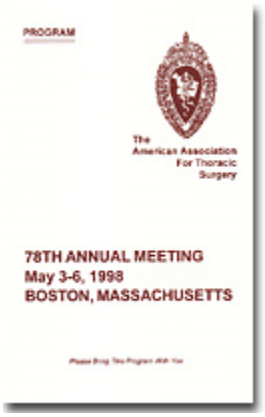
1998 ANNUAL MEETING PROGRAM



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DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

1998

AATS

Symposium

Congenital Heart Disease

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

Sunday, May 3, 1998
8:00 a.m. - 5:00 p.m.

Room 312

John B. Hynes

Convention Center

Boston, Massachusetts

Objectives:

The 1998 Congenital Heart Disease Symposium will address several topics including 1) the natural history and options for surgical management of patients with "congenital corrected" transposition of the great arteries, 2) the surgical management of patients with functional single ventricle beginning in the newborn period and extending through bidirectional cavopulmonary shunt and Fontan procedures with a discussion of the role of fenestration, 3) the options for surgical management of patients with failure of prior atrial level repairs of d-transposition of the great arteries including arterial switch procedures and transplantation, 4) the current status of cardiac and pulmonary transplantation, including the roles of living related lung transplantation and combinations of lung transplantation, including the roles of living related lung transplantation and combinations of lung transplantation with repair of cardiac defects, and 5) medical economic issues of interest to congenital heart surgeons. The symposium will provide attendees the opportunity to interact with recognized experts involved in the development and implementation of new techniques and procedures in these area of congenital heart disease. The format of the symposium will include lectures addressing these topics with expanded time for audience participation in the discussion of the various topics. At the completion of the symposium, participants should have an enhanced understanding of these areas of congenital heart disease management, of the problems which have been identified in follow-up following prior surgical management, and ol the newer techniques which have been designed to either avoid or manage these problems.

Registration:

The registration fee is $100 per person and includes the symposium, coffee breaks and lunch.

Accreditation:

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing education activity for 7 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

Symposium on Congenital Heart Disease

John B. Hynes Convention Center - Room 312
7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**

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

John E. Mayer, Jr., M.D., Chairman
Session I MANAGEMENT OF SINGLE VENTRICULAR PATIENTS

8:05 a.m. **Management of Neonates with Single Ventricle and Aortic Arch Obstruction by Arch Repair and Pulmonary Artery Band**

Hillel Laks, M.D., UCLA School of Medicine, Los Angeles, California

8:25 a.m. **Management of Neonates with Single Ventricle and Aortic Arch Obstruction by Stage I Palliative Operation**

Richard A. Jonas, M.D., The Children's Hospital, Boston, Massachusetts
8:45 a.m. **The Bidirectional Cavopulmonary Shunt: Should Every Single Ventricle Patient Have One?**

John J. Lamberti, M.D., Children' Hospital and Health Center, San Diego, California
9:05 a.m. **Early and Late Outcomes with Fontan Operations: Patient Selection Issues and the Role of Fenestration**

Francisco J. Puga, M.D., Mayo Clinic, Rochester, Minnesota

9:25 a.m. **Early and Late Outcomes of the Fenestrated Fontan Operation**

Nancy Bridges, M.D., Children's Hospital of Philadelphia, Philadelphia, Pennsylvania
9:45 a.m. **Experience with the Extracardiac Fontan Procedure**

Frank L. Hanley, M.D., University of California, San Francisco,San Francisco, California

10:05 a.m. **Panel Discussion**

10:20 a.m. **Coffee Break**

Session II COMPLEX TRANSPOSITION PROBLEMS

10:40 a.m. **Results of "Standard" Therapies for "Corrected" Transposition (L-TGA)**

Michael H. Freed, M.D., Children's Hospital, Boston, Massachusetts

11:00 a.m. **Double Switch Operation for Congenially Corrected Transposition**

Yasuharu Imai, M.D., Tokyo Women's Medical College, Shinjuku-ku, Japan

11:20 a.m. **Late Arterial Switch Operations for Failing Atrial Level Repairs of D-Transposition**

Roger B.B. Mee, M.B., Ch.B., FRACS, The Cleveland Clinic Foundation, Cleveland, Ohio

11:40 a.m. **Transplantation for Failing Atrial Level Repairs of D-Transposition**

Pedro J. Del Nido, M.D., Children's Hospital, Boston, Massachusetts

12:00 p.m. **Panel Discussion**

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

Session III TRANSPLANTATION

2:00 p.m. **Mechanical Circulatory Support in Cardiac Patients**

Brian Duncan, M.D., Children's Hospital, Seattle, Washington

2:20 p.m. **Early and Late Results of Heart, Heart Lung, and Lung Transplantation in Children**

Thomas L. Spray, M.D., The Children's Hospital of Philadelphia, Philadelphia, Pennsylvania

2:40 p.m. **Experience with Living Related Lung Transplantation in Children**

Vaughn A. Starnes, M.D., Childrens Hospital, Los Angeles, California

3:00 p.m.**Panel Discussion**

3:20 p.m.**Coffee Break**

Session IV ECONOMIC AND INSURANCE ISSUES IN CONGENITAL HEART SURGERY

3:40 p.m. **Factors Predicting High Cost in Congenital Heart Surgery**

Ross M. Ungerleider, M.D., Duke University Medical Center, Durham, North Carolina

4:00 p.m. **Strategies to Cope with Changing Insurance and Reimbursement for Congenital Heart Disease**

John E. Mayer, Jr., M.D., Children's Hospital, Boston, Massachusetts

4:20 p.m. **Panel Discussion**

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

1998

AATS

General Thoracic

Surgery

Symposium

Sponsored in

cooperation with

The General Thoracic

Surgical Club

"International Trends in General Thoracic

Surgery"

Co-Chairs:

Leslie J. Kohman, M.D.

Richard H. Feins, M.D.

Sunday, May 3, 1998

8:00 a.m.-5:30 p.m.

Room 311

John B. Hynes

Convention Center

Boston, Massachusetts

Objective:

The General Thoracic Surgery Symposium will comprehensively review the treatment of four major thoracic surgical problems. The subjects covered will be: lung cancer, esophageal cancer, malignant mesothelioma and lung volume reduction surgery for emphysema. For each of these subjects three internationally recognized authorities will discuss diagnosis, treatment options and future therapies. By having discussion from different parts of the world, the symposium should broaden and expand the understanding of treatment for each subject area. A panel discussion will follow each subject presentation to better define management options. Upon completion of this symposium, the participant should have a better understanding of each of the four subjects presented and be familiar with different innovative treatments from throughout the world.

Registration:

The registration fee is $100 per person and includes the symposium, coffee breaks and lunch.

Accreditation:

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

General Thoracic Surgery Symposium

John B. Hynes Convention Center - Room 311

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

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

Leslie J. Kohman, M.D., Co-Chair

Richard H. Feins, M.D., Co-Chair

Session I INTERNATIONAL PERSPECTIVES ON SURGERY FOR ESOPHAGEAL CANCER

Moderator: Leslie J. Kohman, M.D.

8:05 a.m. **The North American Perspective**

Mark B. Orringer, M.D., University of Michigan Medical Center, Ann Arbor, Michigan

8:35 a.m. **The Asian Perspective**

John Wong, M.D., Ph.D., University of Hong Kong Medical Center, Queen Mary Hospital, Hong Kong

9:05 a.m. **The European Perspective**

Antoon E.M.R. Lerut, M.D., Ph.D., University Hospital Leuven, Leuven, Belgium

9:35 a.m. **Panel Discussion**

10:05 a.m. **Coffee Break**

Session II INTERNATIONAL PERSPECTIVES ON SURGERY FOR LUNG CANCER

10:30 a.m. **The European Perspective**

Peter Goldstraw, M.D., Royal Brompton Hospital, London, England

11:00 a.m. **The Asian Perspective**

Tsuguo Naruke, M.D., National Cancer Center Hospital, Tokyo, Japan

11:30 a.m. **The North American Perspective**

Robert J. Ginsberg, M.D., Memorial Sloan-Kettering Cancer Center, New York, New York

12:00 p.m. **Panel Discussion**

12:30 p.m. **Lunch**

Session III INTERNATIONAL PERSPECTIVES ON MALIGNANT MESOTHELIOMA

Moderator: Richard H. Feins, M.D.

