H. Blackstone, Cleveland, Ohio

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

OBJECTIVE: The outcome of achalasia therapy is difficult to measure because repeated physiologic study is impractical and symptom interpretation is subjective. In contrast, timed barium esophagram (TBE) is simple, easily performed, inexpensive, quantitative, repeatable, and comfortable for the patient. The purposes of this study were 1) to evaluate the use of TBE in appraising the outcome of myotomy and 2) to determine the cause of symptoms and their relief by myotomy.

METHODS: 52 patients (pts) ingested 250 ml low-density barium and had upright spot films at 1, 2, and 5 minutes preoperatively (preop) and at 8 weeks (median) after myotomy (postop). Height and width of the barium column and their change over time were measured and related by multi-variable analyses to symptoms of regurgitation, dysphagia and chest pain. Symptoms were scored as 0 (none) to 5 (continuous).

RESULTS: At 1,2, and 5 minutes preop, mean barium column height was 18,16, and 15 cm, and width 5.7,5.3, and 5.0 cm. Surgery reduced these to 7.7, 6.6, and 4.5 cm, and 3.4, 3.1, and 2.6 cm, respectively ($P<.001$). The preop degree of regurgitation was related directly to height of the barium column at 1 minute ($P=.003$).Mean height was 23 cm for grade 4-5 regurgitation and 14 cm for grade 0-1. Degree of dysphagia was related directly to change in width from 1 to 5 minutes ($P=.06$).Mean change was -0.4 cm for grade 4-5 dysphagia ($P=.06$)and -1.0 cm from grade 0-1. Chest pain was related inversely to width at 1 minute ($P=.06$). Width was 5.0 cm for pts with any grade chest pain and 6.1 cm for those without. Surgery relieved symptoms in the majority of pts (grade 4-5 regurgitation from 45% to 5%, grade 4-5 dysphagia from 86% to 7%, and any grade chest pain from 52% to 12%, $P<.001$). Residual symptoms were unrelated to TBE measurements.

CONCLUSIONS: 1) TBE gives objective confirmation of successful myotomy. 2) TBE reveals that regurgitation and its relief are related to the height of the barium column, dysphagia to the rate of esophageal emptying, and chest pain to the less dilated non-myotomized esophagus. TBE is a simple measure of esophageal emptying that elucidates the mechanism of symptoms and their relief by myotomy.

**By Invitation*

4:35 p.m. EXECUTIVE SESSION (Members Only)

Constitution Hall, Metro Toronto Convention Centre

6:15 p.m. Reception at Royal York Hotel

Followed By "The Lion King"
Princess of Wales Theatre

(Separate Subscription Required)

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - 2

CONGENITAL HEART DISEASE Room 201

Metro Toronto Convention Centre

Moderators: John J. Lamberti, M.D.

William G. Williams, M.D.

52. Modifications to the Cavopulmonary Anastomosis Do Not Eliminate Sinus Node Dysfunction

Mitchell I. Cohen*, Nancy D. Bridges*, J. W. Gaynor*, Timothy M. Hoffman*, Gil Wernovsky*, Victoria L. Vetter*, Thomas L. Spray and Larry A. Rhodes*, Philadelphia, Pennsylvania

Discussant: Peter Manning, M.D.

OBJECTIVE: Sinus node dysfunction (SND) occurs frequently after the Fontan (F) operation and can have deleterious effects on F physiology. The objective of this study was to determine whether modifications of the cavopulmonary anastomosis (CPA) which avoid surgery near the sinus node result in a lower incidence of SND.

METHODS: Since 1996, a prospective cohort study has been conducted evaluating the incidence of SND in all patients (pts) staged with either an initial hemi-Fontan (HF) or bidirectional Glenn (BDG) and a subsequent lateral tunnel (LT) or extracardiac conduit (EC). Only pts with normal sinus node function prior to the HF or BDG were included. SND was defined by a heart rate ‰¥ 2 S.D. below age adjusted norms, or predominant junctional rhythm, or a sinus pause ‰¥ 3 sec. as determined by resting ecg and/or 24° Holter monitor at hospital discharge (d/c). The primary outcome evaluated was the difference in the incidence of SND in pts with either HF/LT or BDG/EC. Other perioperative characteristics which might lead to SND were evaluated.

RESULTS: As of 9/99, 74 pts have entered the study: 46 had a HF (mean age 6.8±2 mos) and 28 a BDG (mean age 7.3±3 mos). All 28 BDG pts and 2 additional HF had a subsequent EC F (mean age 24±5 mos). The remaining 44 HF pts had a LT F (mean age 23±6 mos). Diagnoses were, hypoplas-tic left heart (29), single LV (18), single RV (13), heterotaxy (7), and other (7). Among the 74 pts, SND was present in 9 (12%) after the F. In those with a HF/LT the incidence was 14% (n=6) and in BDG/EC pts the incidence was 10% (n=3) (.95 CI .05-.27 vs .02-.28; risk ratio 1.3; p=NS). No diagnostic or intraoperative variables were associated with SND. There were no deaths in the BDG/EC group and 1 death in the HF/LT group. 2 HF/LT pts had pacemakers: 1 for heart block (normal atrial rate)and 1 for prolonged sinus pauses.

CONCLUSIONS: In this cohort study, avoidance of surgery near the sinus node had no discernible effect on the development of SND. Thus concerns about SND should not override pt anatomy or surgeon preference as determinants of which CPA to perform. Longer follow-up is needed to determine if either staging strategy will reduce the long-term incidence of SND and pacemaker implantation.

**By Invitation*

53. Pulmonary Microvessel Density Is a Marker of Angiogenesis in Children After Cavopulmonary Shunt

Sandra L. Starnes*, Brian W. Duncan*, James M. Kneebone*, Shawn States*, Geoffrey L. Rosenthal* and Flavian M. Lupinetti*, Seattle, Washington

Discussant: Richard A. Jonas, M.D.

OBJECTIVE: Pulmonary arteriovenous malformations are a frequent cause of progressive cyanosis in children after cavopulmonary anastomosis and may represent a form of abnormal angiogenesis. Microvessel density has been used as a marker of angiogenesis in tumor studies. We determined the microvessel density in the lungs of children after cavopulmonary anastomosis with and without clinical evidence of pulmonary arteriovenous malformations to determine if they had increased numbers of blood vessels consistent with ongoing angiogenesis.

METHODS: Lung biopsy specimens were obtained from six children following cavopulmonary shunt and four age-matched controls. Of the six children following cavopulmonary anastomosis, two had angiographically documented pulmonary arteriovenous malformations while the other four children had no clinical or angiographic evidence of pulmonary arteriovenous malformations. Microvessels staining positive for a primary antibody to von Willebrand factor were counted in ten high-power fields (200X) per patient.

RESULTS: The mean (±standard error) microvessel density in all patients following cavopulmonary anastomosis was 31.3±7.4 versus 9.3±5.7 in controls, $p = 0.02$. There was no difference in microvessel density in children with and without pulmonary arteriovenous malformations following cavopulmonary anastomosis (33.1±10.6 versus 30.4±8.6, $p = 0.9$).

CONCLUSIONS: Following cavopulmonary anastomosis, children have greatly increased numbers of pulmonary microvessels regardless of whether they have clinically or angiographically significant pulmonary arteriovenous malformations. This supports the evidence of a constant angiogenic stimulus in the lungs of children after cavopulmonary anastomosis.

54. Long-Term Results of the Lateral Tunnel Fontan Operation

Christof Stamm*, Ingeborg Friehs*, John E. Mayer, David Zurakowski*, John K. Triedman*, Edward P. Walsh*, Richard A. Jonas and Pedro J. del Nido, Boston, Massachusetts

Discussant: Gordon K. Danielson, M.D.

OBJECTIVE: Construction of a total cavopulmonary anastomosis using an intra-atrial lateral tunnel is known to yield good early and mid-term results. Given the current controversy regarding indications for a total extracardiac Fontan procedure, we reviewed the long-term outcome after a lateral tunnel Fontan operation.

METHODS: Between 10/87 and 12/91, 220 patients (age: 11 months to 32 years, median = 3.9 ± 0.6 years) underwent a fenestrated or non-fenestrated lateral tunnel Fontan procedure at our institution. Diagnoses included single left ventricle with normally related (n=72) or transposed (n=81) great arteries, single right ventricle (n=28), heterotaxy (n=22), hy-poplastic left heart (n=14), and others (n=3). Current follow-up information was available for 179 patients (mean follow-up 10.2 ± 0.6 years). Risk factor analysis included patient- and procedure-related variables

with death, failure (death, takedown, or transplantation), and brady- or tachyarrhythmia as outcome parameters.

RESULTS: There were 10 early deaths, 2 late deaths, 4 take-down operations, and 2 heart transplantations. Kaplan-Meier estimated survival was 92.7% (70% confidence interval = 91-95%) at 5 and 10 years, freedom from failure was 89% (86-91%) at 5 years and 88% (85-90%) at 10 years. Freedom from bradyarrhythmia was 93% (91-95%) at 5 years and 89% (87-92%) at 10 years, freedom from atrial tachyarryhthmia 98% (97-99%) at 5 years and 94% (92-96%) at 10 years. One patient developed protein losing enteropathy. Risk factors for development of atrial tachyarrhythmia were heterotaxy syndrome (odds ratio = 14.1, P = 0.002) and single morphologic right ventricle (OR = 7.6, P = 0.01). None of the patient-related variables significantly influenced survival.

CONCLUSIONS: The lateral tunnel Fontan operation results in superior long-term survival irrespective of the underlying anatomic diagnosis. The incidence of atrial arrhythmia appears to depend on ventricular morphology. The excellent long-term outcome after an intracardiac lateral tunnel Fontan procedure should serve as a basis for comparison with other surgical alternatives.

**By Invitation*

55. Modified Norwood Procedure Using a High Flow Cardiopulmonary Bypass Strategy Results in Low Mortality Without Late Arch Obstruction.

Nancy C. Poirier*, Jonathan J. Drummond-webb*, Michi Imamura*, Alexander M. Harrison*, Roger B. B. Mee, Cleveland, Ohio

Discussant: Edward L. Bove, M.D.

OBJECTIVE: We reviewed our results of a modified stage 1 Norwood repair (mNr) using only autologous tissue. It is performed with high flow Cardiopulmonary bypass (CPB)followed by aggressive postoperative va-sodilation (both attained using phenoxybenzamine) and a normocapneic ventilatory strategy.
METHODS: Between 1993 and 1999, 59 patients aged 1 to 353 days (median 4 days) and weighing 1.7 to 6.8 kg (median 3.2 kg)underwent a mNr. The procedure consists of excising ductal tissue and augmenting the arch by the combined anastomosis of the main pulmonary artery and descending aorta. Ascending aortic diameter ranged from 1.5 to 8 mm (median 3). The modified Blalock-Taussig shunt was 3 mm in 21 patients (36%) and 3.5 mm or larger in 38 patients (64%).

RESULTS: Circulatory arrest (CA) and CPB times ranged from 15 to 64 min (median 37) and 44 to 144 min (median 88) respectively. Postoperative survival was 83% (49/59). At univariate analysis, early mortality was associated with ascending aortic diameter of less than 2.5 mm (p=0.015). Weight, age at operation, associated procedures, CAand CPB times, diagnosis (HLHS vs variant), shunt size, and date of the procedure did not influence the early or late operative survival. During a median follow-up period of 39 months (range=l-63), 30 patients underwent bidirectional cavopulmonary shunts (BCPS; 61%), 6 Fontan (12%) and 1 cardiac transplantation following a failed BCPS. Four patients died for an overall survival of 76%. Neo-aortic or arch obstruction was corrected in 3 patients (5%).

CONCLUSIONS: Our results of mNr using this perioperative strategy are acceptable with a low incidence of neo-aortic and arch obstruction. Patients with small ascending aorta diameters are a high-risk group and perhaps candidates for alternative approaches.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:25 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - 2

CONGENITAL HEART DISEASE

Metro Toronto Convention Centre

Moderators: John J. Lamberti, M.D.

William G. Williams, M.D.

56. Long-Term Follow-up After Early Repair of Tetralogy of Fallot: the Impact of a Transannular Patch.

Emile A. Bacha*, Lars Erickson*, John E. Mayer, Pedro J. del Nido, Judy Huing*, Peter Lang*, Richard A. Jonas, Boston, Massachusetts

Discussant: Roger B. B. Mee, M.D.

OBJECTIVE: Early primary repair of Tetralogy of Fallot (TOP) has been routinely performed at this institution since 1972. This study examines the impact of a transannular patch (TAP) on mortality, need for reoperation, right heart failure, medication, arrhythmias, and reproductive health in a cohort of patients 20 or more years following this procedure.

METHODS: Sixty patients aged less than 2 years underwent repair of TOP between 1/1972 and 12/1977 (median age 8.1 months). Follow-up data were obtained on 46 (85%) of the 52 patients who survived the operation (median follow-up 17.5 years). A TAP was used in 32 survivors (70%). Data were compared between the TAP-group and non-TAP group.

RESULTS: No late deaths were identified. Nine patients (17 %) required a reoperation: 6 (5 with non-TAP) underwent relief of right ventricular outflow tract obstruction. One patient each required a pacemaker, an AICD, and a pulmonary valve insertion for right ventricular dilation (all with TAP). Forty-two patients (91%) were in NYHA class I and 4 in class II. Two out of 16 females, both with TAP, carried pregnancies to term. Freedom from reoperation of any type was 94 vs. 88% at 10 years and 91 vs. 80% at 18 years for TAP vs. non-TAP ($p=0.11$). No independent predictors of long-term survival could be identified by multivariate analysis.

CONCLUSIONS: Pulmonary valve insertion is rarely indicated after early primary repair of TOP with a TAP. There is equal long-term survival and freedom from reoperation as compared to non-TAP.

**By Invitation*

57. Coronary Arterial Size Late After Atrial Switch Operation in Patients with Transposition of Great Arteries: Implications for Arterial Switch Operation.

Zahid Amin*, Phillip Moore*, Doff B. McElhinney*, Vadiyala M. Reddy and

Frank L. Hanky, Augusta, Georgia; San Francisco, California; Philadelphia,

Pennsylvania

Discussant: Roger B. B. Mee, M.D.

OBJECTIVE: Coronary flow reserve in the hypertrophied ventricle is reduced. One contributing factor may be the size of proximal coronary arteries. In patients who have undergone an atrial switch procedure for transposition of great arteries, the left coronary artery (LCA) supplies the pulmonary ventricle and may be small. We hypothesized that the dimensions of the coronary arteries may be related to symptomatic status in these patients, and that a small LCA may relate to failure of attempted pulmonary artery banding (PAB) and conversion to arterial switch, possibly by affecting blood supply to the interventricular septum.

METHODS: Left and right coronary arteries (RCA) were measured in 10 asymptomatic patients after atrial switch procedure, 9 symptomatic patients after atrial switch procedure, and 10 patients with normal hearts. The size of the coronary arteries was indexed to the body surface area.

RESULTS: Both the absolute and indexed diameters of the LCA were significantly smaller than those of the RCA in symptomatic patients after atrial switch (indexed: 2.3±0.5 vs 3.3±0.6 mm, $p<0.001$), whereas there was no difference in asymptomatic patients (2.2±0.4 vs 2.4±0.4, $p=0.47$). Similarly, the RCAtLCA diameter ratio was significantly larger in symptomatic than asymptomatic patients (1.5±0.3 vs 1.1+0.3, $p=0.007$). There was no difference in coronary artery size between normal patients and asymptomatic patients with TGA after atrial switch procedure.