1:30 p.m. **The British Perspective**

Eric Butchart, M.D., University Hospital, Cardiff, Wales, United Kingdom

2:00 p.m. **The European Perspective**

Christian Boutin, M.D., Hospital de la Conception, Marseille, France

2:30 p.m. **The North American Perspective**

Valerie W. Rusch, M.D., Memorial Sloan-Kettering Cancer Center, New York, New York

3:00 p.m. **Panel Discussion**

Session IV INTERNATIONAL PERSPECTIVES ON LUNG VOLUME REDUCTION SURGERY

3:30 p.m. **The European Perspective**

Walter Weder, M.D., University Hospital, Zurich, Switzerland

4:00 p.m. **The Japanese Perspective**

Hiroshi Date, M.D., Okayama University School of Medicine, Okayama, Japan

4:30 p.m. **The North American Perspective**

Joel D. Cooper, M.D., Washington University, School of Medicine, St. Louis, Missouri

5:00 p.m. **Panel Discussion**

5:30 p.m. **Adjourn**

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

†Presenter has a relationship with Biovascular, Inc.

1998

AATS

Adult Cardiac Surgery Symposium

Chairman:

Delos M. Cosgrove, III, M.D.

Sunday, May 3, 1998

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

Ballroom B

John B. Hynes Convention Center

Boston, Massachusetts

Objective:

The 1998 Adult Cardiac Surgery Symposium will focus on the evolving issues in cardiac surgery and is divided into two sessions. The first session will entail the discussion of options for aortic valve surgery to include the following specific topics: actual versus actuarial analyses, comparison of porcine and pericardial valves and bileaflet valves, aortic valve repair secondary to aortic pathology and for leaflet prolapse, aortic homografts, the Ross Procedure and stentless valves. The second session will deal with Minimal Invasive Surgery to include aortic valve, mitral valve, aortic surgery, the Maze Procedure and CABG.

The symposium is designed for the practicing cardiac surgeon. At the completion of this symposium, participants should have an enhanced knowledge of the procedures using state-of-the-art techniques for minimally invasive adult cardiac surgery with a better understanding of the latest in options for aortic valve surgery which will enable them to better practice their specialty in the current and future environment of managed care.

Registration:

The registration fee is $100 per person and includes the symposium, coffee breaks and lunch.

Accreditation:

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

Adult Cardiac Surgery Symposium

John B. Hynes Convention Center - Ballroom B

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

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

Delos M. Cosgrove, M.D., Chairman

Session I OPTIONS FOR AORTIC VALVE SURGERY

8:00 a.m. **Actual Versus Actuarial Analyses**

Eugene H. Blackstone, M.D., Cleveland Clinic Foundation, Cleveland, Ohio

8:20 a.m. **Comparison of Porcine and Pericardial Valves**

Gary L. Grunkemeier, Ph.D., Providence Health System, Portland, Oregon

8:40 a.m. **Comparison of Bileaflet Valves**

David Naftel, Ph.D., University of Alabama, Birmingham, Alabama

9:00 a.m. **Panel Discussion**

9:20 a.m. **Aortic Valve Repair Secondary to Aortic Pathology**

Magdi H. Yacoub, M.D., Harefield Hospital, London, England

9:40 a.m. **Aortic Valve Repair for Leaflet Prolapse**

Delos M. Cosgrove, M.D., Cleveland Clinic Foundation, Cleveland, Ohio

10:00 a.m. **Panel Discussion**

10:15 a.m. **Coffee Break**

10:30a.m. **Aortic Valve**

Mark F. O'Brien, FRCS, Prince Charles Hospital, Brisbane, Australia

10:50 a.m. **Ross Procedure**

Nicholas T. Kouchoukos, M.D., Missouri Baptist Hospital, St. Louis, Missouri

11:10 a.m. **Stentless Valves**

Tirone E. David, M.D., Toronto General Hospital, Toronto, ON, Canada

11:30 a.m. **Panel Discussion**

12:00 noon **Luncheon**

Session II MINIMAL INVASIVE SURGERY

1:00 p.m. **Aortic Valve Surgery**

Steven R. Gundry, M.D., Loma Linda University Medical Center, Loma Linda, California

1:20 p.m. **Mitral Valve Surgery - Direct Access**

Lawrence H. Cohn, M.D., Brigham and Womens Hospital, Boston, Massachusetts

1:40 p.m. **Mitral Valve Surgery - Port Access**

Donald D. Glower, M.D., Duke University Medical Center, Durham, North Carolina

2:00 p.m. **Aortic Surgery**

Lars G. Svensson, M.D., Lahey Clinic, Burlington, Massachusetts

2:20 p.m. **Maze Procedure - Port Access**

James L. Cox, M.D., Georgetown University Medical Center,

Washington, D.C.

2:40 p.m. **Panel Discussion**

3:00 p.m. **Coffee Break**

3:20 p.m. **Current Morbidity and Mortality for CABG**

Bruce W. Lytle, M.D., Cleveland Clinic Foundation, Cleveland, Ohio

3:40 p.m. **Port Access CABG**

Bruce A. Reitz, M.D., Stanford University Medical Center, Stanford, California

4:00 p.m. **Off-Pump CABG**

Erik W. L. Jansen, M.D., Utrecht University Hospital, Utrecht,

The Netherlands

4:20 p.m. **CABG via Ministernotomy**

Donald B. Doty, M.D., IDS Hospital, Salt Lake City, Utah

4:40 p.m. **Panel Discussion**

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

The American Association for Thoracic Surgery
78TH ANNUAL MEETING
May 3-6, 1998
Hynes Convention Center
Boston, Massachusetts

MONDAY, MAY 4, 1998

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

Ballroom, Hynes Convention Center

8:15 a.m. PLENARY SCIENTIFIC SESSION

Ballroom, Hynes Convention Center

Moderators: Floyd D. Loop, M.D.

James L. Cox, M.D.

1. THIRTY YEARS OF CARDIAC TRANSPLANTATION AT ONE INSTITUTION.

Robert C. Robbins, M.D.*, Clifford Barlow, M.D.*, Philip F. Oyer, M.D., Sharon A. Hunt, M.D.*, Bruce A. Reitz, M.D., Edward B. Stinson, M.D. and Norman E. Shumway, M.D.