CONCLUSIONS: Differences in size of the proximal coronary arteries may be related to symptomatic status in patients with transposition of great arteries who have undergone atrial switch procedure, possibly by affecting blood supply to the interventricular septum.This would predispose to more severe tricuspid regurgitation and failure. When PAB and subsequent arterial switch are considered for patients with atrial switch and a failing systemic right ventricle, size of the LCA may be an important factor to consider, and should be evaluated with preoperative imaging studies.

**By Invitation*

58. Ross Procedure in Children : a Word of Caution.

Antonio Laudito*, V. Mohan Reddy*, Michael M. Brook*, Marc S. Bleiweis*,

Lenardo D. Thompson* and Frank L. Hartley, San Francisco, California

Discussant: Ronald C. Elkins, M.D.

OBJECTIVE: Aortic valve disease in children is often a difficult and complex problem with controversial management strategies. The Ross and Ross-Konno procedures have become the primary choice for aortic valve replacement (AVR) because of optimal hemodynamic performance, no need of anticoagulation and growth potential. However, there is continuing concern regarding

the longevity of the pulmonary autograft especially in patients with primary aortic insufficiency (AI).

METHODS: Between June 1993 and September 1999, 77 Ross and Ross-Konno procedures were performed at our institution: 10 patients were infants (borderline hypoplastic left heart patients were excluded), 61 were children, 6 were adults. Preoperative, postoperative, and follow-up clinical, echocardiographic, and hemodynamic data were reviewed. Statistical analysis was performed to identify the risk factors for deteriorating autograft function.

RESULTS: Primary AI was an indication for Ross procedure in 23 patients and mixed lesion with predominant AI in 20 patients. In the rest aortic stenosis (AS) was the primary or predominant lesion. There were no early or late deaths. Prosthetic AVR was necessary in the follow-up period in 5 children for severe AI: 3 of them had AI as primary lesion, and 2 had mixed lesion with predominant AI in one. At follow-up 4 children have moderate AI: 3 had predominant AI as indication for Ross procedure and 1 had AS as primary lesion. Although it did not reach statistical significance (X^2test, p= 0.077),there is a trend of deteriorating autograft function in patients with primary or predominant AI. In addition other reinterventions included right ventricle to pulmonary artery conduit replacement (n=7, including 5 pts during AVR) and internal mammary artery coronary bypass grafting (n=2).

CONCLUSIONS: Ross and Ross-Konno procedures have altered the prognosis of infants and young children with severe and complex aortic valve disease. However, Ross procedure in older children especially with primary or predominant AI should be performed with caution. Further follow-up to delineate the risk factors for autograft dysfunction in children is necessary to better define the indications for Ross procedure

**By Invitation*

4:35p.m. EXECUTIVE SESSION (Members Only)

Constitution Hall, Metro Toronto Convention Centre

6:15 p.m. Reception at Royal York Hotel

Followed By "The Lion King"
Princess of Wales Theatre

(Separate Subscription Required)

WEDNESDAY MORNING, MAY 3, 2000

7:00 a.m. EMERGING TECHNOLOGIES AND TECHNIQUES

Constitution Hall, Metro Toronto Convention Centre

Moderators: Robert W. Emery, M.D.

Hani Shennib, M.D.

T1. Computer Enhanced "Robotic" Cardiac Surgery - First Clinical Results in 100 Patients

Friedrich W. Mohr, Volkmar Falk*, Anno Diegeler*, Ralf Krakor*, Jan F. Gummert*, Thomas Walther* and Ruediger Autschbach*, Leipzig, Germany

OBJECTIVE: A computer enhanced instrumentation system was tested to enable total endoscopic cardiac surgery.

METHODS: The daVinci™ system provides a 3-D videoscopic image and allows remote, tremorfree and scaled control of endowrist instruments with 6 degrees of freedom. We used the system in 83 patients for CABG and in 17 patients for endoscopic mitral valve repair. The system was used for ITA-take down followed by minithoracotomy (n=44), to perform the coronary anastomoses in standard sternotomy CABG partially in off pump technique (n=15), or total endoscopic coronary artery bypass grafting of the left ITA to the LAD (TECAB, n = 24). In 17 with non-ischemic mitral valve insufficiency the mitral valve was repaired (MVR). Closed-chest cardiopulmonary bypass with cardioplegic arrest (Port-Access technique) was used for TECAB and MVR.

RESULTS: ITA take down was successful in 58/61 pts. in the MIDCAB group and after a steep learning curve is currently performed in less than 50 minutes *(44 ± 13 minutes)*. Endoscopic ITA take-down decreased the size of the mini thoracotomy *(6.8 ± 2.3cm)*. Injury to the ITA with the need of conversion to sternotomy occurred in 2/83 patients (2.4%) TECAB was completed in 19/24 cases. In 5 pts. conversion to an successful open approach was necessary to safely identify the course of LAD in 3 pts. Angiographic control after 3 months revealed a 100% patency (19/19) in the successful TECAB patients and one graft occlusion (1/92 anastomoses) in all other patients. One patient in the CABG group died from a stroke. In the MVR group one patient with Barlow disease had residual MI II and minimal invasive MVR was immediately followed. In the remaining 16/ 17 patients the primary repair was successful.

CONCLUSIONS: The da Vinci™ system allows for precise tissue handling and enables the endoscopic performance of cardiac surgical tasks thatrequire a high degree of dexterity (coronary anastomosis, MVR). No technical mishaps have occurred. The steep learning curve with long procedural times in the beginning may be preventable by the future use of simulation technology. This experimental study may pave the way for fully endoscopic computer guided surgery.

**By Invitation*

T2. Animal and Clinical Study of a New Sutureless Anastomotic Device for the Proximal SVG Anastomosis.

Antonio M. Calafiore, Yaron Barel*, Giuseppe Vitolla*, Assaf Dekel*, Chieti, Italy; Haifa and Herzelia, Israel.

OBJECTIVE: Evaluate the safety and efficacy of a new self expanding nitinol Aortic Anastomotic Device (AAD), designed for off-pump proximal SVG anastomosis without manipulation of the aorta.

METHODS and RESULTS: 16 sheep were used as the animal model. 45 SVG anastomosis were created (patency verified by flow meter), 32 using the AAD and 13 using conventional hand suturing techniques. All procedures were done off pump by a cardiovascular specialist. At sacrifice patency rates were 28 of 32 for AAD bypasses (87.5%) and 11 of 13 for hand sutured bypasses (85.0%). Average anastomosis diameter was 4.5mm and 3.5mm for AAD and hand sutured anastomoses respectively. The AAD was used in 11 coronary patients, 7 on pump and 4 off-pump. Flow meter showed all bypasses to be patent, measurement ranging from 9 to 65 ml/min (distal

anastomoses:PDA-9, marginal-4, diagonal-1). The AAD procedure took between 1 to 2 minutes. One AAD did not deploy and was replaced by hand suturing. There were no surgical or post-surgical complications with the AAD. 1 patients had early angiography (5 days), that showed perfect patency (elective angiography was scheduled for 3 months).

CONCLUSIONS: The AAD seems to be a reliable and effective device in creating a sutureless fast SVG anastomosis, especially in beating heart surgery. Avoiding manipulation of the aorta will reduce the risk of embolization from side clamping. The self expanding nature of the device creates a large diameter anastomosis.

**By Invitation*

T3. Facilitated Coronary Anastomosis Using the Nitinol Sutured-Clip™ Device.

Arthur C. Hill*, Eric Monnet*, Timothy Maroney* and Renu Virmani*,
Omaha, Nebraska; Fort Collins, Colorado; Richmond, Virginia; Washington, District of Columbia

OBJECTIVE: The Sutured-Clip™ device, (Coalescent Surgical, Inc.), was designed to facilitate minimally invasive coronary anastomosis by eliminating the need for suture management, knot tying, and surgical assistance. The device employs the superelastic properties of Nitinol and consists of two components: 1.) a needle/suture delivery system, and; 2.) a detachable Nitinol self-closing wire. The device was tested in the bovine OPCAB model to determine its ease of use and chronic anastomotic functional characteristics.

METHODS: The Sutured-Clip device was tested in 14 calves. RITA-to-coronary artery anastomosis was performed on the beating heart using stabilizer via a left thoracotomy. Functional characteristics of each anastomosis produced by the Sutured-Clip device were evaluated using intra-operative angiography (n=14), intra-operative Doppler flowimetry (n=13), angiography at one week (n=4), angiography at 8 weeks (n=8), and histology at one week (n=4) and 8 weeks (n=8).

RESULTS: All calves survived to completion of the study. All Sutured-Clip anastomoses and grafts were patent and without stenosis by Doppler flowimetry, intra-operative angiography, post-operative angiography, and histology. Histological evaluation snowed smooth neointimal resurfacing of the Nitinol device and interposed tissue with no tissue necrosis and minimal inflammation. Duration of coronary occlusion during anastomosis averaged 14.70 (range: 10-21) minutes. Graft flow by Dopper flowimetry averaged 74.41 (range: 22-180) ml/minute. Two calves, intended for extended long term survival, are still alive and showed angiographic patency at 6 and 10 weeks following device implantation.

CONCLUSIONS: The Nitinol Sutured-Clip device produced high quality interrupted anastomoses in this initial bovine OPCAB study. The device facilitates coronary anastomosis by simplifying and decreasing the amount of manipulation and complexity required in minimally invasive CABG procedures. Nitinol technology also has potential in robotic and conventional surgical applications.

**By Invitation*

T4. Modified Glenn Connection for Acute Ischemic Right Ventricular Failure Reverses Secondary Left Ventricular Systolic Dysfunction.

Mark H. Danton*, John G. Byrne*, Kathryn Q. Flores*, Jeffrey S. Martin*, Michael Hsin*, Lawrence H. Cohn and Lishan Aklog*[§], Boston, Massachusetts

OBJECTIVE: Acute ischemic RV failure results in serve haemodynamic compromise, we hypothesize: 1 In addition to reducing LV preload, acute RV dilation may directly compromise LV contractility and 2 By unloading the RV, through a modified Glenn anastomosis, this effect will be attentuated.

METHODS: A SVC main PA connection was performed in 8 pigs and isolated RV ischemic failure induced by selective coronary ligation. Simultaneous measurements of ventricular pressure with volume (RV) and segment length (LV) were made.

RESULTS: Ischemia caused acute RV dilation and, independent of preload, LV systolic and diastolic dysfunction. RV unloading significantly reversed the regional LV contractility deficit without improving diastolic parameters.

CONCLUSION: In acute ischemic RV failure with normal pulmonary vascular resistance a modified Glenn shunt preserves LV systolic performance by limiting RV dilation.

Indicies	Baseline	RV ischemia	Glenn circuit
RV EDV (ml)	102	143†	118‡
LV ESPLR mitiHg/mm	52.1	18.6†	31 .2‡
LV PRSW mm Hg	83.0	45.5†	63.6‡
RV ESPVR mmHg/mL	0.82	0.46†	0.46
RVPRSWmmHg	15.2	11. 6†	11.8
LV Tau ms	35.6	58.8†	51.6
LV dP/dt min	790	476†	533
Cardiac output, L/min	4.01	3.1†	3.0

EDV end diastolic volume, ESPV(L)R end systolic pressure volume (length) relationship. PRSW precruitable stroke work index.

†<0.05 between baseline and ischemia

‡<0.05 between ischemia and Glenn circuit

[§]*International Traveling Fellow*

**By Invitation*

[§]T5. Device Based Left Ventricular Shape Change Immediately Reduces Regional Areas and Improves Fractional Shortening Indices in a Canine Cardiomyopathy Model

Patrick M. McCarthy, Kiyotaka Fukamachi*, Masami Takagaki*, Guy Armstrong*, James B. Young*, Cyril J. Schweich*, Todd J. Mortier* and Marc R. Raffe*, Cleveland, Ohio; Auckland, New Zealand; Plymouth, Minnesota

OBJECTIVE: To test the acute effect of left ventricular (LV) shape change on regional areas, contractile indices, and hemodynamics in a heart failure model

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METHODS: Heart failure was induced in 6 healthy dogs implanted with pacemakers by pacing at 230 beats per minute for an average of 23 days. A novel device, the Myocor™ Myosplint™, was surgically implanted in the LV to produce a calculated 20% or 30% decrease in wall stress by changing LV shape. This was accomplished by placing 3 Myosplint devices perpendicular to the LV long axis, creating a symmetric, bUobular LV. Papillary and mitral level diastolic (PD,MD) and systolic (PS,MS)areas were measured by 2D echo prior to, and following tightening at 20% and 30% stress reduction levels. Papillary level (PFAS), mitral level (MFAS) and mean (FAS_{avg}) fractional area shortening were calculated. Cardiac output (CO), stroke volume (SV), heart rate (HR), and blood pressure (BP) were measured.

RESULTS: Comparing pre and post tightened values, PD, PS,and MS areas were significantly ($p<0.05$, paired t-test) lower at 20% and 30% stress reduction. MD area was significantly lower at 30% stress reduction. FAS_{av} and MFAS were significantly higher at 20% and 30% stress reduction. PFAS was significantly higher at 30% stress reduction. HR, SV, CO, and BP remained unchanged.

CONCLUSIONS: Shape change produced by the Myocor Myosplint immediately improved contractile indices and reduced ventricular area measurements with maintenance of stable hemodynamics. The shape change concept has been clinically replicated as evidenced by the results of our first 2 human cases.

	PD (cm^2)_	PS (cm^2)_	MD (cm^2)_	MS (cm^2)_	PFAS	MFAS	FAS_{avg}_
Pretighten	19.7	15.9	20.8	16.3	0.23	0.22	0.22
20%	17	10.9	18.6	12.9	0.36	0.30	0.33
Pretighten	18.3	14.7	20.4	16	0.24	0.22	0.23
30%	13.8	9	16.1	10.8	0.35	0.33	0.34

[§]*Authors have a relationship with Myocor*

**By Invitation*

8:00 a.m. GENERAL THORACIC SURGERY

FORUM SESSION Room 205

Metro Toronto Convention Centre

Moderators: Robert J. Keenan, M.D.

Nasser K. Altorki, M.D.

F1. Increase in Vascular Endothelial Growth Factor and Basic Fibroblast Growth Factor Expression in Esophageal Adenocarcinoma and Barrett's Esophagus.

Reginald V. Lord*, Ji Min Park*, Kathleen D. Danenberg*, Tom R. DeMeester, Jeffrey H. Peters*, Dennis Salonga*, Steven R. DeMeester*,

Jeffrey A. Hagen*, Stefan Oberg*, Jon Singer*, Cedric G. Bremner* and Peter V. Danenberg*, Los Angeles, California

OBJECTIVE: To determine the role of the angiogenesis factors Vascular Endothelial Growth Factor (VEGF) and basic Fibroblast Growth Factor (bFGF) in the development and progression of Barrett's esophagus and adenocarcinomas of the esophageal body and gastroesophageal junction.

METHODS: VEGF and bFGF mRNA expression levels, relative to the stably expressed internal control genefi-actin, were measured in specimens of Barrett's intestinal metaplasia (IM, n=21), dysplasia (n=11), adenocarci-noma (n=17), and matching normal squamous esophagus tissues. A quantitative reverse transcription-polymerase chain reaction (RT-PCR) method was used (ABI 7700 Sequence Detector (Taqman® system)).