Stanford, California

Discussant: Bartley P. Griffith, M.D., Pittsburgh, Pennsylvania

We reviewed the records of all 865 patients (pts) who have received a cardiac transplant (Tx) at our center. Patients were divided into 3 groups (Gps) based on immunosuppression (IMS) received: Gpl = no cyclosporin (CSA) (n = 200, 1/68-11/80), Gpl = CSA (n = 269, 12/80 - 5/86), Gp3 = CSA + OKT3 (n = 396, 6/86-10/97). Important data relating to the groups are as follows (mean ± SD):

	Gp1	Gp2	Gp3
Recipient age (years) (range)	38 ± 12 (12-55)	39 ± 14 (0.1-63)	42 ± 19 (0.1-70)
Donor age (years) (range)	22 ±6 (13-41)	23 ± 7 (1-53)	27 ± 12 (0.5-54)
Wait list time (days)	44 ± 35	49 ± 69	157 ± 195
Length hospital stay (days)	69 ± 36	36 ± 23	21 ± 34
Time to first rejection (days)	36 ± 55	63 ± 175	95 ± 259

Actuarial analyses of short and long-term outcomes for the Gps are as follows:

		3 months	1 year	5 years	10 years	15 years	p value (ANOVA)
Survival (%)	Gp1	82	63	36	22	17	$p < 0.05$
	Gp2	87	80	59	39	22	
	Gp3	91	83	66	50	-	

Death from rejection (%)	Gp1	2	10	15	17	17	p < 0.05
	Gp2	2	5	7	8	8	
	Gp3	2	3	6	7	-	
Death from infection (%)	Gp1	14	29	47	51	55	p < 0.705
	Gp2	6	9	17	28	32	
	Gp3	3	*6*	10	14	-	
Death from lymphoid malignancy (%)	Gp1	-	-	2	9	13	NS
	Gp2	-	-	2	5	8	
	Gp3	-	-	1	2	-	
Death from coronary artery disease (%)	Gp1	-	-	4	11	16	NS
	Gp2	-	-	10	19	31	
	Gp3	-	-	8	10	-	

Retrospective HLA typing did not affect outcome. There are 141 pts who have survived > 10 years with the longest survivor being 23 years post Tx. There have been 65 re-Tx in 63 pts with 1, 5, and 10 year actuarial survivals of 51, 25, and 18 % respectively. Conclusions: The evolution of three decades of experience with cardiac Tx has resulted in an improved overall survival of pts who are currently at both extremes of age, wait longer for Tx, and have more complex medical conditions. The incidence of death from infection and rejection has decreased with time as a result of improved IMS and treatment. Continued advances in peri-operative management and IMS could further refine this initial Tx experience and improve the survival and quality of life of pts with end-stage heart failure.
*By invitation

2. RESULTS OF FIRST 100 CASES OF COMPLETE REPAIR OFCONGENITAL HEART DEFECTS IN PATIENTS WEIGHING700 TO 2500 GRAMS.

V. Mohan Reddy, M.D.*, Doff B. McElhinney, M.S.*, Theresa Sagrado, B.A.*, Andrew J. Parry, M.D.*, David F. Teitel, M.D.*, Norman J. Silverman, M.D.* and Frank L. Hanley, M.D.

San Francisco, California

Discussant: Roger B.B. Mee, M.B., Ch.B., Cleveland, Ohio

Infants born with congenital heart disease are more likely than normal children to be small for gestational age at birth. Published data suggest that low birth weight is a risk factor for corrective surgery for many cardiac defects. Congenital heart defects in this patient population are typically managed with supportive therapy or palliative surgery, and definitive repair is delayed. However, the morbidity of such an approach has been shown to be high. Since 1990, complete repair of congenital heart defects (other than isolated patent ductus arteriosus) has been performed in 102 infants ‰¤ 2500 g (median 2100 g, range 700-2500 g), including 16 who weighed ‰¤ 1500 g. Defects included ventricular septal defect with other left to right shunt lesions (23), tetralogy of Fallot (19), transposition complexes (13), coarctation of the aorta (12), interrupted aortic arch (9), truncus arteriosus (8), atrioventricular septal defect (6), total anomalous pulmonary venous return (4), and other defects (8). Preoperative morbidity was more common in patients who were referred late for corrective surgery. Standard techniques of neonatal cardiopulmonary bypass were used, including circulatory arrest in 17

pts. There were 10 early deaths (10%), due to cardiac failure (4), arrhythmia (1), sepsis (1), idiopathic coronary artery intimal necrosis (1), foot gangrene (1), pulmonary hemorrhage (1), and technical error (1). No patient had evidence of post-bypass intracerebral hemorrhage. At a median follow-up of 36 months, there had been 6 late deaths and 7 patients had undergone surgical and/or catheter reintervention for subaortic stenosis (2), conduit obstruction (2), pulmonary artery stenosis (2), or recurrent coarctation (1). Among the first 50 patents, followed for > 3 yrs, there was no evidence of neurological sequelae. Median weight for age was at the 20th percentile, with a direct correlation between weight for age and birth weight ($p = 0.01$). In most cases, delay in repair of congenital heart defects in low and very low birth weight infants does not confer any benefit and is associated with a higher incidence of preoperative morbidity. Complete repair of both simple and complex congenital heart lesions can be performed successfully in low and very low birth weight infants with good early and medium term results. Postoperative growth is accelerated following repair and approximates the normal growth curve for low birth weight infants without congenital heart disease. It is recommended that such infants, especially when symptomatic, undergo early surgical correction rather than prolonged medical management or other forms of palliation.

*By invitation

3. CLINICAL EXPERIENCE WITH CARINAL RESECTION.

John D. Mitchell, M.D.*, Douglas J. Mathisen, M.D. Dean M. Donahue, M.D.*, Ashby C. Moncure, M.D. John C. Wain, M.D.*, Cameron D. Wright, M.D. and Hermes C. Grillo, M.D.
San Diego, California and Boston, Massachusetts

Discussant: Jean DesLauriers, M.D., Sainte-Foy, Quebec, Canada

Pathologic processes that involve the carina pose a tremendous challenge to thoracic surgeons. Techniques have been developed to allow primary resection and reconstruction. The procedures are demanding, management of patients complex, and the potential for complications high. Few institutions have accumulated sufficient experience to allow meaningful conclusions about indications and contraindications for surgery, morbidity and mortality rates. Since 1962 135 patients have undergone 143 carinal resections (134 primary resection, 9 re-resection) at our institution. Indications for carinal resection included bronchogenic cancer (58), other airway neoplasms (60), benign or inflammatory strictures (16). Thirty-seven patients had prior lung or airway surgery not involving the carina. Carinal resection without pulmonary resection was accomplished in 52 patients; 57 patients had carinal pneumonectomy (44 right, 13 left); 14 patients had carinal plus lobar resection; and 11 patients had carinal resection following prior pneumonectomy (9 left, 2 right). There were 15 different combinations of reconstruction. Techniques were employed to reduce anastomotic tension. The overall mortality in the 134 patients for primary carinal resection was 12.7% (17/134). Adult respiratory distress syndrome was responsible for 9 early deaths. Significant multivariate predictors of postoperative death included length of resected airway, development of anastomotic complications, and postoperative mechanical ventilation. Complications occurred in 35 patients (26%) including atrial arrhythmias (17), anastomotic complications (9) end pneumonia (7). Mortality by procedure and indication for surgery was as follows.

	Right Carinal Pneumon.	Left Carinal Pneumon.	Carina alone	Carina & Lobe	Prior Pneu-Monect.	Carinal Re-resect.	Lung Cancer	Other Neoplasms	Benign Strictures

N	44(7)	13(4)	52(4)	14(1)	11(1)	9(1)	58(9)	60(5)	16(3)
Mortality	15.9%	30.8%	7.7%	7.1%	9.1%	11.1%	15.5%	8.3%	18.7%

Left carinal pneumonectomy is associated with high operative mortality and consideration of the underlying pathologic process and chance for long-term survival must be carefully considered before recommending such a procedure. Carinal resection with preservation of lung and for low grade neoplasms is associated with acceptable mortality rates. Chance for long-term survival must be carefully balanced against operative mortality in recommending carinal resection for lung cancer.

*By invitation

4. PRIMARY AORTIC VALVE REPLACEMENT WITH HOMOGRAFTS OVER 25 YEARS: VALVE AND PROCEDURE RELATED DETERMINANTS OF OUTCOME.(106)

Ole Lund, M.D., Ph.D.*, Magdi Yacoub, F.R.C.S., DSc., V. Chandrasekaran, F.R.C.S.*, Richard Grocott-Mason, M.R.C.P., M.D.*, Hassan Elwidaa, M.R.C.P.* and Rashid Mazhar, F.R.C.S.*

London, England

Discussant: Mark F. O'Brien, M.D., Brisbane, Australia

Homografts offer many advantages over prosthetic valves, however, homograft durability varies considerably.

From 1969 through 1993, 618 patients aged 15-84 (mean 51) years underwent their first AYR with an aortic homograft. Concomitant surgery included root tailoring (N = 64), replacement of the ascending aorta (N = 56), coronary bypass grafting (N = 87). Homograft implantation was done using a ‘freehand' subcoronary technique (N = 551) or total root replacement (N = 67). The homografts were antibiotic sterilized (N = 479), cryo-preserved (N=12), or ‘homovitals' (nutrient medium, inserted within 72 h; N = 127). Maximum follow-up was 27.1 (mean 10.1) years.

Thirty-day mortality was 5.0% and 10- and 20-year crude survivals ± standard error (SE) 67 ± 2% and 35 ± 3%, respectively. Ten- and 20-year complication freedoms ± SE included: endocarditis 93 ± 1% and 89 ± 2%, respectively; primary tissue failure, 62 ± 3% and 18 ±3%, respectively; and re-do AYR, 81 ± 2% and 35 ± 4%, respectively. Multivariate COX analyses identified several valve and procedure related determinants which included: rising homograft donor age and antibiotic sterilized/cryopreserved homograft for mortality; donor > 10 years older than patient for endocarditis; rising donor minus patient age, rising implantation (from harvest to AYR) time, and donor age > 65 years for tissue failure; and rising donor minus patients age, young patient age, rising implantation time, subcoronary implantation, and aortic root tailoring for re-do AYR. Estimated 10- and 20-year freedoms from tissue failure for a 60-year-old patient with a ‘homovital' and no other risk factors were 88% and 52%, respectively, for a donor age of 30 years and 72% and 21%, respectively, for a donor of 60 years; the freedoms for a 30-year-old patient were 78% and 31%, respectively, for a 30-year-old donor and 56% and 7%, respectively, for a 60-year-old donor. Beneficial influences of a ‘homovital' were largely covered by short harvest time (0 for homografts from brain dead organ donors or heart transplant recipients) and short implantation time.

It is concluded that primary homograft AYR can give acceptable results for up to 25 years and that the late results can be improved by the use of a ‘homovital' valve, by matching patient and

donor age, and by more liberal use of a free root replacement with re-implantation of the coronary arteries rather than tailoring the root to accomodate a subcoronary implantation.

*By invitation

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

Jun Wang, M.D. Beijing, People's Republic of China

9:45 a.m. INTERMISSION - VISIT EXHIBITS

MONDAY, MAY 4, 1998

10:30 a.m. PLENARY SCIENTIFIC SESSION

Ballroom, Hynes Convention Center

Moderators: Lawrence H. Cohn, M.D.

James L. Cox, M.D.

5. THE FIRST GENERATION OF ENDOVASCULAR STENT-GRAFTS FOR DESCENDING THORACIC ANEURYSMS.

D. Craig Miller, M.D., R. Scott Mitchell, M.D.*, James I. Fann, M.D.* and Michael D. Dake, M.D.*
Stanford, California

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

From July 1992 to October 1997, 109 patients (mean age 68 years [range 34-89 years]) underwent endovascular stent-grafting of descending thoracic aortic disorders using a custom fabricated, self-expanding device. Follow-up was 100% complete, and averaged 21 months. Eighty-five (78%) patients had atherosclerotic aneurysms, 8 were due to aortic dissection, 8 post-traumatic, 5 anastomotic, 2 mycotic (healed), and 1 was degenerative. Twelve patients presented with aortic rupture. Fourteen (13%) patients had undergone one or more previous thoracic aortic surgical procedures. Over one-half of patients had been judged **not** to be reasonable candidates for "open" conventional surgical repair. Access was from the femoral artery in 60% of patients, abdominal aorta in 31%, iliac artery in 7%, and the aortic arch in 2%. Twenty-four patients underwent simultaneous abdominal aortic aneurysm repair. Complete thrombosis of the aneurysm was achieved in 104 (95%) patients (14 required post-procedure stent/graft extensions or coil embolization) prior to discharge. One patient died intraoperatively of aneursym perforation. Early mortality rate was 8 ± 3% (± 70% CL). The only multivariate independent risk factor for early death was smaller aortic diameter. Early complications included paraplegia in 4%, stroke in 3%, iliac artery avulsion in 2%, proximal aortic dissection in 2%, myocardial infarction in 1%, colon ischemia in 1%, and renal failure in 1%. Acturial survival estimates at 6 months, 1 year, and 5 years were 88%, 84%, and 77 ± 5% (± 1 SE), respectively. According to the intent to treat principle, "Treatment Failure" (conservatively defined as all late sudden, unexplained deaths, deaths due to aneurysm rupture, need for subsequent stent-grafting or operation, and endoleak) occurred in 18 patients (or 16%). Actuarial estimates of freedom from Treatment Failure at 1 and 5 years were 84% and 71 ± 8%, respectively. Emergency stent-grafting was the only independent risk factor for Treatment Failure. Three (3%) patients subsequently required definitive (open) surgical repair after 4, 43, and 61 months.

Conclusions: This 5 year clinical trial employing a relatively primitive stent/graft device indicates that endovascular descending thoracic aortic stent-grafting is feasible with acceptable medium-term results, which are comparable to the outcome after open operation; however, additional follow-up is still required to assess fully the long-term efficacy of this technique. The more refined, commercially-developed devices used today offer less traumatic and more precise stent/graft deployment; these major technical advantages, coupled with the important lessons we have learned over time, should be associated with even more salutary long-term clinical results in the future.

*By invitation

6. TWO INTERNAL THORACIC ARTERY GRAFTS ARE BETTER THAN ONE.

Bruce W. Lytle, M.D., John H. Arnold, M.D.*, Floyd D. Loop, M.D., Penny Whiteman, M.S.*, Robert W. Stewart, M.D., Patrick M. McCarthy, M.D. and Delos M. Cosgrove, M.D.
Cleveland, Ohio

Discussant: Hendrick B. Barnes, M.D., St. Louis, Missouri

Do bilateral ITA (BITA) grafts produce better outcomes than a single ITA graft (SITA)? To examine this issue for patients undergoing primary coronary artery bypass grafting, we reviewed the surgical strategies and outcomes for 2015 consecutive patients receiving BITA grafting with or without additional vein grafts and, for comparison, 8059 patients receiving SITA grafts and at least one vein graft. To achieve long follow-up intervals, patients were selected for review who underwent operation from 1971 through 1989. Treatment selection was not randomized and the BITA group had a lower mean age, included more men, fewer patients with diabetes, and more patients with triple vessel disease (all $p < 0.01$). The in-hospital mortality rate was 0.7% for both groups. Late follow-up (mean postoperative interval 10.3 years after BITA, 304 BITA patients with > 12 year follow-up) documented 51 reoperations and 177 percutaneous interventions (PTCA) after BITA grafting. Survival for the BITA group was 94%, 84%, and 67% and for the SITA group 91%, 79% and 64% at 5, 10 and 15 postoperative years, respectively ($p < 0.001$). Cox regression analyses were used to test patient and treatment related variables in multi-variate models for their associations with late outcomes and to establish the increases in relative risks associated with those variables. The table shows tests of significance and the adjusted risk ratios (p value [relative risk ratio]) for the patients in the SITA group relative to the BITA patients according to age group and outcomes. For all age groups, the risks of death and reoperation were higher for patients in the SITA group, except for the risk of death for patients < 50 years old.