RESULTS: Expression levels of both VEGF and bFGF were significantly increased in adenocarcinoma compared to either Barrett's esophagus or normal esophageal mucosa (table). Compared to normal mucosa, VEGF expression was significantly increased in non-dysplastic Barrett's mucosa, while bFGF expression was significantly increased in dysplastic Barrett's mucosa (all Mann-Whitney U test).

CONCLUSIONS: Angiogenesis factors are significantly upregulated in esophageal body and gastroesophageal junction adenocarcinomas and are likely to be important in the development of these cancers. Increased expression of angiogenesis factors can also be found in some patients with Barrett's esophagus, suggesting that Barrett's epithelium can have cellular migratory potential at an early stage. Expression levels of these genes may be useful markers for cancer detection in patients with Barrett's esophagus.

	VEGF	bFGF
CA vs. Normal	<0.0001	0.001
CA vs.IM	0.039	0.001
CA. vs. dysplasia	0.019	0.4

**By Invitation*

F2. Sequential 5-Aza-2' Deoxycytidine (DAC)/Depsipeptide (DP) Treatment of Esophageal Cancer Cells Facilitates Their Recognition by Cytolytic T Cells (CTL) Specific for NY-ESO-1

Todd S. Weiser*, Galen Ohrunacht*, Julie Hong*, R-F Wang*, Dao M. Nguyen* and David S. Schrump, Bethesda, Maryland

OBJECTIVE: Previously we demonstrated synergistic enhancement of MAGE-3 expression and apoptosis in lung and esophageal cancer cells by sequential exposure to the demethylating agent 5-Aza-2' deoxycytidine, and the histone deacerylase inhibitor, Depsipeptide. This study was performed to determine if similar treatment could induce expression of NY-ESO-1 in esophageal cancer cells, and enable their recognition by CTL specific for this tumor antigen.

METHODS: Five esophageal cancer cell lines (all negative for NY-ESO-1 expression) were exposed to normal media, DAC, DP, or sequential DAC/ DP treatment using optimized induction regimens. Quantitative RT-PCR techniques were utilized to determine NY-ESO-1 mRNAcopy number relative to Î²-actin. Class I HLA expression was evaluated by PCR and flow cytometry techniques. TE-12 cells were transduced with a retroviral HLA-A31 construct or a retroviral control

vector to examine recognition by HLA-A31 restricted NY-ESO-1 specific CTL utilizing y-IFN release assays.

RESULTS: Relative to baseline, NY-ESO-1 expression was enhanced 81-fold (range 11.8-198) by DAC alone (0.1 μM x 72h), 2.2-fold (0.8-4.1) by DP alone (25 ng/ml x 6h), and 139-fold (24.4-428) by sequential DAC/DP treatment. HLA expression was not altered under these conditions. CTL-mediated y-IFN release was observed in response to HLA-A31 transduced TE-12 cells following exposure to DAC (139 pg/ml) or DAC/DP (140 pg/ ml) but not normal media (0 pg/ml) or DP alone (0 pg/ml). Cytokine release in response to HLA-A31 transduced TE-12 cells following DAC or sequential DAC/DP treatment approximated that observed in response to autologous melanoma cells (157 pg/ml).

CONCLUSIONS: Sequential DAC/DP treatment represents a novel strategy to augment antitumor immunity in esophageal cancer patients.

**By Invitation*

F3. Adenoviral Mediated Bak Gene Transfer Induces Apoptosis in Mesothelioma Cell Lines

Abujiang Pataer*, W. Roy Smythe*, Singling Fang*, Timothy J. McDonnell*, Jack A. Roth and Stephen G. Swisher*, Houston, Texas

OBJECTIVE: Mesothelioma cell lines are resistant to the induction of apoptosis following p53 gene therapy. We, therefore, evaluated the novel approach of adenoviral gene transfer of the pro-apoptotic Bcl-2 family member: Bak.

METHODS: Binary adenoviral Bak (Ad-GTBak +Ad-GV16) and Lac-Z (Ad-GTLac-Z+Ad-GV16) vectors were constructed for transduction of the mesothelioma cell lines: Ren and 145.

RESULTS: High levels of Bak gene transfer were seen following coadministration of Ad-GTBak and AdGV16 in both mesothelioma cell lines. High levels of apoptosis were induced 24 hours after Bak gene transfer with characteristic morphologic changes, Caspase 3 cleavage and subdiploid populations on FACS analysis (Table 1): Cell viability was decreased 48 - 72 hours after Bak gene transfer (Figure 1):

CONCLUSIONS: Adenoviral mediated overexpression of the Bak gene induces apoptosis and decreased cellular viability in mesothelioma cells. These data suggest that the gene transfer of pro-apoptotic Bcl-2 family members may represent a novel gene therapy strategy to treat mesothelioma.

Table 1: Percent Apoptosis of Mesothelioma Cell Lines

Mesothelioma Cell Line	PBS Control	Ad-GTLac-Z	Ad-GTBak
I45 (p53 wild-type)	1%	1%	36%*
REN (p53 mutant)	2%	3%	25%*

* $p < 0.05$

**By Invitation*

F4. Differential Display Analysis Identifies New Highly Activated Gene in Mesothelioma: the Folate Receptor.

Raphael Bueno*, Krishnarao Appasani*, Harriet Mercer* and David J. Sugarbaker, Boston, Massachusetts

OBJECTIVE: The Folate Receptor is activated in certain solid tumors such as breast, renal and testicular carcinomas. As a consequence, antifolate drugs are effective in these neoplasms as they are in malignant pleural mesothelioma. We have, therefore, studied mesothelioma specimens to evaluate the Folate Receptor expression in this neoplasm.

METHODS: RNA was prepared from fresh tissues obtained from patients with mesothelioma. Differential display analysis was performed using normal lung RNA, normal pleura RNA, and tumor RNA. The analysis yielded 60 differentially expressed genes which were then characterized. One of the genes that was overexpressed in mesothelioma versus normal tissue was the Folate Receptor gene. In order to better characterize the expression of the Folate Receptor gene in mesothelioma, we performed *in-situ* hybridization utilizing antisense probes based on the sequence of the Folate Receptor. Frozen sections from 37 different patients(21 epithelial, 14 mixed, 2 sarcomatoid tumors)with pleural mesothelioma were analyzed using this technique. The controls included normal pleura, normal lung, and sense probes for all of the rumors. We also tested tumors known to have high expression of the Folate Receptor such as breast cancer, testicular cancer, and fallopian tube cancer as controls.

RESULTS: 31 of the 37 tumor specimens had between two- and four-fold higher Folate Receptor mRNA expression when compared with the control tissues. The slides of the other tested rumors demonstrated a similar elevation in the expression of the Folate Receptor. 6 tumors (4 epithelial and 2 mixed) were found to have the same or lower Folate Receptor mRNA expression.

CONCLUSIONS: The majority of mesothelioma tumors examined were found to express the Folate Receptor at two- to four-fold higher levels compared with normal pleura or lung. This is the first report of Folate Receptor overexpression in mesothelioma. This finding may explain the reports of up to a 30% clinical response to the antifolate drug methotrex-ate in mesothelioma. Furthermore, it encourages the testing of newer antifolate agents in the rational treatment of mesothelioma.

**By Invitation*

F5. Obliterative Airway Disease Requires Direct Allorecognition

Wilson Y. Szeto*, Alyssa M. Krasinskas*, Daniel Kreisel*, Sicco H. Popma* and Bruce R. Rosengard*, Philadelphia, Pennsylvania

OBJECTIVE: Obliterative bronchiolitis (OB)is the primary cause of late death after lung transplantation. In *a* murine model, heterotopic tracheal allografts develop Obliterative airway disease (OAD), which resembles OB histologically. Chimeric tracheas bearing recipient-type antigen presenting cells (APCs) were used to examine whether direct allorecognition triggered by donor-type APCs is required for the development of OAD.

METHODS: Chimeric tracheas (i.e. tracheas having parenchyma and APCs of differing genotypes) were created via bone marrow transplantation (BMT). Tracheal segments from naive B6 mice, C3H†'B6 chimeras, B6 Class I ($B6^{I-}$) or B6 Class II MHC antigen knockout mice ($B6^{II-}$)

were transplanted subcutaneously in the dorsum of the recipients. The grafts were harvested at days 14 and 28, and the degree of luminal occlusion was determined by light microscopy.

RESULTS: At day 28, near complete occlusion was seen in all groups except for the chimeric tracheas, which lack donor-type APCs and, therefore, have substantial reduction of direct allorecognition. All isografts showed minimal luminal occlusion at both time points.

CONCLUSIONS: OAD is minimal in chimeric tracheas lacking donor-type APCs, but develops in the absence of either Class I or Class II MHC antigens. These findings suggest that direct allorecognition, triggered by donor APCs, is required for OAD, and that either the $CD4^+$ or $CD8^+$ direct allorecognition pathway is sufficient to initiate the process.

Tracheal	Tracheal	Tracheal		% Luminal Occlusion	
Donor	Parenchyma	APCs	Reciplent	Day 14	Day 28
C3H	C3H	C3H	C3H	0% (n=2)	0% (n=3)
naive B6	B6	B6	C3H	not done	87.5% (n=4)
C3H†'B6	B6	C3H	C3H	3% (n=3)	24% (n=5)
$B6^{I-}$	$B6^{I-}$	$B6^{I-}$	C3H	0% (n=1)	100% (n=2)
B6II-	$B6^{II-}$	$B6^{II-}$	C3H	100% (n=2)	100% (n=3)

**By Invitation*

F6. Adenovirus-Mediated Gene Transfer of Human Interleukin-10 Ameliorates Reperfusion Injury of Rat Lung Isografts

Hideki Itano*, Wanjiang Zhang*, Thalachallour Mohanakumar* and G. Alexander Patterson, St. Louis, Missouri

OBJECTIVE: Interleukin-10 has been identified as a potent inhibitor of various inflammatory responses. The objective of this study was to examine the feasibility of human interleukin-10 (hIL-10) gene transfer into rat lung isografts and its effect on subsequent ischemia reperfusion (I/R) injury.

METHODS: Male F344 rats were divided into four groups and underwent left lung isotransplantation. Twenty four hours prior to harvest, 5 x 10E9 pfu (Group I, n=6) or 1 x 10E10 pfu (Group II, n=7) of AdRSVhIL-10 was intravenously administered to donor rats. In Group I-C (n=6) and Group II-C (n=6), serving as control, 5 x 10E9 pfu and 1 x 10E10 pfu of AdCMVLacZ were administered respectively. In all groups, grafts were preserved forlS hours at 4 degree prior to implantation, and assessed 24 hours after reperfusion. Transgene expression of hIL-10 was assessed by both RT-PCR and immunohistochemistry (IHC). Graft iNOS mRNA expression was assessed by RT-PCR. Isograft gas exchange and MPO activity were also assessed.
RESULTS: Dose-dependent transgene expression was detected by RT-PCR ($p < 0.05$) and IHC. Gas exchange in Groups I and II was significantly better than in Groups I-C and II-C (Table). MPO activity (OD/min/ng protein) in Group II was significantly lower than in Group II-C (0.082 ± 0.034 vs. 0.117 ± 0.028, $p < 0.05$). The iNOS mRNA expression in Group II was significantly lower than in Group II-C (0.332 ± 0.39 vs. 0.745 ± 0.29, $p< 0.05$).

CONCLUSION: In vivo lung isograft adenovirus-mediated hIL-10 gene transfer ameliorates I/R injury.

Isograft gas exchange

Groups	PaO2	PaCO2	Groups	PaO2	PaCO2
Group 1	164.72 ±85.3*	33.40 ± 6.80*	Group II	153.19±113*	43.64 ±15.1
Group I-C	82.37±19.1	51.23 ± 11.9	Group II-C	77.95 ±33.4	50.60 ±17.7

* $p < 0.05$ vs. control, (mmHg)

**By Invitation*

F7. SCRlslex Is Superior to Scrl in Reducing Ischemia/Reperfusion Injury in Experimental Lung Transplantation

Uz Stammberger*, Sven Hillinger*, Giovanni L. Carboni*, Walter Weder*, Juerg Hamacher* and Ralph A. Schmid*, Berne and Zurich, Switzerland

OBJECTIVE: The nonspecific immune response including activation of the complement system and polymporphonuclear leukocytes (PMN) is critical for the mediation of reperfusion injury. We investigated the combined blockade of the complement system and leukocyte adhesion by a novel drug, sCR1sLex. In this glycoprotein, the sugar molecules of soluble complement receptor type 1 (sCR1; CD35) are substituted with the selectin ligand sialyl LewisX (sLex; CD15s)(AVANTIMMUN, Needham MA).

METHODS: Orthotopic allogeneic single left lung transplantation was performed in rats (BN to F344) after 20 hours ischemia. Three groups (n=5) were studied: Group I (control), Group II received sCRl (10 mg/kg) and group III sCRlsLex (10 mg/kg) 15 min prior to reperfusion by intracar-diac injection. Twenty-four hours after reperfusion, the native contralat-eral lung was occluded to assess isolated gas exchange of the graft. In additional animals (n=5/group), lung tissue was frozen 24 h after reperfusion and assessed for myeloperoxidase activity (MPO; neutrophil migration), and thiobarbituric acid reactive substances (TEARS; lipid peroxidation).
RESULTS: Graft function in group III was superior to group I and group II:

CONCLUSIONS: Our data indicate that combined inhibition of the complement system and leukocyte adhesion with sCR1sLex reduces reperfusion injury significantly, and that both mechanisms are effectively inhibited in this model.

Results

Group	*PaO_2 [mmHg]*	*_MPO [Î”OD/mg/min]*	*TBARS [pmol/g]*
I	56±7	1.0+±0.09	10.6±0.54
II	243±45**	0.48±0.07***	8.32+0.89*
III	383±53***#	0.33±0.05***	6.210.37***#
Native lung		0.2210.05#	3.9±0.75*#

p<0.05,p<="" span="">#p<0.05vs. sCR1*

**By Invitation*

F8. Manganese Superoxide Dismutase Gene Insertion Protects Normal Cells During Photodynamic Therapy

Hsien Yean Wong*, Micheal Epperly*, Tony Godfrey*, Joel Greenberger* and James D. Luketich*, Pittsburgh, Pennsylvania

OBJECTIVE: PDT is a new modality for the treatment of esophageal and lung cancers which involves free radical mediated cell death leading to mitochondrial disruption and apoptosis. PDT efficacy is limited by damage to surrounding normal cells. MnSOD localizes to mitochondria and scavenges free radicals. Our Objective was to determine if MnSOD overexpression would protect against PDT-induced cell death.

METHODS: Normal mouse hematopoetic cell line (32 D c!3) served as control. Two subclones transfected with human MnSOD transgene (2C6, 1F2) were produced which overexpress MnSOD. Photofrin (lug/ml) was added to cell culture 24 hrs before light activation. Survival was determined by plating cells in methylcellulose and colony counting. Staining for apoptosis was performed.

RESULTS: PDT dose response curve slope (D_0) for 32D c!3, 2C6 and 1F2 were 2.8,13, and 6.7 indicating better survival for MnSOD incorporation. Percent cell kill was determined by apoptotic staining (see table).