	Late Death	Reoperation	Late Death or Reoperation	Late Death, Reoperation, or PTCA
All patients	<0.001 (1.31)	<0.001 (3.74)	<0.001 (1.61)	<0.001 (1.51)
< 50 years	N/S (1.05)	<0.001 (6.78)	<0.001 (1.77)	<0.001 (1.55)
50-60 years	0.002 (1.41)	<0.001 (3.58)	<0.001 (1.77)	<0.001 (1.56)
> 60 years	<0.001 (1.36)	0.001 (2.33)	<0.001 (1.44)	<0.001 (1.44)

Neither total arterial revascularization nor the specific vessels grafted with BITA improved results when compared to the general strategy of BITA grafting.

These data suggest that BITA grafting improved the survival rate over that for

SITA grafting in all age groups except for those < 50 years old. Futhermore, they provide strong evidence that BITA grafting substantially decreases the risk of reoperation for all age groups.

11:15 a.m. PRESIDENTIAL ADDRESS

"...The First Living and the Last Dying"

Floyd D. Loop, M.D., Cleveland, Ohio

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

*By invitation

MONDAY AFTERNOON, MAY 4, 1998

1:30 p.m. PLENARY SCIENTIFIC SESSION

Ballroom, Hynes Convention Center

Moderators: David B. Skinner, M.D.

Tirone E. David, M.D.

7. TECHNIQUES AND RESULTS OF MINIMALLY INVASIVE MITRAL VALVE SURGERY IN 100 PATIENTS: A PARADIGM FOR THE FUTURE.

†Lishan Aklog, M.D.*, ‡David H. Adams, M.D.*, Gregory S. Couper, M.D.*, Samuel Sears, B.A.* and Lawrence H. Cohn, M.D.

Boston, Massachusetts

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

Minimally invasive techniques for mitral valve (MV) surgery have recently been developed to decrease surgical trauma and presumably reduce pain, morbidity, recovery time and cost. We used a minimally invasive right parasternal approach to determine if this technique could match the efficacy of standard MV surgery.

Between 7/96 and 10/97, 100 patients underwent minimally invasive MV repair or replacement. They represented 75% of all patients undergoing isolated, primary MV surgery during that period. The patients ranged in age from 30 to 83 years (mean 58.2), 56% were male and 44% female. The mean New York Heart Association functional class was 2.4. The indication for surgery was mitral regurgitation in 90% and mitral stenosis in 10% The most common pathologic findings were myxomatous degeneration (76%), rheumatic disease (12%) and endocarditis (6%).

A 5-8 centimeter, right parasternal incision was performed with resection of a portion of two costal cartilage. Bypass was instituted via the femoral vessels vein and superior vena cava in the majority of patients. However, most recently, we have used direct cannulation of the ascending aorta and a percutaneous femoral venous cannula to avoid groin incisions. The MV was approached transeptally through the right atrium in 85 patients and directly into the left atrium through the interatrial groove in 15.

Eighty-five patients underwent MV repair using standard reconstructive techniques and an annuloplasty ring in most patients (76 Cosgrove, 3 Carpentier-Edwards). Fifteen patients underwent MV replacement (11 St. Jude, 3 Hancock, 1 Carpentier-Edwards). All repairs and replacements were performed under direct vision with standard instruments. No patient required conversion to median sternotomy. The mean ischemic and bypass times were 100 and 151 minutes. Transesophageal echocardiography was used in every patients to evaluate the operation and monitor de-airing of the heart.

There were no operative deaths. All repairs were successful and there were no paravalvular leaks after valve replacement. Perioperative morbidity included new onset atrial fibrillation in 14 of 69 patients (20%), one reoperation for bleeding (1%), and one transient ischemic attack (1%). Blood transfusions were required in only 39% of patients. There were no wound infectious. The median hospital length of stay was 5 days (range 3-30) with nearly 40% of patients staying 4 days or less. Only 7% of patients required discharge to a rehabilitation facility. There were two late deaths (2%) and one stroke (1%). One patient required reoperation and valve replacement four months after MV repair. Post-operative pain and rate of return to normality" were improved when compared to patients having standard median sternotomy. By decreasing surgical trauma, minimally invasive MV surgery appears to have an associated decrease in morbidity over standard approaches. It may also result in increased patient comfort, a better cosmetic result, a faster rate of recovery, decreased length of stay and decreased post-hospital costs. Most importantly, these results show that minimally invasive MV surgery maintains the quality and efficacy of MV surgery.

†1998 International Traveling Fellowship

‡1992-94 AATS Alton Ochsner Research Scholar

*By invitation

8. FACTORS INFLUENCING TEN YEAR SURVIVAL IN RESECTED STAGES I-IHA NON-SMALL CELL LUNG CANCER.

Nael Martini, M.D., Valerie W. Rusch, M.D., Manjit S. Bains, M.D., Mark G. Kris, M.D.*, Robert J. Downey, M.D.* and Robert J. Ginsberg, M.D.

New York, New York

Discussant: Mark K. Ferguson, M.D., Chicago, Illinois

The objectives of this study were to determine: a) If patients continue to have recurrence of the primary tumor beyond five years; b) If there is a difference of survival beyond five years between N0 vs. N1, N0 vs. N2, and N1 vs. N2; c) Whether histology, age, or sex influence survival beyond five years. From 1973 to 1989, 686 patients were identified to be alive and well five years following complete resection of their lung cancers. The stage of their disease was IA in 263, IB in 261, IIA in 12, and IIIA in 82 patients. The histology was adenocarcinoma in 328, bronchiolar carcinoma in 84, squamous cell carcinoma in 244, and large cell carcinoma in 30. The extent of pulmonary resection was a lobectomy or bilobectomy in 579, pneumonectomy in 55, and wedge resection or segmentectomy in 52. A recurrence and a new lung primary were considered as failures to remain free of lung cancer. Of the 686 patients, 26 developed late recurrence and 36 new lung cancers. The median followup on all patients was 122 months. The overall survival at 10 years was 92.4%. Survival by nodal status at 10 years was 93% for N0 tumors, 95% for N1 tumors and 90% for N2. Survival by stage at 10 years was 93% for Stage I tumors and 91% for Stage II or III tumors.

There were 369 patients who lived 10 or more years from treatment of their initial lung cancer. In these patients, the incidence of late recurrence was 3 of 369 patients or less than 1%, where as the incidence of new lung cancers, although, reduced was more frequent than late recurrences (N = 15). The conclusions are: 1) Late recurrence is infrequent. 2) Second primary cancers exceed recurrence after five years. 3) Age, sex, histology do not influence 10 year survival. 4) Once the patients survive beyond five years there is no difference in survival between N0 vs. N1 or N2. They behave the same after 5 years.