CONCLUSIONS: These results suggest MnSOD gene incorporation protects normal cells from PDT-induced death. Further work by our groups in ongoing to determine if MnSOD gene incorporation leads to overexpression and protection in the normal human esophagus.

Mean Apoptosis (%)

Light exposure (J)	32 D	2C6	1F2
4	2.5	8	0.0
6	14	6.5	15
7	19	2.0*	22
8	58	4.0*	38*

* indicates significant resistance to apoptosis compared to 32D, $p < 0.05$

**By Invitation*

F9. Lung Volume Reduction Surgery Restores the Normal Diaphragmatic Length-Tension Relationship in Emphysematous Rats

Joseph B. Shrager*, Dong-Kwan Kim*, Yahya Hashmi*, Hansell H. Stedman*, Sanford Levine* and Larry R. Kaiser, Philadelphia, Pennsylvania

OBJECTIVE: Improved respiratory muscle function is a major effect of lung volume reduction surgery (LVRS). There has been no previous study of respiratory muscle in an experimental model of LVRS. We sought to elucidate the mechanism by which diaphragmatic function improves following LVRS.

METHODS: We developed a model of elastase-induced emphysema and LVRS via median sternotomy (MS) in rats. Five months following emphysema induction, lung volume (VC) was determined in intubated, anesthetized emphysema and control animals. Costal diaphragmatic length (LC) was measured in vivo, and Lo (the length at which maximal twitch force is generated) was

determined in vitro. Also 5 months following elastase administration, a cohort underwent LVRS or sham MS. Five months following operation, these animals were similarly studied.

RESULTS: VC was increased in emphysematous rats versus controls (50.9+/-4.8 vs 45.4+/-3.8cc, p=.024). VC was decreased in emphysematous animals which had undergone LVRS versus sham MS (44.7+/-2.1 vs 49.4+/-2.7cc, p=.001). LC (1.99+/-.11 vs 2.24+/-.11cm, p=.001) and Lo (2.25+/-.21 vs 2.48+/-.29cm, p=.038) were shorter in emphysematous than controls animals. Following LVRS, LC (2.13+/-.07 vs 1.83+/-.16cm, p<.001) and Lo (2.50+/-.17 vs 2.27+/-.15cm, p=.013) were longer than in sham MS animals.
CONCLUSIONS: In this experimental model of emphysema and LVRS, emphysema shortens Lo and shifts the diaphragmatic length-tension curve to the left; LVRS returns Lo toward normal and shifts the diaphragmatic length-tension curve back to the right. This restoration toward normal physiology may enable the improvement in diaphragmatic function seen following LVRS. The mechanism by which Lo lengthens merits further investigation.

§Author has a relationship with Metabolix, Inc.

**By Invitation*

F10. Replacement of the Trachea with an Autologous Aortic Graft

Emmanuel Martinod*, Rachid Zegdi*, Gilbert Zakine*, Paul Fornes*, Alexandre D'Audiffret*, Juan-Carlos Chachques*, Jacques Azorin* and Alain Carpentier, Paris and Bobigny, France.

OBJECTIVE(s): Tracheal reconstruction after extensive resection remains an unsolved surgical problem. Many studies have evaluated various substitutes including prostheses, tracheal allografts or autologous grafts with disappointing results. The goal of this experimental study was to evaluate the replacement of a large segment of the trachea using an autologous aortic graft, selected for specific advantages: similar diameter, resistance to infection and lack of immune response.

METHODS: In 20 sheep, a 5 cm segment of the cervical trachea was resected and replaced by a 5 cm segment of the descending thoracic aorta. The thoracic aorta was reconstructed with a vascular prosthesis using an arterial shunt. A definitive Ultraflex TM stent (n=13) or a temporary Novatech TM stent (n=7) was placed in the lumen of the aortic graft to prevent collapse. Clinical, bronchoscopic and histologic examinations were performed at 1, 3, 6, 9 and 12 months.
RESULTS: All animals survived the operation and there were no paraplegia. There has been only 3 complications : one stent displacement, one laryngeal edema, one infection. In the remaining 17 animals, there was no anastomotic leakage, rupture or stenosis. Histology showed a progressive transformation of the arterial segment into a tracheal tissue including a neoformation of cartilage and a continuous epithelium.

CONCLUSIONS: This study shows that an autologous aortic graft could be a valuable substitute for tracheal replacement. In humans, the abdominal aorta could be used and replaced with a prosthesis, a minor inconvenience compared to the benefit of a durable tracheal reconstruction.

**By Invitation*

8:00 a.m. ADULT CARDIAC SURGERY FORUM SESSION

Constitution Hall, Metro Toronto Convention Centre

Moderators: Andrew S. Wechsler, M.D.

Fred A. Crawford, M.D.

F11. Marrow Stromal Cells for Cellular Cardiomyoplasty: Feasibility and Clinical Advantages

Jih-Shiuan Wang*, Dominique Shum-Tim*, Jacques Galipeau*, Edgar Chedrawy*, Nicoletta Eliopoulos* and Ray Chu-Jeng Chiu, Taipei, Taiwan ROC; Montreal, PQ, Canada.

OBJECTIVE: Bone marrow stromal cells (MSC) are mesenchymal stem cells able to differentiate into cardiomyocytes in vitro. We tested the hypothesis that MSC, when implanted into myocardium, can undergo milieu-dependent differentiation and form long-term, incorporated grafts expressing cardiomyogenic phenotypes in vivo.

METHODS: Isogenic adult rats were used as donors and recipients to simulate autologous transplant in patients. MSC isolated from donor leg bones were expanded in culture, labeled with DAPI (4', 6-diamidino-2-phenylindole), and then injected directly into the myocardium of the recipients. The hearts were harvested from 4 days to 12 weeks after implantation, and the implant sites were sectioned for histological and immuno-histochemical studies to identify the phenotypes of the labeled cells.

RESULTS: Viable DAPI labeled cells can be identified in host myocardium at all time points after implantation. The implanted cells can be seen to juxtapose to the host myocardium, or pooled together at the implantation area. At the periphery of implantation area and within the adjacent host myocardium, DAPI labeled cells appear to be incorporated into the host myofibers and join with myocytes not labeled by DAPI. Such DAPI labeled cells showed positive stain for sarcomeric myosin heavy chain (Using MF20 antibody) and connexin 43 (Gap junction protein in the intercalated disks) by 4 weeks after implantation.

CONCLUSIONS: Fetal and "altered" adult cardiomyocytes, as well as skeletal myoblasts had been used as donor cells for cellular cardiomyoplasty, and shown to improve the function of impaired ventricles. Our findings indicate that MSC can also be used as donor cells, and in appropriate microenvironment, they can exhibit cardiomyogenic phenotypes, and may replace native cardiomyocytes lost by necrosis or apoptosis. However, in contrast to other cell sources, autologous MSC can be obtained repeatedly by simple routine bone marrow aspiration, and expanded vastly in vitro prior to implantation. Furthermore since autologous implants will not require immunosuppression, clinical use of MSC for cellular cardiomyoplasty appears to be most advantageous.

**By Invitation*

F12. Cellular Therapy Reverses Myocardial Dysfunction

Juan C. Chachques*, Charissa Rajnoch*, Alain Berrebi*, Nicolas Borenstein*, Ming Shen*, Nicola D'Attellis*, Jean N. Fabiani*, Alain F. Carpentier, Paris, France.

OBJECTIVE(s): The aim of cell transplantation into pathologic myocardium is to repair, replace or enhance the biological function of the altered ventricle, restoring a functional myocardial mass, and hence improving the contractile performance of the heart.

METHODS: Autologous myoblasts obtained from skeletal muscles were implanted into the LV wall in an experimental model of partial ventricular akinesia. *Step I:* Chronic cardiac deficiency was developed by local injection (3 mg/1.5 ml) of snake cardiotoxin (C 9759, Sigma Chem.) in the LV wall of sheep, through a left mini-thoracotomy. *Step II:* Autologous myoblasts taken from skeletal muscle biopsy of animal limbs were cultured for 3 weeks. A selection procedure was performed to obtain a pure culture. Then, through a sternotomy, myoblasts were introduced in the injured myocardium. A cell suspension of 2 x 10^7 cells diluted in 500mL of Ham-F12 medium was injected. Echocardiographic studies (Color Kinesis H.P.) were performed after toxin, after cell injections and at 2 months. Histopathologic studies were carried out at 2 months. Control groups consisted of injection of toxin alone (6 sheep) and toxin + culture medium (6 sheep).

RESULTS: All animals survived. After toxin injections, serum levels of troponin increased significantly (up to 125±16 ng/ml), and LV wall morion (regional fraction area change: RFAC) decreased from 71±4 to 40±2.5%, $p<0.05$. Two months following myoblasts implantation, ventricular remodeling partially reversed and myocardial contractility significantly recovered in the cell implanted group (RFAC: 65±7%). Healthy myoblasts were observed in 75% of myocardial histological studies. Cardiotoxin administration generated transmural necrosis, with severe damage of cardiomyocytes and interstitial tissues (myocardial matrix).

CONCLUSIONS: Myoblast implantation was associated with the recovery of myocardial contractility in an experimental model of segmentary ventricular akinesia ("infarct-like" myocardial lesion). Healthy myoblasts were observed 2 months after myocardial implantation. Further studies on host-cell interactions (mechanical and electrical coupling) are necessary.

**By Invitation*

F13. Hemodynamic Unloading Leads to Regression of Pulmonary Vascular Disease in Rats

Stacy B. O'Blenes*, Stefan Fischer*, Brendan Mclntyre*, Shaf Keshavjee,

Marlene Rabinovitch*, Toronto, ON, Canada.

OBJECTIVE: Treatment options for patients with pulmonary vascular disease secondary to a congenital heart defect are still mainly limited to heart-lung transplantation or lung transplantation with repair of the cardiac lesion. However, we have previously shown that the structural changes associated with pulmonary hypertension can be reversed by stress unloading in an organ culture model. We now test the hypothesis that hemodynamic unloading will lead to regression of pulmonary vascular disease in the intact animal.

METHODS: Right middle+lower lobectomy and monocrotaline injection was performed in Lewis rats (n=22) to cause pulmonary vascular disease from a combined hemodynamic and toxic injury. Twenty eight days later the left lungs were examined (n=10) or exposed to normal pulmonary artery (PA) pressure for an additional 14 (n=5) or 28 (n=7) days by transplantation into healthy recipients. PA pressure, ventricular weight, and PA morphology was evaluated in each group.

RESULTS: Pulmonary hypertension (50 vs. 16 mmHg, $p<0.001$) and right ventricular hypertrophy (RV/LV weight 0.69 vs. 0.32, $p<0.001$) associated with PA medial hypertrophy (28.2 vs. 7.2 % of wall thickness, $p<0.001$) and muscularization of small PAs (92.3% vs. 19.4%, $p<0.001$) developed by day 28 (compared to untreated controls). However, transplantation into healthy recipients effectively unloaded the lungs (mean PA pressure 17 and 25 mmHg at 14 and 28 days post transplant) and resulted in progressive normalization of medial hypertrophy (15.6 and 12.1% at 14

and 28 days) and muscularization (65.1 and 42.2% at 14 and 28 days) relative to untransplanted controls ($p<0.005$ in each case).

CONCLUSIONS: Hemodynamic unloading of lungs with pulmonary vascular disease results in progressive normalization of PA structure. These results are the first to provide a rationale for attempting to induce regression of pulmonary vascular disease by pressure unloading of the pulmonary circulation. PA banding should be critically evaluated as a strategy for staged surgical repair of congenital heart defects despite presumed irreversible pulmonary hypertension.

**By Invitation*

F14 Cerebral Effects of Reperfusion After Hypothermic Circulatory Arrest

Marek P. Ehrlich*, Donald Weisz*, David Wolfe*, Carol A. Bodian*, Ning Zhang*, Jock N. McCullough* and Randall B. Griepp, New York, New York

OBJECTIVE: This study was undertaken to explore whether an interval of cold reperfusion can improve cerebral outcome following prolonged hypothermic circulatory arrest (HCA).

METHODS: Sixteen pigs (27-30 kg) underwent 90 minutes of HCA at a brain temperature of 20C. Eight animals were rewarmed immediately after HCA; eight were reperfused for 20 minutes at 20C and then rewarmed. Electrophysiological recordings, fluorescent microsphere determinations of cerebral blood flow (CBF), calculations of cerebral oxygen consumption (CMRO2), and direct measurements of intracranial pressure (ICP, mm Hg) were obtained at baseline (37C), before HCA, after cardiopulmonary bypass (CPB) was restarted, and at 2, 4, and 6 hours thereafter. Histopathology and wet/dry brain weight were determined after sacrifice.

RESULTS: CBF and CMRO2 decreased during cooling: CMRO2 returned to baseline levels after 4 hours, but CBF remained depressed until 6 hours in both groups. Cold reperfusion failed to improve electrophysiological recovery or to reduce brain weight, but the increase in median ICP usually seen after prolonged HCA was significantly less after cold reperfusion than after immediate rewarming ($p = 0.02$),), as seen in the table below. Although no significant difference in the incidence of cerebral histopathology between groups was found, all three animals with ICP > 15 following immediate rewarming had histopathological lesions, and high ICP was more prevalent among all animals with subsequent histopathology ($p = 0.03$).

CONCLUSIONS: Cold reperfusion significantly inhibited the rise in ICP usually seen after 90 minutes of HCA at 20C, suggesting that it may decrease cerebral edema and thereby improve outcome following prolonged HCA.

Intracranial Pressure (ICP, mmHg)

	Before HCA	After CPB	2 Hours	4 Hours	6 Hours
Immediate Rewarming	9.5	13	15	15	14.5
Cold Reperfusion	9	9	10	11.5	12.5

**By Invitation*

F15. The Fate of a Tissue Engineered Cardiac Graft in the Right Ventricular Outflow Tract

Tetsuro Sakai*, Ren-Ke Li*, Richard D. Weisel, Donald A. G. Mickle*, Eung Joong Kim*, Zhi-Qian Jia*, Terrence M. Yau*, Toronto, ON, Canada.

OBJECTIVE: Currently available graft materials for repair of cardiac defects are nonviable and contribute to late morbidity and mortality. We have developed a beating cardiomyocyte-seeded biodegradable graft *in vitro.* We evaluated the *in vivo* fate of this graft in the right ventricular outflow tract (RVOT) of adult rats.

METHODS: Cultured fetal or adult rat cardiomyocytes (1 x 10^6 cells) were seeded into a gelatin mesh (15 x 15 x 1 mm) and maintained in tissue culture for 1 or 3 weeks. 15% of the cells were prelabelled with BrdU prior to graft seeding, to permit cell identification after graft implantation. The RVOT free wall of syngeneic adult rats was partially resected and replaced with grafts seeded with fetal or adult cardiomyocytes, or unseeded grafts (N=10 per group). The hearts were excised at 4 or 12 weeks, and the grafts and perigraft tissue examined histologically. Graft endothelialization was evaluated by immunostaining for Factor VIII, and persistence of seeded cells by staining for BrdU.

RESULTS: Mean endocardial surface area of the grafts 4 weeks after implantation was 8.8±3.0 mm^2, which represented 6.7±2.9% of the area of the RV free wall. Factor VHI staining confirmed complete endothelialization of the graft surface at 4 weeks. A significant perigraft lymphocytic infiltrate was noted. By 12 weeks after implantation, the original gelatin framework of the graft had been completely resorbed. BrdU positive cells were noted throughout the graft, but cell numbers decreased with time. Unseeded grafts were completely replaced by fibrous tissue. Graft thickness decreased significantly between 4 and 12 weeks in the unseeded ($p=0.003$), fetal ($p=0.0001$) and adult ($p=0.07$) cardiomyocyte groups, but to a similar degree ($p=0.2$).

CONCLUSIONS: The RVOT of adult rats can be replaced with a cardiomyocyte-seeded biodegradable graft. The seeded cells persist within the graft over 12 weeks, but cell survival is limited by the host inflammatory response to the gelatin substrate. A less antigenic substrate may enhance cell survival. These techniques may lead to the development of a viable, cellular graft with growth potential for reconstruction of congenital cardiac defects.

**By Invitation*

F16. A Mitral Annuloplasty Ring Preserves Competent Leaflet Geometry During Acute Left Ventricular Ischemia

David T. M. Lai*, Tomasz A. Timek*, Paul Dagum*, Julie R. Glasson*, G. Randall Green*, George T. Daughters*, David Liang*, Neil B. Ingels* and D. Craig Miller, Stanford and Palo Alto, California

OBJECTIVE: To determine the perturbations of mitral leaflet geometry during acute left ventricular (LV) ischemia which lead to mitral regurgita-tion (IMR), and to determine whether annuloplasty rings can prevent these detrimental changes.

METHODS: Radiopaque markers were implanted in 3 groups of male sheep: 4 along the middle of the anterior mitral leaflet, 2 along the middle of the posterior mitral leaflet and 8 at equidistant points around the mitral annulus. One group served as control (C, n=7) and the others underwent Duran (D, n=6) and Physio (P, n=5) ring annuloplasty. Following an 8 ± 2 day recovery period, 3-D marker coordinates were obtained by biplane videofluoroscopy before and during balloon

occlusion of the circumflex artery to create LV ischemia. Leaflet geometry was defined by measuring distances between annular and leaflet markers, and perpendicular distances from the best-fit annular plane (derived from least-squares estimate regression) to the leaflet markers. Leaflet coordinate measurements were compared using student's t-test for paired observations. The presence or absence of IMR was assessed by transthoracic echocardiography.

RESULTS: In all C animals, LV ischemia was associated with apical displacement of the posterior leaflet away from the annular plane by 0.6 mm ($p<0.01$) and IMR. Compared to its non-ischemic position, the posterior leaflet edge moved 0.5 mm, 0.3 mm and 0.3 mm, respectively, further away from the septal annulus ($p<0.01$), anterior ($p<0.05$) and posterior ($p<0.01$) commissures with ischemia. The anterior leaflet edge moved 0.3 mm further away from the septal annulus ($p<0.05$), but compared to its non-ischemic position, the anterior leaflet was not displaced from the annular plane with ischemia. In all D and P animals, leaflet geometry was unchanged by ischemia and no IMR was detected.
CONCLUSIONS: IMR was associated with restricted motion of the posterior leaflet yet flattening of the anterior leaflet. An annuloplasty ring prevented these geometrical perturbations of the mitral leaflets during acute LV ischemia and preserved valvular competence.

**By Invitation*

F17. Transplantation of Cryopreserved Cardiomyocytes

Hiroki Yokomuro*, Ren-Ke Li*, Richard D. Weisel, Donald A. G. Mickle*,

Terrence M. Yau*, Toronto, ON, Canada.

OBJECTIVE: Cardiomyocyte transplantation has been shown to improve the function of infarcted hearts. However, fresh primary cardiomyocyte cultures may be difficult to obtain when transplantation is required. We therefore evaluated the survival and immunogenicity of cryopreserved fetal rat cardiomyocytes transplanted into subcutaneous connective tissue.

METHODS: Fresh isolated fetal rat cardiomyocytes were slowly frozen and stored in liquid nitrogen for 4 weeks. The cells were then rapidly thawed and cultured. Adult rats were transplanted with cryopreserved cardiomyocytes, non-cryopreserved cardiomyocytes, or culture medium alone (N=5 per group), by injection into the subcutaneous tissue of the hindlimb. Contractility of the grafted cells was evaluated by echocardiography 2 and 4 weeks after transplantation. At 4 weeks,the graft and perigraft tissue was excised and cell survival and lymphocyte infiltration, a marker of the host immune response, evaluated by histology.

RESULTS: Two weeks after transplantation, a block of spontaneously contractile tissue was observed at the site of transplantation in animals injected with cryopreserved (60±19bpm) and non-cryopreserved (34±2 bpm) cardiomyocytes, but not in control animals injected with culture medium alone. Histologic examination revealed engrafted cells in all transplanted animals but not in control rats. Perigraft lymphocyte infiltration was significantly greater around the non-cryopreserved cardiomyocytes than the cryopreserved cells.

CONCLUSIONS: Cryopreserved cardiomyocytes survived and demonstrated spontaneous contractile activity after transplantation into the connective tissue of rat hindlimbs. Cryopreservation may have reduced the immunogenicity of the transplanted cells.

**By Invitation*

F18. Expression of Human Angiopoietin Gene Determines Degree of Pulmonary Vascular Resistance in Patients Undergoing Pulmonary Thromboendarterectomy

Patricia A. Thistlethwaite*, Sang Lee*, Paul Wolf*, David P. Kapelanski* and Stuart W. Jamieson, San Diego, California

OBJECTIVE: A consistent pathologic feature seen in lungs of patients with pulmonary hypertension from thromboembolic disease is hyperplasia of the media of pulmonary arterioles. The molecular factors responsible for these vessel wall changes are unknown. Angiopoietin is a gene responsible for the formation of the media of blood vessels in utero. We hypothesized that aberrant expression of the angiopoietin gene in the adult lung would be a major contributing factor in the development of pulmonary hypertension.

METHODS: From January-September 1999 thirty-five patients (18 males, 17 females; mean age 52 years) with pulmonary hypertension and pulmonary vascular resistance ranging from 407 to 2006 underwent pulmonary thromboendarterectomy at our institution. Prior to cardiopulmonary bypass, lung biopsies were taken from each patient. Biopsies were also obtained from 10 patients (5 females, 5 males; mean age 55 yrs) undergoing lung resection for causes other than pulmonary hypertension. All specimens were blindly scored by a pathologist for degree of medial hyperplasia. Quantitative reverse transcriptase polymerase chain reaction, Western blot, and immunohistochemistry were used to quantitate angiopoietin mRNA and protein in each sample.

RESULTS: Lung specimens from all patients with pulmonary hypertension demonstrated upregulation of angiopoietin at the mRNA level. The degree of angiopoietin transcription was directly proportional to the pre-operative pulmonary vascular resistance and medial wall hyperplasia in each patient. By immunohistochemistry, angiopoietin protein was confined to the media of pulmonary arterioles. Lung biopsies from patients without pulmonary hypertension had no detectable expression of angiopoietin at mRNA or protein level.

CONCLUSIONS: Angiopoietin, agene responsible for vessel development in the embryonic lung, is upregulated in the lung parenchyma of patients with pulmonary hypertension. This gene defines, for the first time, a molecular step in the pathogenesis of pulmonary hypertension and serves as a target for strategies to treat this disease.

**By Invitation*

F19. Prospective Randomized Neurocognitive and S-100 Study of Hypothermic Circulatory Arrest, Retrograde Brain Perfusion, and Antegrade Brain Perfusion for Aortic Arch Surgery

Lars G. Svensson*, Edward Nadolny*, Dana L. Penney*, Wendy A. Kimmel* and Richard S. D'Agostino*, Burlington, Massachusetts

OBJECTIVE: To determine the best method of brain protection during deep hypothermic circulatory arrest (DHCA) for arch repairs.

METHODS: We randomized 30 circulatory arrest (CA) patients to either DHCA alone, DHCA + retrograde brain perfusion (RBP) or antegrade perfusion (ANTE); 5 coronary bypass (CAB) were controls. Each patient underwent 51 neurocognitive scored tests preoperatively, 3-6 days post-

operatively, 2-3 weeks, and 6 months. Intraoperative (pre-bypass, end cooling, end CA, end bypass, and end OR) and postoperative 6-hourly S-100 blood levels for 48 hours and EEGs were obtained.

RESULTS: One RBP patient withdrew and one ANTE died before surgery. The 30-day and hospital survival rate was 100% and no patient suffered a stroke or seizure. CA times were not different (DHCA:RBP:ANTE) for 11 total arch repairs (mean 41.4 minutes, SD15,6 elephant trunk). Hemi-arch repairs (n=17) were quickest with DHCA (10.0 minutes, SD 3.6, p=0.011) and ANTE longest (23.8 minutes, SD 10.28, p=0.004). Of 28 CA patients, 96% had neurocognitive impairment at 3-6 days, but by 2-3 weeks, only 9% had a new deficit (1 DHCA, 1 RBP, 1 ANTE) and by 6 months, these three had recovered. In comparison of postoperative scores, DHCA did better than RBP in 6 of 7 significantly different ($p<0.05$) scores and versus ANTE 9 of 9. There were no S-100 level differences between CA groups, but levels were significantly higher versus CAB, particularly at the end of bypass ($p<0.0001$), and correlated with the 3-6 day neurocognitive Mental Control (MMS-R) (p=0.053); California Verbal Learning Test Recognition Trial (p=0.051) and Intrusion (p=0.031); and Block Design (WAIS-R) Scaled Score (p=0.027), but by 2-3 weeks, this was no longer significant. CA (p=0.01) and pump time (p=0.057) correlated with peak S-100 levels.
CONCLUSIONS: There is no neurocognitive advantage with RBP or ANTE, although RBP may potentially reduce the risk of stroke related to embolic material in a larger study if more strokes were to occur.

**By Invitation*

F20 Rebound Pulmonary Hypertension Following Inhaled Nitric Oxide Therapy: a Role for Endothelin-1

D. Michael McMullan*, Janine M Bekker*, Michael J. Johengen*, R. Scott Heidersbach*, Stephen M. Black* and Jeffrey R. Fineman*, San Francisco, California; Chicago, Illinois

Inhaled nitric oxide (iNO) is a selective pulmonary vasodilator, and a promising new therapy for perioperative pulmonary hypertension. However, life-threatening increases in pulmonary vascular resistance (PVR) have been noted upon its acute withdrawal. Recent data suggest that iNO decreases endogenous NO synthase (NOS) activity, which induces, in part, this rebound pulmonary hypertension. Both endogenous NO and endothelin-1 (ET-1) mediate pulmonary vascular tone, and co-regulate each other through an autocrine feedback loop. ET-linduces pulmonary vaso-constriction via the ETa receptor. However, the role of ET-1 in rebound pulmonary hypertension has not been studied.
OBJECTIVE: To study the role of ET-1 in rebound pulmonary hypertension, *in vivo,* in the intact lamb.

METHODS: 13 one-month-old lambs were mechanically ventilated and instrumented to measure vascular pressures and left pulmonary blood flow. An IV infusion of 0.9% saline (control, n=7) or PD 156707 (an ETa receptor blocker, 1.0 mg/kg/hr, n=6) was continued throughout the study period. iNO (40 ppm) was administered for 24 hours and acutely withdrawn. Before and during iNO, lung biopsies and plasma were obtained for NOS activity and cGMP concentrations (the 2nd messenger of NO-induced vasodilation).

RESULTS: In control lambs, iNO decreased left PVR by 26.2% ($P<0.05$). After 24 hours, tissue NOS activity decreased to 63.8% of pre-NO values ($P<0.05$). Upon withdrawal of iNO, PVR acutely increased by 77.8% in these lambs ($P<0.05$). In PD 156707-treated lambs, iNO induced a similar decrease in PVR (22.0%,$P<0.05$). However, after 24 hours, NOS activity did not change (96.0%), and upon withdrawal of iNO PVR did not change (-5.5%). iNO increased plasma cGMP

in both groups; however, the increase was augmented in PD 156707-treated lambs (58.6% (control) vs. 154.8%,P<0.05).

CONCLUSIONS: ETa receptor blockade preserves endogenous NOS activity during iNO therapy, and prevents the rebound pulmonary hypertension induced by its acute withdrawal. Further studies are warranted to delineate these regulatory mechanisms and to investigate the potential therapeutic role of ET blockade during iNO therapy.

**By Invitation*

WEDNESDAY MORNING, MAY 3, 2000

9:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY

ACQUIRED CARDIAC CONTROVERSIESRoom ?

Constitution Hall, Metro Toronto Convention Centre

1. Moderate Ischemic Mitral Regurgitation Should be Corrected at the time of CABG

Moderator: Stephen B. Colvin

Pro: Lawrence H. Cohn

Con: Robert A. Guyton

2. Aortic Valve Replacement Should be Performed through a Mini-sternotomy

Moderator: Nicholas T. Kouchoukos

Pro: Steven R. Gundry

Con: Craig R. Smith

3. Multivessel CABG Should be Done on the Pump

Moderator: O. Wayne Isom

Pro: Lynda L. Mickleborough
Con: Raymond Cartier

12:00 p.m. ADJOURN

WEDNESDAY MORNING, MAY 3, 2000

9:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY

GENERAL THORACIC CONTROVERSIESRoom 205

Metro Toronto Convention Centre

1. The NETT Trial is Essential.

Moderator: Joseph I. Miller, Jr.