9. MOBILISATION OF THE LEFT AND RIGHT FIBROUS TRIGONES FOR RELIEF OF SEVERE LEFT VENTRICULAR OUTFLOW OBSTRUCTION.

Magdi H. Yacoub, F.R.C.S., DSc. And Rosemary Radley-Smith, F.R.C.P.*

Harefield, England

Discussant: Frank L. Hanley, M.D., San Francisco, California

The management of fixed subaortic stenosis including tunnel obstruction continues to be controversial with a variable number of patients reported to undergo aortoventriculoplasty or insertion of an extracardiac conduit from the apex. We believe that the "ideal" operation should relieve the obstruction while maintaining the anatomic integrity of the left ventricular outflow tract (LVOT). To achieve this, a technique of radical excision of the obstructive tissue combined with mobilisation of the two fibrous trigones has been devised. This results in backwards displacement of the subaortic curtain and anterior leaflet of the mitral valve with further widening of the LVOT. This technique has been used since 1971 in 52 patients aged between 5 months - 56 (mean 15.5 ± 10.6) years. Additional lesions were present in 10; a ventricular septal defect (VSD) was present in 7 - proximal (between the valve and ring) in 3 and distal to the ring in 4. Coarctation of the aorta, persistant ductus arteriosus and congenital mitral stenosis were present in 3 patients respectively. Three patients had a bicuspid non-stenotic aortic valve, 6 patients a tricuspid stenotic valve, 1 patient endocarditis on a normal aortic valve and 1 aortic regurgitation due to damage from a previous operation. Five patients had had 7 previous operations on the outflow tract. In addition to resection of the obstruction, 3 patients had an aortic valvotomy, 2 a homograft replacement of the aortic valve, 7 patients patch closure of VSD and 1 an open mitral valvotomy. There were 2 (4%) early deaths (both early in the series), one in an 18 month old infant with a proximal VSD and one in a 7 year old with extremely poor LV function pre-operatively. With a followup of 1-25 years, no patient has required re-operation for recurrence of the subaortic obstruction. There has been 1 sudden late death 16 years after operation. All patients have echocardiographic evidence of good relief of the obstruction. It is concluded that mobilisation of the 2 trigones is a safe operation and gives lasting relief of the obstruction up to 25 years later.

2:30 p.m. BASIC SCIENCE LECTURE

The Future of Coronary Thrombosis Prophylaxis and Management

Eric J. Topol, M.D., Cleveland, Ohio

3:15 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

4:00 p.m. PLENARY SCIENTIFIC SESSION

Ballroom, Hynes Convention Center

Moderators: David B. Skinner, M.D.

Tirone E. David, M.D.

10. MULTICENTER STUDY OF STENTLESS VALVES IN SMALL AORTIC ROOTS: DO STENTLESS VALVES RULE OUT REPLACEMENT DEVICE MISMATCH?

Ulrik Hvass, M.D.*, George Palatianos, M.D.*, Romeo Frassani, M.D.*, Cesare Puricelli, M.D.* and Mark O'Brien, M.D.

Paris, France; Athens, Greece; Udine, Italy; Brisbane, Australia

Discussant: Edward D. Verrier, M.D., Seattle, Washington

Four Cardiac Centers participating in an on-going clinical study of stentless porcine valves evaluated the results in patients with small aortic roots (19 and 21 mm annulus).

Out of a cohort of 567 patients, 171 patients (30,1%) had a small aortic root. Patient population comprises 163 cases with calcified aortic stenosis, 8 with valvular insufficiency. Sixty patients presented associated lesions mitral or coronary. Eighteen were redo operations. Mean age was 72 ± 4.2. Forty-seven of the small aortic root patients had an annulus of 19 mm and 124 had a 21 mm annulus. The body surface area (BSA) was respectively 1.55 ± 0.2 and 1.78 ± 0.45.
Hemodynamic evaluation of the stentless valve comprised serial measures of mean gradients, effective orifice area and left ventricular mass reduction. Complication rates for secondary events were evaluated over a 6 year period. Hospital mortality was 3.5% (6 patients: 2 were emergency redo operations, 1 sepsis, 1 mitral annular disruption, 1 bleeding and 1 myocardial infarction). Mean gradients after the first year were, in patients with 19 mm and 21 mm annulus respectively of 9 ± 2 mmHg and 6 ± 1.7 mmHg. EOA was 1.45 ± 0.3 and 1.72 ± 0.4 cm^2. Gradients and surfaces remain stable throughout the study period. Aortic regurgitation is zero to trace.

Left ventricular mass at discharge and at 1 year were respectively 296g ± 127 and 215g ± 102 for patients with a 19 mm annulus and 281g ± 75 and 236g ± 15 for patients with a 21 mm annulus.
Complication rate for secondary events is low: 2 embolic events in patients with atrial arrhythmias, 2 operated valvular endocarditis, no structural valve deterioration.
These results show that with stentless valves, patients with small aortic roots undergoing aortic valve replacement demonstrate excellent clinical and hemodynamic results, with low gradients, resulting in significant left ventricular mass reduction, ruling out any replacement device mismatch in this increasing group of patients.
*By invitation

11. PET-FDG IMAGING IS EFFICACIOUS IN EVALUATING PULMONARY MALIGNANCIES.

Geoffrey M. Graeber, M.D., Naresh Gupta, M.D.* and Gordon F. Murray, M.D.

Morgantown, West Virginia
Discussant: Robert J. Ginsberg, M.D., New York, New York

Positron emission tomography (PET), when used with the intravenously administered radiopharmaceutical F-18 Fluorodeoxyglucose (FDG), has the potential to help in the evaluation of patients with lung cancer since the radio-pharmaceutical is concentrated by metabolically active cells. We conducted a retrospective evaluation of PET-FDG in 96 patients assessed at our institution over the last two years for suspected primary pulmonary neoplasms. PET-FDG results were compared with the findings of CT scans on the same patients. All patients underwent surgical exploration and/or resection of their malignancies. All sites of potential malignancy underwent biopsy and/or excision of the masses with subsequent complete pathologic evaluation. Results: A total of 96 patients with suspected or proven primary pulmonary malignancies were evaluated. The male: female ratio was 68: 28. The age range was 43-80 years. Sixty-six patients had histologically confirmed malignancies: 35 adeno-carcinoma, 14 non-small cell, 10 squamous cell, 2 small cell, and 5 had various other malignancies. Thirty had benign masses histologically. PET-FDG had an accuracy of detecting malignancy in pulmonary lesions of 92% (Sensitivity 97%, specificity 89%). A total of 111 surgically sampled sites were from lymph nodes. PET-FDG was accurate in

predicting the malignancy of nodes in 91% of instances whereas CT was correct in 64%. PET-FDG changed preoperative staging for "N" in 31/88 (35%) of patients (upstaged in 24 patients and downstaged in 7). Preoperative evaluation of lymph nodes using CT showed 17 enlarged (>1 cm) and consistent with malignant invasion; all 17 were predicted as true negatives by PET-FDG. Similarly six lymph nodes predicted by CT to be negative (<1.0 cm) were accurately detected by PET-FDG to have metastases. The sensitivity, specificity, and predictive accuracy of PET-FDG in detecting metastatic lymphadenopathy in mediastinal lymph nodes was 98%, 94% and 95% respectively. PET-FDG also changed "M" staging in 8 patients (6 with and 2 without metastases). Four patients were accurately shown to have osseous mets with negative or nonspecific bone scans. Three other patients with suspicious lesions on bone scan showed true negative PET-FDG findings. These initial results suggest that PET-FDG has high accuracy in identifying and staging patients with lung cancer. PET-FDG also appears to be more accurate in detecting metastatic mediastinal lymphadenopathy than CT scan.
*By invitation

12. INTERNATIONAL MULTI-CENTER APROTININ GRAFT PATENCY EXPERIENCE.

Jeffrey B. Rich, M.D.*, Edwin L. Alderman, M.D.*, Jerrold H. Levy, M.D.* and Hartzell Schaff, M.D. and the IMAGE Investigators.

Norfolk, Virginia; Stanford, California; Atlanta, Georgia and Rochester, Minnesota

Discussant: Stephen Westaby, M.D., Oxford, England

Background: Aprotinin, a serine proteinase inhibitor, reduces bleeding during coronary artery bypass graft (CABG) surgery. We examined the effects of aprotinin on graft patency, incidence of myocardial infarction (MI) and blood loss in patients undergoing primary CAGE surgery with cardiopulmonary bypass.

Methods: Patients (N = 870) from 13 international sites were randomized to receive intraoperative aprotinin (N = 436) or placebo (N = 434). Graft angiography was conducted a mean of 10.8 days postoperatively. Electrocardiograms, cardiac enzyme levels and blood loss and replacement were evaluated.