Pro: Douglas E. Wood

Con: G. Alexander Patterson

2. Radical Lymphadenectomy is Necessary in the Management of Early Stage Lung Cancer

Moderator: Mark S. Allen

Pro: Robert J. Ginsberg
Con: F. Griffith Pearson

3. Invasive Staging is Mandatory in the Treatment of Resectable Esophageal Cancer

Moderator: Valerie W. Rusch

Pro: Mark J. Krasna
Con: Larry R. Kaiser

12:00 p.m. ADJOURN

**By Invitation*

9:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY

CONGENITAL HEART CONTROVERSIESRoom 201

Metro Toronto Convention Centre

1. Primary treatment of Pulmonary Atresia with Intact Ventricular Septum: Interventional Catheterization or Surgery

Moderator: Thomas L. Spray

Pro: Lee Benson
Con: Roger B. B. Mee

2. Coarctation of the Aorta: Primary Balloon vs. Primary Surgical Repair

Moderator: William G. Williams

Pro: Peter Lang

Con: John W. Brown

3. Ross Procedure is Superior to Valve Repair for Aortic Regurgitation

Moderator: Michel N. Ilbawi

Pro: Frank L. Hanley
Con: Richard A. Jonas

12:00 p.m. ADJOURN

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Rancho Mirage

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Oyer, Philip E

Reitz, Bruce A

Robbins, Robert C

Shumway, Norman E

Tiburon

Heydorn, William H

Torrance

Carey, Joseph S

Cukingnan, Ramon A

Moore, Thomas C

State, Davin

Victorville

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Walnut Creek

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Bartley, Thomas D

Denver

Campbell, David N

Clarke, David R

Eiseman, Ben

Grover, Frederick L

Harken, Alden H

Hopeman, Alan R

Paton, Bruce C

Pomerantz, Marvin

Rainer, W. Gerald

Englewood

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Littleton

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Mangiardi, Joseph L

Miami

Bolooki, Hooshang

Daughtry, Dewitt C

Greenberg, Jack J

Jude, James R

Kaiser, Gerard A

Papper, Emanuel M

Ripstein, Charles B

Subramanian, S

Thurer, Richard J

Wilder, Robert J

Miami Beach

Reis, Robert L

Spear, Harold C

Naples

Battersby, James S

Linberg, Eugene J

MacGregor, David C

Mundth, Eldred D

Smyth, Nicholas P D

Orlando

Kraeft, Nelson H

Tamarac
Mendelssohn, Edwin

Tampa
Angell, William W

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Seiler, Hawley H

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GEORGIA

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Mansour, Kamal A

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Augusta

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Geha, Alexander S

Goldin, Marshall D

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Head, Louis R

Hunter, James A

Ilbawi, Michel N

Jeevanandam, Valluvan

Karp, Robert B

Maywood
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Pifarre, Roque

Oak Brook

Javid, Hushang

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Springfield

Hazelrigg, Stephen R

Wellons, Harry A, Jr

Willowbrook

Leininger, Bernard J

Winnetka

Mackler, S Allen

INDIANA

Bloomington

O'Neill, Martin J, Jr

Fort Wayne

Ladowski, Joseph S

Indianapolis

Brown, John W

King, Harold

King, Robert D

Kittle, C Frederick

Mavroudis, Constantine

Michaelis, Lawrence

Montoya, Alvaro

Najafi, Hassan

Raffensperger, John

Replogle, Robert L

Snow, Norman J

Tatooles, C. J

Thomas, Paul A, Jr

Vanecko, Robert M

Warren, William H

Zajtchuk, Rostik

Elk Grove Village

Sullivan, Henry J

Evanston

Fre, Willard A

Rosengart, Todd K

Glencoe

Rubenstein, L H

Mandelbaum, Isidore

Siderys, Harry

IOWA

Cedar Rapids

Lawrence, Montague S

Levett, James M

Council Bluffs

Sellers, Robert D

Des Moines

Dorner, Ralph A

Phillips, Steven J

Zeff, Robert H

Iowa City

Behrendt, Douglas M

Ehrenhaft, Johann L

Richenbacher, Wayne E

Rossi, Nicholas P

Stanford William

KANSAS

Cunningham

Allbritten, Frank F, Jr

Lawrence

Miller, Don R

Mission Hills

Ashcraft, Keith W

Prairie Village

Holder, Thomas M

Shawnee Mission

Adelman, Arthur

Padula, Richard T

Wichita

Tocker, Alfred M

KENTUCKY

MAINE

Portland

Bredenberg, Carl E

Morton, Jeremy R

Rockport

Swenson, Orvar

Windham

Hiebert, Clement

MARYLAND

Baltimore

Attar, Safuh

Baker, R. Robinson

Baumgartner, William A

Cameron, Duke Edward

Gott, Vincent

Lexington

Crutcher, Richard R

Mentzer, Robert M, Jr

Todd, Edward P

Louisville

Austin, Erle H, III

Gary, Laman A, Jr

Mahaffey, Daniel E

Ransdell, Herbert Jr

LOUISIANA

Baton Rouge

Berry, B Eugene

Beskin, Charles A

Campti

Bloodwell, Robert D

Metairie

Oschner, Alton, Jr

New Orleans

Blalock, John B

DeCamp, Paul T

DeLeon, Serafin Y

Ferguson, T Bruce, Jr

Harrison, Lynn H, Jr

Hartz, Renee S

Hewitt, Robert L

Lindsey, Edward S

McFadden, P Michael

Mills, Noel L

Moulder, Peter V

Ochsner, John L

Schramel, Robert J

VanMeter, Clifford H

Webb, Watts R

Heller, J. Alex, Jr

Hankins, John R

Krasna, Mark J

McLaughlin, Joseph S

Michelson, Elliott

Watkins, Levi, Jr

Whitman Glenn J R

Bethesda

Swain, Julie A

Glenarm

Turney, Stephen Z

Lutherville

Salomon, Neal W

Reisterstown

Heitmiller, Richard F

Worton

Walkup, Harry E

MASSACHUSETTS

Boston

Adams, David H

Akins, Cary W

Aranki, Sary F

Austen, W. Gerald

Bougas, James A

Burke, John F

Cohn, Lawrence H

Collins, John J, Jr

Couper, Gregory S

Daggett, Willard M

Daly, Benedict D T

Del Nido, Pedro J

Ellis, F. Henry, Jr

Folkman, M Judah

Gaensler, Edward A

Grillo, Hermes C

Hilgenberg, Alan D

Springfield

Jonas, Richard A

Lahey, Stephen J

Lazar, Harold L

Levitsky, Sidney

LoCicero, Joseph, III

Mathisen, Douglas J

Mayer, John E

Mentzer, Steven J

Moncure, Ashby C

Rheinlander, Harold F

Sellke, Frank W

Shemin, Richard J

Sugarbaker, David J

Thurer, Robert L

Torchiana, David F

Vlahakes, Gus J

Wain, John C, Jr

Weintraub, Ronald M

Wright, Cameron D

Boylston

Okike, Okike N

Brookline

Berger, Robert L

Frank, Howard A

Burlington

Shahain, David M

Cambridge

Malcom, John A

Chestnut Hill

Laforet, Eugene G

Concord

Norman, John C

Dover

Blackm Harrison

Falmouth

McElvein, Richard B

Engelman, Richard M

Rousou, John A

Vineyard Haven

Malm, James R

Wellesley Hills

Cleveland, Richard J

West Newton

Neptune, Wildford B

West Roxbury

Barsamian, Ernest M

Khuri, Shukri F

Westport Harbor

Findlay, Charles W

Westwood

Scannell, J. Gordon

Williamstown

Wilkins, Earle W

Worcester

Vander Salm, Thomas J

MICHIGAN

Ann Arbor

Bartlett, Robert H

Bolling, Steven F

Bove, Edward L

Deeb, G. Michael

Gago, Otto

Greenfield, Lazar J

Kirsh, Marvin M

Morris, Joe D

Neerken, A John

Orringer, Mark B

Prager, Richard L

Sloan, Herbert E

Beverly Hills

Timmis, Hilary H

Detroit

Framingham

Bernhard, William F

Schuster, Samuel R

Medford

Desforges, Gerard

Needham

Woods, Francis M

North Andover
Cook, William A

Osterville

Buckley, Mortimer J

Shrewsbury
Moran John M

West Bloomfield
Arciniegas, Eduardo

MINNESOTA

Coon Rapids

Gannon, Paul G

Mendota Heights

Dennis, Clarence

Minneapolis
Arom, Kit V

Bolman, R. Morton, III

Emery, Robert W

Foker, John E

Garamella, Joseph J

Helseth, Hovald K

Molina, J. Ernesto

Nicoloff, Demetre M

Shumway, Sara J

Ward, Herbert B

Rochester

Allen, Mark S

Bernatz, Philip E

Danielson, Gordon K

Deschamps, Claude

Arbulu, Augustin

Pass, Harvey I

Silverman, Norman A

Steiger, Zwi

Stephenson, Larry W

Walters, Henry L, III

Wilson, Robert F

Grand Rapids
Harrison, Robert W

Rasmusen, Richard A

Tomatis, Luis A

St Joseph
Levine, Frederick H

Chesterfield
Bergmann, Martin

Columbia
Curtis, Jack J

Jones, James W

Silver, Donald

Walls, Joseph T

Fontenac
Strevey, Tracy E, Jr

Kansas City

Borkon, A Michael

Killen, Duncan A

Mayer, John H, Jr

Piehler, Jeffrey M

Reed, William A

VanWay, Charles W, III

St Louis

Barner Hendrick B

Connors, John P

Cooper, Joel D

Ferguson, Thomas B

Fiore, Andrew C

Flye, M Wayne

McGregor, Chirstopher G A

Mullaney, Charles J

Olsen, Arthur M

Orzulak, Thomas A

Pairolero, Peter C

Puga, Francisco J

Schaff, Hartzell V

Trastek, Victor F

Shorewood

Kiser, Joseph C

Stillwater

Kaye, Michael P

Waubun

DeNiord, Richard N

MISSISSIPPI

Carthage

Logan, William D, Jr

Jackson

Heath, Bobby J

Johnston, J. Harvey, Jr

Madison

Hardy, James D

MISSOURI

Bridgeton

Codd, John E

Huddelston, Charles B

Johnson, Frank E

Johnson, Robert G

Kouchoukos, Nicholas T

Lewis, J Eugene, Jr

McBride, Lawrence R

Naunheim, Keith S

Pasque, Michael K

Patterson, G. Alexander

Roper, Charles L

Sasser, William F

Sundt, Thoralf M

Willman, Vallee L

Webster Groves

Kaiser, George C

MONTANA

Missoula

Duran, Carlos Gomez

Oury, James H

NEBRASKA

Omaha

Fleming, William H

Schultz, Richard D

NEVADA

Las Vegas

Little, Alex G

NEW HAMPSHIRE

Center Harbor

Aaron, Benjamin L

Franconia

Taylor, Warren J

Hanover

Baldwin, John C

Lebanon

Short Hills

Hochberg, Mark S

Tenafly

Gerst, Paul H

Wyckoff

Adler, Richard H

NEW MEXICO

Albuquerque

Nugent, William C

Sanders, John H, Jr

Windham

Burbank, Benjamin

NEW JERSEY

Alpine
Holswade, George R

Belleville
Gerard, Franklyn P

Browns Mills
Fernandez, Javier

McGrath, Lynn B

Camden

Camishion, Rudolph C

DelRossi, Anthony J

Ft. Lee

Conklin, Edward F

Hackensack

Hutchinson, John E, III

Jersey City

Demos, Nicholas J

Millburn

Parsonet, Victor

Moorsetown

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, Issac

Swan, Kenneth G

Dietl, Charles A

Edwards, W. Sterling

Wernly, Jorge A

Buena Vista

Thal, Alan P

Santa Fe
Davila, Julio C

Santa Teresa
Glass, Bertram A

Silver City
Waddell, William R

NEW YORK

Albany

Moores, Darroch W.O

Bay Shore

Ryan, Bernard J

Bellport

Finnerty, James

Bronx

Attai, Lari A

Fell, Stanley C

Ford, Joseph M

Frater, Robert W M

Gold, Jeffrey P

Hirose, Teruo

Veith, Frank J

Brooklyn

Acinapura, Anthony J

Cunningham, Joseph N, Jr

Levowitz, Bernard S

Sawyer, Philip N

Buffalo

Bhayana, Joginder N

Guiraudon, Gerard M

Hoover, Eddie L

Lajos, Thomas Z

Salerno, Tomas

Pittstown
Garzon, Antonio A

East Amherst
Anderson, Murray

East Quogue

McCormack, Patricia M

Fayetteville

Budgen, Walter F

Effler, Donald B

Fishers Island
Baue, Arthur E

Floral Park
Crastnopol, Philip

Honeoye Falls

Craver, William L

Larchmont

Steichen, Felicien M

Lido Beach

Hines, George L

Millerton
Green, George E

New York
Altorki, Nasser K

Anagnostopoulos, C E

Bains, Manjit S

Bloomberg, Allan E

Boyd, Arthur D

Brodman, Richard F

Cahan, William G

Clauss, Roy H

Colvin, Stephen B

Culliford, Alfred T

Ergin, M Arisan

Friedlander, Ralph

Calloway, Aubrey C, Jr

Ginsberg, Robert J

Griepp, Randall B

Grossi, Eugene A

Tice, David A

Tyras, Denis H

Waters Paul F

Wichern, Walter, Jr

Wolff, William I

Plattsburgh
Potter, Robert T

Rochester
DeWeese, James A

Hicks, George L

Schwartz, Seymore I

Stewart Scott

Roslyn

Thomson, Norman B, Jr

Wisloff George

Saranac Lake
Decker, Alfred M, Jr

Slingerlands
Kausel, Harvey W

Staten Island

Adams, Peter X

Stony Brook

Soroff, Harry S

Syracuse

Brandt, Berkeley III

Khoman, Leslie J

Meyer, John A

Parker, Frederick, Jr

Valhalla

Moggio, Richard A

Reed, George E

Voorhees Ville

Foster, Eric D

NORTH CAROLINA

Ison, O. Wayne

King, Thomas C

Kirschner, Paul A

Krieger, Karl H

Lansman, Steven L

Litwak, Robert S

Martini, Nael

McCord, Colin W

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

Asheville

Betts, Reeve H

Bryant, Lester R

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

Kay, Earle B

Kirby, Thomas J

Loop, Floyd D

Lytle, Bruce W

McCarthy, Patrick M

Mee, Roger B. B

Petterson, Gosta B

Rice, Thomas W

Van Heeckeren, Daniel W

Columbus

Davis, J Terrance

Kakos, Gerard S

Meckstroth, Charles

Michler, Robert E

Williams, Thomas E, Jr

High Point
Mills, Stephen A

Morehead City
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

Pennington, D. Glenn

OHIO

Blacklick

Myerowitz, P. David

Chagrin Falls
Ankeney, Jay L

Cross, Frederick S

Cincinnati

Albers, JohnE

Callard, George M

Flege, John B, Jr

Gonzalez, Luiz L

Helmsworth, James A

Hiratzka, Loren F

Ivey, Tom D

Wilson, James M

Wright, Creighton B

Cleveland
Blackstone, Eugene H

Cosgrove, Delos M

Groves, Laurence K

Dayton
DeWall, Richard A

Delaware
Clatworthy, H. Williams, Jr

Grove City
Kilman, James W

OKLAHOMA

Jenks

LeBeck, Martin B

Oklahoma City

Elkins, Roland 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

Rosemont
Sink, James D

Carlisle
DeMuth, William E, Jr

Darby
McKeown, John J, Jr

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

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

Karl, Tom R

MacVaugh, Horace

Mannion, John D

Spotnitz, William D

Spray, Thomas L

Wechsler, Andrew S

Rydel
Frobese, Alfred S

Sewickley
Clark Richard E

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

Reed, Carolyn E

Sade, Robert M

Columbia

Almond, Carl H

Hilton Head Island

Humphrey, Edward W

Isle of Palms

Mullen, Donald C

Landrum

Stayman, Joseph W

Spartanburg

Utley, Joe R

TENNESSEE

Konxville

Blake, Hu Al

Pittsburg

Bahnson, Henry T

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

Ponitys, Robert G

Rams, James J

Siewers, Ralph D

Shochat, Stephen J

Watson, Donald C

Nashville

Alford, William, Jr

Bender, Harvey W, Jr

Drinkwater, Davis C

Gobbel, Walter G, Jr

Merrill, Walter H

Pierson, Richard N, III

Randolph, Judson G

Rankin, J. Scott

Sawyers, John L

Stoney, William S

Thomas, Clarence, Jr

TEXAS

Austin

Tyson, Kenneth R T

Dallas

Adam, Maurice

Estrera, Aaron S

Holland, Robert H

Brott, Walter H

Domm, Sheldon E

Memphis

Cole, Francis H

McBurney, Robert P

Pate, James W

Robbins, S Gwin, Sr

Rosenweig, Jacob

Reardon, Michael J

Reul, George J, Jr

Roth, Jack A

Safi, Hazim J

Walker, William E

Wukasch, Don C

Kemp

Davis, Milton V

Lubbock

Bricker, Donald L

Feola, Mario

Wallsh, Eugene

Marble Falls

Hood, R. Maurice

San Antonio

Calhoon, John H

Cohen, David J

Dooley, Byron N

Heaney, John P

Treasure, Robert L

Temple

Jessen, Michael E

Lambert, Cary J

Mack, Michael J

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

Horseshoe Bay

Sutherland, R. Duncan

Houston

Beall, Arthur C, Jr

Burdette, Walter J

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

Ott, David A

Overstreet, John W

Putnam, Joe B, Jr

Arlington

Klepser, Roy G

Aylett

Brindley, G. Valter, Jr

UTAH

Salt Lake City

Doty, Donald B

Jones, Kent W

Karwande, Shreekanth V

Liddle, Harold V

McGough, Edwin C

Mortensen, J D

Nelson, Russell M

VERMONT

Burlington

Leavitt, Bruce J

Richford

Grondin, Claude M

West Dover

Humphreys, George H, II

VIRGINIA

Altavista

Pierucci, Louis, Jr

Annandale

Alk, Bechara F

Burton, Nelson A

Lefrak, Edward A

Rittenhouse, Edward

Sauvage, lester R

Thomas, George I

Gwathmehy, Owen

Charolottesville

Dammann, John F

Daniel, Thomas M

Kron, Irving L

Minor, George R

Muller, William H, Jr

Nolan, Stanton P

Tribble, Curtis G

Fredericksburg

Armitage, John M

McLean

Conrad, Peter W

Comes, Mario N

Mills, Mitchell

Wallace, Robert B

Norfolk

Baker, Lenox D

Reston

Boyd, Thomas F

Richmond

Bosher, Lewis H, Jr

Brooks, James W

Lower, Richard R

WASHINGTON

Belfair

Jones, Thomas W

Bellingham

Varco, Richard L

Friday Harbor

Lawrence, G, Hugh

Kirkland

Mills, Waldo O

Mercer Island

Li, Wei-I

Poulsbo

Malette, William G

Seattle

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

Parkenburg

Tarnay, Thomas J

WISCONSON

Altoona

McEnany, M Terry

Madison

Chopra, Paramjeet S

Cochran, Richard P

Young, William P

Marshfield

Myers, William O

Ray, Jefferson F, III

Sautter, Richard D

Mequon

Narodick, Benjamin

Milwaukee

Almassi, G. Hossein

Haasler, George B

Johnson, W. Dudley

Litwin, S Bert

Olinger, Gordon N

Tector, Alfred

West Bend

Aldea, Gabriel S

Allen, Margaret D

Anderson, Richard P

Hill, Lucius D

Lupinetti, F. Mark

Manhas, Dev R

Mansfield, Peter B

Merendino, K. Alvin

Miller, Donald W, Jr

Gardner, Robert J

WYOMING

Teton Village

Kaunitz, Victor H

OTHER COUNTRIES

ARGENTINA

Buenos Aires

Favaloro, Rene G

Kreutzer, Guillermo O

AUSTRALIA

QUEENSLAND

Brisbane

O'Brien, Mark F

SOUTH AUSTRALIA

Beaumont

Sutherland, H D'Arcy

AUSTRIA

Leonding

Bruecke, Peter E

Salzburg

Unger, Felix H

Vienna

Wolner, Ernst

BELGIUM

Bertem

Sergeant, Paul T

Leuven

Flameng, Willem J

Lerut, Antoon E. M. R

BRAZIL

Rio de Janeiro

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

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

Meier, Milton A

SÃ£o Paulo

Jatene, Adib D

ONTARIO

Collingwood

Heimbecker, Raymond

London

McKenzie, F Neil

Menkis, Alan H

Novick, Richard J

Mansfield

Pearson, F. Griffith

North York

Goldman, Bernard S

Oakville

Allen, Peter

Ottwa

Hendry, Paul J

Keon, Wilbert J

Toronto

Barid, Ronald J

Bigelow, Wilfred G

Christakis, George T

Coles, John G

David, Tirone R

Feindel, Christopher M

Fremes, Stephen E

Keshavjee, Shaf

McKneally, Martin F

Mickleborough, Lynda L

Scully, Hugh E

Trimble, Alan S

Trusler, George A

Weisel, Richard D

Williams, William G

Westbrook

Lynn, R Beverley

QUEBEC

Montreal

Blundell, Peter E

ENGLAND

Bath, Avon

Belsey, Ronald

Cambridge

Kennedy, John H

Wallwork, John

Herts

Lennox, Stuart C

London

Braimbridge, Mark V

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

Helsinki

Harjula, Ari L. J

Kauniainen

Mattila, Severi P

Carrier, Michel

Chartland, Claude C. C

Chui, 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

Castadena, Aldo, R

Herrera-Llerandi, Rodolfo

Blondeau, Philip

Cabrol, Christian E. A

Carpentier, Alain F

Lacour-Gayet, Francois

Menasche, Philippe

Piwnica, Armand H

Planche, Claude

Weldon, Clarance S

Pessac

Couraud, Louis

GERMANY

Aachen

Messmer, Bruno J

Bad Oeynhausen

Korfer, Reiner

Berlin

Alexi-Meskshivili, Vladmir

FRANCE

Bordeaux

Fontan, Francis M

Bordeaux-Pessac

Baudet, Eugene M

Creteil

Loisance, Daniel

Le Plessis Robinson

Binet, Jean-Paul

Dartevelle, Phillippe G

Lyon

Champsaur, Gerard L

Marseille

Metras, Dominique R

Montpellier

Thevenet, Andre A

Paris

Bachet, Jean E

Milan

Peracchia, Alberto

Naples

Cortufo, Maurizio

Pisa

Bortolotti, Uberto

Rome

Marcelletti, Carlo

JAPAN

Kamakura

Suma, Hisayoshi

Kanazawa

Iwa, Takashi

Watanabe, Yoh

Kitakyushushi

Miyamoto, Alfonso T

Osaka

Hertzer, Roland

Freiburg

Beyersdorf, Friedhelm

Hannover

Haverich, Axel

Leipzig

Mohr, Freidrich W

Loiching

Sebening, Fritz

Munich

Borst, Hans G

Neuss

Bricks, Wolfgang H

GREECE

Kallithea, Athens

Palatianos, George M

HONG KONG

Aberdeen

He, Guo-Wei

IRELAND

Dublin

O'Malley, Eoin

ITALY

Bergamo

Parenzan, Lucio

Chieti
Calafiore, Antonio M

P.R. OF CHINA

Beijing

Ying-Kai, WU

PORTRUGAL

Canaxide

Melo, Joao Q

Coimbra

Antunes, Manuel J

Lisbon
Machafo Macedo, Manuel E M

Kawashima, Yasunaru

Kitamura, Soichiro

Matsuda, Hikaru

Okita, Yutaka

Sendai

Mohri, Hitoshi

Shinjuku-ku

Imai, Yasusharu

Tokyo

Kyoanagi, Hitoshi

Naruke, Tsugo

Wada, Juro J

KOREA

Seoul

Cho, Bum-Koo

MONACO

Monaco Cedex
Dor, Vincent

NETHERLANDS

Wassenaar

Brom, A Gerard

NEW ZELAND

Waiwera Auckland

Barratt-Boyes, Brian G

SWEDEN

Sollentuna

Bjork, Viking

Umea
Aberg, Torkel H

SWITZERLAND

Arzier

Hahn, Charles J

Lausanne
vonSegesser, Ludqig K

ROMANIA

Targu-Mures

Deac, Radu C

RUSSIA

Moscow

Bockeria, Leo A

SAUDI ARABIA

Riyadh

Al-Halees, Zohair

SCOTLAND

Edinburgh

Logan, Andrew

Glasgow

Wheatley David J

SPAIN

Barcelona

Aris, Alejandro

Murtra, Marcos

Madrid

Rivera, Ramiro

Santander

Revuelta, Jose Manuel

Pully

Naef, Andreas P

Zurich

Senning, Ake

Turina, Marko I

SYRIA

Damascus

Kabbani, Sami S

UNITED ARAB EMIRATES

Abu Dhabi

Todd, Thomas R J

VENEZUELA

Caracas

Tricerri, Fernando E

WEST INDIES

Grenada

Landymore, Roderick W

AMERICAN ASSOCIATION FOR THORACIC SURGERY CHARTER MEMBERS

E. Wyllis Andrews

John Auer

Edward R. Baldwin

Walter M. Boothby

William Branower

Harlow Brooks

Lawrason Brown

Kenneth Bulkley

Alexis Carrel

Norman B. Carson

Arthur A. Law

William Lerche

Howard Lilienthal

William H. Luckett

Morris Manges

Walton Martin

Rudolph Matas

E.S. McSweeney

Samuel J. Metzler

Willy Meyer (Founder)

J. Frank Corbett	James Alexander Miller
Armistead C. Crump	Robert T. Miller
Charles N. Dowd	Fred J. Murphy
Kennon Dunham	Leo S. Peterson
Edmond Melchior Eberts	Eugene H. Pool
Max Einhorn	Walter 1. Rathbun
Herman Fischer	Martin Rehling
Albert H. Carvin	B. Merrill Ricketts
Nathan W. Green	Samuel Robinson
John R. Hartwell	Charles 1. Scudder
George J. Heuer	William H. Stewart
Chevalier Jackson	Franz Torek
H.H. Janeway	Martin W. Ware
James H. Kenyon	Abraham O. Wilensky
Adrian V. S. Lambert	Sidney Yankauer

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 physician and a citizen of the United States of America or Canada, unless is 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 resent 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 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 habe 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-officio members of the Council of the Association.

Section 2. The Council may, for the purposes of Article IV, give status as officers of the Association to the individual members of an ad hoc Committee appointed by the Council.

Section 3. The President, Vice-President, Secretary and Treasurer shall be elected at the annual meeting of the Association and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for *a* one-year term of office and neither 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 that 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 meeting 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 President. The Council shall advance the Vice-President to the Presidency and appoint an interim 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 a 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 fro 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 VHI, 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 Nomination 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 Director. 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.

Section 13. The Editorial Advisory Committee shall consist of five members appointed by the council including the Secretary, who shall serve as Chairman. One member shall be appointed each year for a four year term. The committee shall have advisory oversight for all

official scientific publications of the Association and make recommendations to 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 form one fiscal year to another. The Association may, in any regularly convened meeting, vote a 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 expenses and shall be placed in aspecial 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 that thirty nor more than forty day s 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 no 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 Article 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 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 state 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 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 hall be $200.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Senior Members are exempt from dues.

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

Section 5. Active Members must subscribe to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY to retain their membership status.

Section 8. Subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY is optional for Senior Members.

Section 9. Bills for membership dues and for subscriptions to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY will be mailed to members by the Treasurer after the Annual Meeting.

ARTICLE XII. Parliamentary Procedure

Except where otherwise provided in these By-Laws or by law, all parliamentary proceedings at the meetings of this Association and its Council and Committees shall be governed by the then current Sturgis Standard Code of Parliamentary Procedure.

ARTICLE XIII. Amendments

Section 1. These By-Laws may be amended by a two-thirds vote of the members present and voting at an executive session of a properly convened annual or special meeting of the Association provided that the proposed amendment has been moved and seconded by not less than three members at a prior executive session of that meeting or a prior meeting of the Association.

Section 2. These By-Laws may be suspended in whole or in part for a period of not more than twelve hours by a unanimous vote of those present and voting at any regularly convened meeting of the Association.

As amended, April 1999

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
1999-New Orleans, LA..... President, Lawrence H. Cohn

GRAHAM EDUCATION AND RESEARCH FOUNDATION

13 Elm Street, Manchester, Massachusetts 01944, (978) 526-8330

President Tirone E. David, M.D.
Toronto, Ontario, Canada
Vice President Richard A. Jonas, M.D.
Boston, Massachusetts
Secretary-Treasurer William T. Maloney
Manchester, Massachusetts
Director James K. Kirklin, M.D.
Birmingham, Alabama

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, 47 young surgeons from 24 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. Peter Clarke 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. SI im 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. DeWet Lubbe 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	Anders Franco-Cereceda Stockholm, SWEDEN
48th	2000-01	Albertus Scheule Tuebingen, GERMANY

THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION

THIS IS YOUR FOUNDATION

Unlike other organizations to which you make philanthropic contributions, The Thoracic Surgery Foundation works directly for your specialty. The Foundation supports research and education initiatives to increase knowledge and enhance treatment of patients with cardiothoracic diseases; develops the skills of cardiothoracic surgeons as surgeon-scientists and health policy leaders; and, strengthens society's understanding and trust in the profession.

Your Foundation is making a difference in cardiothoracic surgery. This is possible only because of your support. The Foundation is entirely supported through private donations. So, please don't forget your gift to The Foundation. If you have not yet made your annual gift to your Foundation, now is the time! If you make an annual gift to The Foundation of appreciated stocks, bonds or mutual funds, you avoid capital gains tax and earn an income tax deduction by donating rather that selling these assets. This may be better for you than a gift of cash.

If you have been thinking of making a charitable contribution to TSFRE, this may be the time to consider a planned gift. Often, this type of giving enables an individual to give a larger gift at a cost that is actually lower than if the gift were to be made outright. You may also find that planned giving enables you to meet other personal financial goals while making significant charitable gifts.

You may give to The Foundation through a revocable instrument, such as a bequest in your will, or through an irrevocable instrument like a charitable lead trust or a charitable remainder trust. You may also give through a life insurance policy or your retirement plan. For more information about your annual gift or a deferred gift, contact Frank Kurtz, TSFRE Executive Director, 312/464-6100, extension 3425; FAX 312/527-6635; Email frank_kurtz@sba.com.

THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION

ORGANIZATION

The Thoracic Surgery Foundation for Research and Education was established eight years ago to identify and provide funding for 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 cardiothoratic diseases; develop the skills of cardiothoratic surgeons as surgeon-scientists and health policy leaders; and, strengthen society's understanding and trust in the profession.

2000 BOARD OF DIRECTORS

Robert B. Wallace, M.D., *President* Thomas J. Fogarty, MD

David B. Skinner, M.D., *Vice President* Richard G. Fosburg, MD

Andrew S. Wechsler, M.D., *Secretary* O. Wayne Isom, MD

Richard P. Anderson, M.D., *Treasurer* George C. Kaiser, MD

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L. Penfield Faber, M.D., *Councillor* F. M. "Mac" Mauney, Jr., MD

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George J. Magovern, MD Benson R. Wilcox, MD

Martin F. McKneally, MD

STAFF
Frank J. Kurtz, Ph.D. Lainie Castle

Executive Director Development Associate

Mari Glass

Administrative Assistant

COMMITTEES OF THE FOUNDATION

CORPORATE COMMITTEE

This committee seeks the support of industry to join with The Foundation members in an enlightened collaboration to sponsor and share in the results of our research programs.

Gary W. Akins, MD, *Chairman* Delos M. Cosgrove, III, MD

John C. Baldwin, MD Thomas J. Fogarty MD

Denton A. Cooley, MD George J. Magovem, MD

DEVELOPMENT COMMITTEE

AH fund raising activities of The Foundation are monitored and coordinated by this committee.

Gary W. Akins, MD Stanton, P. Nolan, MD

A. Robert Coidell, MD Cecil C. Vaughn, MD

Robert W. Jamplis, MD Robert B. Wallace, MD, *Chairman*

EDUCATION COMMITTEE

This committee organizes health care policy education for cardiothoraric surgeons. The committee works closely with the Kennedy School of Government of Harvard University in developing programs of continuing education for thoracic surgeons. These programs are supported by the Alley-Sheridan Fund.