Results: In 796 assessable patients, aprotinin reduced the thoracic drainage volume by 43%($p<0.0001$) and the requirement for allogeneic red blood cell administration by 49% ($p<0.0001$). Of 703 patients with assessable saphenous vein grafts, occlusions occurred in 15.4% of aprotinin-treated patients versus 10.9% of placebo-treated patients ($p = 0.032$). At U.S. sites, occlusions were seen in 9.4% of the aprotinin-treated patients and 9.5% of the placebo-treated patients ($p = 0.720$). At European sites, occlusions occurred in 23.0% and 12.4% of the aprotinin- and placebo-treated patients, respectively ($p = 0.014$). When adjusting for risk factors which were found to be associated with vein graft occlusion, the aprotinin versus placebo risk ratio was reduced from 1.7 to 1.5 (90% confidence interval, 0.6-1.8). Aprotinin did not affect the occurrence of definite MI (aprotinin: 2.9% versus placebo: 3.8%) or mortality (aprotinin: 1.4% versus placebo 1.6%). **Conclusions:** Given the costs and risks associated with transfusions, aprotinin remains an important and safe approach to surgical blood conservation.

*By invitation

13. VIDEO-ASSISTED SURGICAL MANAGEMENT OF ACHALASIA OF THE ESOPHAGUS.

Robert J. Wiechmann, M.D.*, §Mark K. Ferguson, M.D.*, Michael J. Mack, M.D.*, Ronald J. Aronoff, M.D.*, Steven Hazelrigg, M.D.*, Keith S. Naunheim, M.D.* and Rodney J. Landreneau, M.D.
Pittsburgh, Pennsylvania; Chicago, Illinois; Dallas, Texas; Springfield, Illinois; St. Louis, Missouri

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

Video-assisted surgical approaches to esophageal achalasia continue to be explored by many thoracic and general surgeons involved in the management of this esophageal motor disorder. We report our experience with thoracoscopic (VATS) and laparoscopic (LAP) esophagomyotomy to more clearly define the efficacy and safety of these approaches. Over a 70 month period, 54 patients with manometrically confirmed achalasia underwent VATS myotomy (n = 1 9) alone or LAP myotomy (n = 35) with partial fundoplication (D'or=15; Toupet = 20). Mean age was 47.9 years and average length of symptoms was 32 months. Prior management consisted of dilation in all patients, endoscopic botulinum toxin injection in 8 and prior myotomy in 1. Primary complaints were dysphagia-100%, respiratory-20%, weight loss-46%, and pain-31%. Mean esophageal diameter was 5.94 cm and tortuosity was present in 10% of patients. In the operating room all patients underwent endoscopic exam and evacuation of retained esophageal contents. The esophagomyotomy was routinely extended to the level of the lower pulmonary vein and inferiorly to one cm beyond the lower esophageal sphincter. VATS and LAP procedures were successfully performed in all patients. Mean operative time was 172 minutes and hospital stay averaged 2.5 days. There were no operative mortalities. Operative complications consisted of 3 perforations identified intraoperatively and repaired endoscopically. Primary symptoms were improved in 96% of patients with mean dysphagia scores(range 0-10) changing from 8.8 to 0.73 (p = 0.001) at a mean follow-up 8.4 months. Post-op management included dilation in 3 patients and 2 patients required esophagectomy. Post-op reflux symptoms have been noted in 11% (2/19) of VATS and 6% (2/35) LAP patients. At this intermediate analysis, video-assisted approaches to correction of achalasia appear to be a reasonable alternative to extended medical therapy or open surgery. Conversion to an open approach should be considered when technical mishap or compromise of therapeutic benefit is of concern. Thoracic surgeons interested in this esophageal motor disorder should become familiar with these surgical approaches.
§1986-88 AATS Edward D. Churchill Research Scholar

*By invitation

TUESDAY MORNING, MAY 5, 1998

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

FORUM SESSION

SUPPORTED BY AN EDUCATIONAL GRANT FROM ST. JUDE MEDICAL, INC.

Ballroom, Hynes Convention Center

Moderators: Eric A. Rose, M.D.

F1. MYOCARDIAL TISSUE ENGINEERING WITH AUTOLOGOUS MYOBLAST IMPLANTATION.

Julia Dorfman, M.D.*, Minh Duong, B.Sc.*, Audrius Zibaitis, M.D.*, M.Sc., Marc Pelletier, M.D.* and Ray C.-J. Chiu, M.D., Ph.D.

Montreal, Quebec, Canada

Implanting autologuos skeletal muscle-derived myoblasts, i.e. satellite cells, for myocardial replacement has many advantage, compared to other approaches of implanting either fetal cardiac myocytes (ethical and donor availability issues) or established cell lines (oncogenicity). Furthermore, autologous myoblasts do not require immunosuppression. The feasibility of satellite cells implanted into the myocardium differentiating into muscle fibers was confirmed in this study using a unique cell labeling technique.

Methods: Myoblasts are isolated from the skeletal muscle of adult rats, and cultured in vitro to proliferate the number of cells. These cells are labelled with DAPI (4', 6-diamidino-2-phenylindole) which binds to the cellular DNA and to the protein tubulin, and form a fluorescent complex. One million labeled satellite cells are implanted into the myocardium of rats isogenic to the donors. The cells were injected into the left ventricular wall with the heart exposed. The cardiac specimens are harvested 1 to 4 weeks following myoblast implantation. Histological sections are examined under fluorescent microscopy.

Results: By examining the cell culture plates under fluorescent microscope, the labeling efficiency of satellite cells with DAPI is found to be nearly 100%. In 8 rat hearts, the progressive differentiation of implanted myoblasts from isolated cells to fully developed striated muscle fibers can be observed. These new muscle fibers clearly labeled with DAPI fluorescence grew adjacent to unlabelled native cardiac muscle fibers.

Discussion: Our earlier studies of implanting autologous canine satellite cells into cryo-injured myocardium indicated that these cells could differentiate into cardiad phenotype myocytes. The presence of intercalated disks in these fibers suggested that they could fuse with native cardiac fibers to supplement ventricular function. However, it had been difficult to firmly establish these findings using cell markers, proving these neo-myocardium had indeed been derived from the implanted myoblasts. This study shows that DAPI has excellent labeling efficiency for satellite cells in vitro and in vivo. For the first time, we are able to confirm that the satellite cells implanted into the myocardium did in fact differentiate into fully developed, labeled muscle fibers.

Conclusions: Because of the obvious advantages of using autologous donor myoblast cells, clinical application of this approach appears desirable, and may provide a novel strategy for the management of heart failure patients in the future.

*By invitation

F2. COMPLETE REVERSAL OF ISCHEMIC WALL MOTION ABNORMALITIES BY COMBINED USE OF GENE THERAPY WITH TRANSMYOCARDIAL LASER REVASCULARIZATION.

Umer Sayeed-Shah, M.D.*, Michael J. Mann, M.D.*, Jeffrey Martin, M.D.*, Sergey Grachev, M.D.*, Sharon Reimold, M.D.*, Rita Laurence, B.S.*, Victor Dzau, M.D.* and Lawrence H. Conn, M.D.

Boston, Massachusetts

Introduction: Transmyocardial Laser Revascularization (TMR) results in symptomatic improvement in patients with chronic ischemic heart disease. This effect is thought to result from an angiogenic response to TMR in the ischemic myocardium. Preliminary data from our laboratory indicate that TMR enhances both the efficiency of gene delivery and the degree of transgene expression with the direct myocardial injection of non-viral vectors. We hypothesized that direct myocardial injection of plasmid DNA encoding the gene for vascular endothelial growth factor-1 (VEGF-1) could enhance the revascularization achieved by TMR in ischemic hearts, resulting in improved cardiac function.