William A. Baumgartner, MD Robert L. Replogle, MD

Stanley W. Dziuban, Jr., MD Richard G. Rouse, MD

Sidney Levitsky MD Miles Shore, MD

Douglas J. Mathisen, MD Paul N. Uhlig, MD

Jack M. Matloff, MD, *Chairman* Harold C. Urschel, Jr., MD

John E. Mayer, Jr., MD

EXECUTIVE COMMITTEE

Richard P. Anderson, MD L. Penfield Faber, MD

Delos M. Cosgrove, III, MD Robert B. Wallace, MD, *Chairman*

James L. Cox, MD Andrew S. Wechsler, MD

FINANCE COMMITTEE

This committee oversees all finances of The Foundation. Each of the thoracic surgical organizations is represented by its treasurer and one other member of the organization.

Richard P. Anderson, MD, *Chairman* William T. Maloney

James L. Cox, MD Harvey I. Pass, MD

Robert A. Guyton, MD D. Glenn Pennington, MD

Steven W. Guyton MD Andrew S. Wechsler, MD

Renee S. Hartz, MD

FOUNDATION SUBCOMMITTEE

This committee will be responsible for identifying and coordinating approaches to private foundations which include large established foundations, as well as those foundations which have been established by thoracic surgeons.

Cecil C. Vaughn, MD, *Chairman* I. Penfield Faber, MD

David S. Mulder, MD Edward Verrier, MD

JOINT COMMITTEE ON ALLOCATIONS FOR RESEARCH

AND EDUCATION PROGRAMS

This committee detemmines the goals of The Foundation relating to funding for research and education and recommends appropriate allocations to meet these goals. It comprises the chairs of the Research Committee, the Education Committee, and Finance Committee, the Development Committee and the Foundation's President.

Richard P. Anderson, MD Jack M. Matloff, MD

James L. Cox, MD Robert B. Wallace, MD, *Chairman*

Bartley P. Griffith, MD

MEMBERSHIP SUBCOMMITTEE

This committee will be responsible for developing initiatives to increase the number of individuals making contributions of less than $1,000 per year.

Joseph E. Bavaria, MD David A. Fullerton, MD

A. Robert Cordell, MD, *Chairman*

THE NEW CENTURY SOCIETY COMMITTEE

Members of this committee seek the financial support of their colleagues through annual gifts of $1,000 or more to The Foundation.

William A. Baumgartner, MD George L. Hicks, Jr., MD

Arthur C. Beall, Jr., MD Robert W. Jamplis, MD

John H. Bell, MD Sidney Levitsky, MD

Lawrence I. Bonchek, MD Alex G. Little, MD

Robbin Gerald Cohen, MD Christopher T. Maloney, MD

Lawrence H. Cohn, MD Robert M. Mentzer, MD

A. Robert Cordell, MD Stanton P. Nolan, MD, *Chairman*

James L. Cox, MD John L. Ochsner, MD

Ivan K. Crosby, MD Robert L. Replogle, MD

Richard M. Engelman, MD Richard J. Shernin, MD

Frederick L. Grover, MD Edward D. Verrier, MD

John W. Hammon, MD Robert B. Wallace, MD

RESEARCH COMMITTEE

This panel of nationally recognized researchers reviews The Foundation's grant application and provides recommendations to the Board of Directors for approval for funding.

Ralph J. Damiano, MD G. Alexander Patterson, MD

Hartley P. Griffith, MD, *Chairman* D. Glenn Pennington, MD

Alden H. Harken, MD Richard N. Pierson, ID, MD

Larry R. Kaiser, MD Valerie W. Rusch, MD

Joren C. Madsen,MD David H. Sachs, MD

James A. Magovern, MD Julie A. Swain, MD

Lynda L. Mickleborough, MD Ross M. Ungerleider, MD

PLANNED GIVING SUBCOMMITTEE

This committee is responsible for developing, publicizing and soliciting planned and major gifts to The Foundation.

Leonard L. Bailey, MD P.M. "Mac" Mauney, Jr., MD

L. Penfield Faber, MD Steve L. Mourning

George L. Hicks, Jr., MD Harold C. Urschel, Jr., MD

Robert W. Jamplis, MD, *Chairman*

THE THORACIC SURGERY FOUNDATION AWARDS

2000 RESEARCH AND EDUCATION AWARD RECIPIENTS

* Individual Research Investigator Grants

* Research Fellowship Awards

* Career Development Awards

* Alley-Sheridan Scholarships

Thoracic Surgery Foundation Research Fellowship Award provides salary support to surgeons and surgical trainees who wish to acquire investigational skills.

Raja S. Mahidhara, M.D., University of Pittsburgh

Steffen Pfeiffer, M.D., Vanderbilt University Medical Center

Wilson Y. Szeto, M.D., Hospital of the University of Pennsylvania

Mohan Thanikachalam, M.D., University of Miami

The Thoracic Surgery Foundation Research Grant provides operational support of original research projects by cardiothoracic surgeons who have completed their formal training and who are certified or eligible by The American Board of Thoracic Surgery or its equivalent.

Paul M. Kirshbom, M.D., Children's Hospital of Pennsylvania

Thomas K. Waddell, Ph.D., M.D., Toronto General Hospital and the University of Toronto

Nina S. Braunwald Career Development Award provides salary support to women in academic cardiothoracic surgery at early stages of their faculty careers.

Lynne A. Skaryak, M.D., University of Massachusetts Medical Center

Alley-Sheridan Scholar-in-Residence at Harvard
The Foundation offers Alley-Sheridan tuition scholarships for cardiothoracic surgeons to pursue a year of study in health care policy at Harvard University. The following individual has been awarded a scholarship to attend in 2000.

Juan A. Sanchez, M.D., Lexington, KY

PREVIOUS RESEARCH AWARD RECIPIENTS

The Thoracic Surgery Foundation Research Fellowship

Edward M. Boyle, Jr., M.D., The University of Washington

Sitaram M. Emani, M.D., Duke University Medical Center

Seth Force, M.D., The University of Pennsylvania

Julie R. Glasson, M.D., Stanford University School of Medicine

Joseph H. Gorman, III, M.D., Hospital of the University of Pennsylvania

Daniel Kreisel, M.D., University of Pennsylvania

Baiya Krishnadasan, M.D., University of Washington

Paul C. Lee, M.D., University of Pittsburgh

Sang H. Lee, M.D., University of California, San Diego Medical Center

Robert S. Poston, Jr.,M.D., Stanford University Medical Center

Andrew J. Sherman,M.D., Northwestern University Medical School

Christopher L. Skelly, M.D., The University of Chicago

Michael A. Smith, M.D., Washington University

Vinod H. Thourani, M.D., Emory University School of Medicine

Tomasz A. Timek, M.D., Stanford University

Edward Yiming Woo, M.D., University of Pennsylvania

Baxter Healthcare Corporation Research Fellowship Award provides salary support to surgeons and surgical trainees who wish to acquire investigational skills.

Richard W. Kim, M.D., Yale University School of Medicine

The Thoracic Surgery Foundation Research Grant

James S. Allan, M.D., Massachusetts General Hospital

Richard P. Embrey, M.D., The Medical College of Virginia

Joren C. Madsen, M.D., Massachusetts General Hospital

John D. Mannion, M.D., Thomas Jefferson University

Si M. Pham, M.D., University of Pittsburgh

Todd K. Rosengart, M.D., The New York Hospital - Cornell Medical Center

David S. Schrump, M.D., National Cancer Institute

Nina S. Braunwald Career Development Award

Margaret D. Allen, M.D., University of Washington School of Medicine

Mary C. Mancini, M.D., Louisiana State University Medical Center

Patricia A. Thistlethwatie, M.D., University of California - San Diego

Nina S. Braunwald Research Fellowship provides salary support to women who wish to acquire investigational skills.

Kathryn Quadracci Flores, M.D., Brigham and Women's Hospital

Melina R. Kibbe, M.D., University of Pittsburgh

Elizabeth N. Morgan, M.D., University of Washington

Elaine E. Tseng, M.D., Johns Hopkins Hospital

Jennifer Dale Walker, M.D., Medical University of South Carolina

PREVIOUS EDUCATION AWARD RECIPIENTS

Alley-Sheridan Scholar-in-Residence at Harvard

Edward J. Dunn, M.D., Milwaukee, WI

Edgar L. Feinberg, III, M.D., Lafayette, LA

Peter P. McKeown, M.D., Tampa, FL

Joseph J. McNamara, M.D., Honolulu, HI

Paul N. Uhlig, M.D., Wichita, KS

Alley-Sheridan Executive Course Scholars
The Alley-Sheridan Fund was established within The Thoracic Surgery Foundation by Mr. David Sheridan on behalf of his life-long friend and collaborator, Dr. Ralph Alley, to provide educational opportunities, especially in health care policy matters for cardiothoracic surgeons. This fund has been used to make a generous grant from The Foundation to the Kennedy School of Government at Harvard University to develop an intensive executive course in management and health care policy, Understanding the New World of Health Care: A Health Policy Program for Physicians, Trustees and Health Care Leaders. The Foundation named the following individuals to receive Alley-Sheridan Scholarships to attend this course.

May, 1996 Alley-Sheridan Executive Course Scholars

E. Pendleton Alexander, M.D., Washington, DC

Richard P. Embrey, M.D., Richmond, VA

Timothy J. Gardner, M.D., Philadelphia, PA

Keith S. Naunheim, M.D., St. Louis, MO

Anthony Louis Picone, M.D., Syracuse, NY

Keith Eric Sommers, M.D., Pittsburgh, PA

Clifford H. Van Meter, M.D., New Orleans, LA

April, 1997 Alley-Sheridan Executive Course Scholars

Aurelio Chaux, M.D., Los Angeles, CA

Stanley W. Dziuban, Jr., M.D., Albany, NY

Steven R. Hazelrigg, M.D., Springfield, IL

Bruce M. Toporoff, M.D., New Orleans, LA

November, 1997 Alley-Sheridan Executive Course Scholars

Daniel P. Harley, M.D., Baltimore, MD

Robert S.D. Higgins, M.D., Detroit, MI

Mitchell J. Magee, M.D., Springfield, IL

Peter C. Pairolero, M.D., Rochester, MN

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

Thomas RJ. Todd, M.D., Toronto, Ontario, Canada

Daniel W. van Heeckeren, M.D., Cleveland, OH

Henry L. Walters, III, M.D., Detroit, MI

March, 1998 Alley-Sheridan Executive Course Scholars

Thomas R. Calhoun, M.D., Houston, TX

Charles C. Canver, M.D., Madison, WI

Vincent R. Conti, M.D., Galveston, TX

Thomas E. Gaines, M.D., Knoxville, TN

D. Tyler Greenfield, M.D., Kingsport, TN

Frederick L. Grover, M.D., Denver, CO

Vassyl A. Lonchyna, M.D., Maywood, IL

Victor F. Trastek, M.D., Rochester, MN

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

November, 1998 Alley-Sheridan Executive Course Scholars

Kevin D. Accola, M.D., Orlando, PL

William A. Baumgartner, M.D., Balitmore, MD

Mark Ian Block, M.D., San Francisco, CA

J. W. Randolph Bolton, M.D., Temple, TX

Davene Brown, Granville, NY

John H. Calhoon, M.D., San Antonio, TX

Robert A. Lancey, M.D., Worcester, MA

Garth R. McDonald, M.D., Towson, MD
William C. Nugent, M.D., Lebanon, NH

James R. Reynold, M.D., Sioux Falls, SD

David M. Shahian, M.D., Burlington, MA

Mark S. Soberman, M.D., Washington, MD

Wilson W. Strong, Jr., M.D., Cedar Rapids, IA

March, 1999 Alley-Sheridan Executive Course Scholars

Margaret D. Allen, M.D., Seattle, WA

Alan G. Casson, M.D., Halifax, Nova Scotia

Michael D. Crittenden, M.D., West Roxbury, MA

R. C. Stuart Finney, M.D., Towson, MD

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

Joseph S. Mclaughlin, M.D., Baltimore, MD

Hiep Nguyen, M.D., Newark, DE

Craig R. Saunders, M.D., Newark, NJ

Edward B. Savage, M.D., Chicago, IL

Lars G. Svensson, M.D., Boston, MA

Scott J. Swanson, M.D., Boston, MA

Michael K. Wood, M.D., Burlingame, CA

Edward R. Zech, M.D., Bethesda, MD

November, 1999 Alley-Sheridan Executive Course Scholars

Mark S. Allen, M.D., Rochester, MN

William R. Berry, M.D., Napa, CA

Nora L. Burgess, M.D., San Francisco, CA

Andrea J. Carpenter, M.D., Lackland AFB, TX

David J. Cohen, M.D., Fort Sam Houston, TX

Thomas A. D'Amico, M.D., Durham, NC

Richard N. Edie, M.D., Philadelphia, PA

Rafael Espada, M.D., Houston, TX

Peter J. Horneffer, M.D., Towson, ME

Wade Leon Knight, M.D., Temple, TX

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

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

Richard J. Novick, M.D., London, ON

Mark E. Sand, M.D., Orlando, FL

Sanjeev Sharma, M.D., Tucson, AZ

Mark S. Slaughter, M.D., Oak Lawn, IL

Scott J. Swanson, M.D., Boston, MA

AMERICAN ASSOCIATION FOR THORACIC SURGERY

RESEARCH SCHOLARSHIP

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

EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

1986-1988 Mark K. Ferguson

University of Chicago, Department of Surgery

ALFRED BLALOCK RESEARCH SCHOLARSHIP

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

1988-1990 Gus J. Vlahakes

Massachusetts General Hospital and Harvard Medical School

JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

"Load-Independent Assessment of Cardiac Performance by Noninvasive Means"
1990-1992 Donald D. Glover

Duke University Medical Center

ALTON OCHSNER RESEARCH SCHOLARSHIP

"Experimental Cardiac Graft Arteriosclerosis: Pathogenesis and Prevention of Lesion Development"

1992-1994 David H. Adams

Brigham and Women's Hospital

ROBERT E. GROSS RESEARCH SCHOLARSHIP

"Role of Intra-Cellular Messenger cAMP and cGMP in Organ Preservation"

1994-1996 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 Norris Pierson, III

Vanderbilt University Medical Center

*Charter Member

+Board of Directors

†Director Emeritus

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 SCHOLARSHIP

"The Role of Respiratory Muscle Adaptation in Lung Volume Reduction Surgery"
1999-2001 Joseph B. Shrager, M.D.

Philadelphia, Pennsylvania

SECOND ALFRED E. BLALOCK RESEARCH SCHOLARSHIP

"CD-4 Lymphocytes and Cardiac Allograft Vasculopathy"
2000-2002 Abbas Ardehali

UCLA School of Medicine

"Monocyte-Endothelial Cell Interactions in Delayed Xenograft Rejection"
2000-2002 Thomas K. Waddell

University of Toronto and Toronto General Hospital

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

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 Cardiothoracic 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 LishanAklog

West Roxbury, MA

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

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 honoreeÃ•s name and biography is printed in the Journal of Thoracic and Cardiovascular Surgery.

SCIENTIFIC ACHIEVEMENT AWARD RECIPIENTS

1995 John W. Kirklin, Birmingham, Alabama

1998 Norman E. Shumway, Stanford, California

1999 Michael E. DeBakey, Houston, TX

2000 Denton A. Cooley, Houston, Texas