Methods: 29 Yorkshire pigs underwent the placement of an ameroid constrictor at the proximal left circumflex artery via a left mini-thoracotomy, and were allowed to recover. Group I, (Ischemic controls, n = 5) had no further intervention. Group II, (n = 4) underwent TMR in the area at risk at 6 weeks. Group III, (n = 5) underwent TMR at 6 weeks with 3 equidistant intramyocardial injections of 100µg of an expression plasmid encoding the gene for Î²-galactosidase (pSV-Î²-gal) surrounding each TMR site. Group IV, (n = 4) had sets of 3 equidistant injections of l00 µg of an expression plasmid encoding the gene for VEGF-1, (pSV-VEGF) without TMR at 6 weeks. Group V, (n = 5) had TMR with each site surrounded by 3 injections of 100µg of pSV-VEGF. All animals were harvested at 12 weeks. Six additional pigs underwent TMR with either pSV-0-gal or pSV-VEGF injections, and were harvested at 8 weeks.

Results: Left ventricular free-wall motion by transesophageal and epicardial echocardiography was assessed by a cardiologist in a blinded manner. Hearts were scored as normal (no regional wall motion abnormality, normal systolic function), or abnormal. All (0/5) of the ameroid alone hearts were abnormal, whereas 75% (3/4) of the TMR hearts, 60% (3/5) of the TMR-Î²-gal hearts, and 50% (2/4) of the VEGF hearts displayed evidence of persistent wall motion abnormality. In contrast, all (5/5) of the heart treated with TMP-VEGF displayed no evidence of wall motion abnormality. Only the TMR-VEGF hearts had a statistically significantly different rate of wall motion abnormality compared to untreated ischemic hearts ($p = 0.004$ by two-tailed Fisher's Exact Test). Of note, 2 out of 3 TMR-VEGF pigs examined at 2 weeks post-treatment had completely normal ventricular function, whereas 3 TMR-p-gal pigs examined at the same time point all had persistent abnormalities ($p>0.05$).

Conclusion: These results suggest that the combined use of TMR with direct injection of an expression plasmid encoding VEGF-1 completely reverses ischemic wall motion abnormalities at 6 weeks after therapy, and that the resolution of wall motion abnormalities may occur as early as 2 weeks after therapy.

*By invitation

F3. TRANSMYOCARDIAL LASER REVASCULARIZATION FAILS TO PREVENT LEFT VENTRICULAR FUNCTIONAL DETERIORATION AND ANEURYSM FORMATION AFTER ACUTE MYOCARDIAL INFARCTION IN SHEEP.

Ramin Malekan, M.D.*, Scott T. Kelley, M.D.*, Yasuyuki Suzuki, M.D.*, Carol Reynolds, M.D.*, Theodore Plappert, C.V.T.*, Martin St. John-Sutton, M.D.*, L. Henry Edmunds, Jr., M.D. and Charles R. Bridges, M.D., D.Sc.

Philadelphia, Pennsylvania

Transmyocardial laser revascularization (TMLR) seeks to nourish left ventricular (LV) myocardium from cavity blood. We tested the hypothesis that TMLR prevents subsequent LV functional deterioration and aneurysm formation after acute antero-apical myocardial infarction.

In 21 anesthetized Dorset-hybrid sheep, snares were placed around the distal left ant. descending and second diagonal coronary arteries and a flow probe was fitted around the ascending aorta. After baseline hemodynamic measurements and transdiaphragmatic echocardiograms 10 days later, myocardium supplied by snared arteries was perforated using a CO^2 laser (4 channels/cm^2; 29-35 transmural holes) immediately prior to infarction in 11 sheep. Ten control animals did not have TMLR. Tightened snares infarcted 23% of the LV mass. Hemodynamic and echocardiographic studies were repeated one hour and 2, 5 and 8 weeks after infarction.

Control animals developed large antero-apical aneurysms confirmed at autopsy. At 8 weeks stroke work decreased from 253 ± 78 to 139 ± 54 ergs x 10^3 ($p<0.01$); cardiac output decreased from 2.4 ± 0.7 to 1.6 ± 0.7 1/min ($p<0.01$); ejection fraction decreased from 47.4 ± 8.6 to 23.4 ± 5.8 % ($p<0.01$); left ventricular end diastolic pressure increased from 1.7 ± 1.0 to 5.7 ± 1.0 mmHg ($p<0.05$); end systolic volume increased from 28.3 ± 6.9 to 75.9 ± 18.8 ml ($p<0.01$) and end diastolic volume increased from 54.0 ± 11.3 to 98.3 ± 16.0 ml ($p<0.01$). Mean arterial and central venous pressures did not change significantly. Histology two days after TMLR showed holes filled with fibrin and scattered red cells. At 5 and 8 weeks the infarct consisted of dense collagen, fibroblasts and lymphocytes with occasional islands of viable myocytes in both groups. Laser holes could not be found. No significant ($p<0.05$) differences were found between TMLR and control sheep for any measurement at any time point after infarction; histologic sections also did not differ between groups at 5 and 8 weeks. We conclude that TMLR does not revascularize acute myocardial infarction sufficiently to attenuate LV functional deterioration and aneurysm formation.

*By invitation

F4. METHYLPREDNISOLONE REDUCES THE INFLAMMATORY RESPONSE TO CARDIOPULMONARY BYPASS IN NEONATES: TIMING OF DOSE IS IMPORTANT.

Andrew J. Lodge, M.D.*, Paul J. Chai, M.D.*, C. William Daggett, M.D.*, Ross M. Ungerleider, M.D. and James Jaggers, M.D.*

Durham, North Carolina

Introduction: Outcome after neonatal heart surgery is sometimes hampered by a severe inflammatory response to cardiopulmonary bypass (CPB). This can present as total body edema or pulmonary dysfunction. This study was designed to evaluate the efficacy of preoperative methylprednisolone (MP) administration in limiting this response and compares the effect of giving MP eight hours preoperatively to the more clinically common practice of adding MP to the CPB circuit prime. ***Methods:*** Three groups of neonatal piglets were studied. A control group (Control, n = 6) received no preoperative medication. One experimental group (PreOp-MP, n = 6) received methylprednisolone sodium succinate (30 mg/kg) both 8 hours and immediately preoperatively. Another experimental group (Prime-MP) received no preoperative treatment, but MP (30 mg/kg) was added to the CPB circuit prime. All animals underwent CPB and 45 minutes of deep hypothermic circulatory arrest. Hemodynamic and pulmonary function data were acquired prior to CPB (BSL) and at 30 (Post30) and 60 (Post60) minutes after CPB. ***Results:*** Post CPB lung water content was significantly lower in the PreOp-MP group compared to the control group ($p = 0.001$)

and the Prime-MP group (p = 0.026). Total body water gain was significantly less in the PreOp-MP group compared to the control group (p = 0.003) and the Prime-MP group (p = 0.01). Pulmonary function data are presented in the following table (results for each parameter were not significantly different at Post60 compared to Post30):

	Stage	Control	Prime-MP	PreOp-MP
Compliance	BSL	3. 05 ± 0.24	2.99 ± 0.22	3. 28 ± 0.28
(ml/cm H_2O)	Post30	1.59 ± 0.26*	2.43 ± 0.28*†	3.14 ± 0.30†‡
A-a Gradient	BSL	132 ± 12	139 ± 22	112 ± 10
(mmHg)	Post30	396 ± 49*	241 ± 31†	179 ± 25†
PVR	BSL	2503 ± 674	3268 ± 345	1766 ± 377
(dyne-sec-cm^{-5})	Post3 0	11421 ± 787*	7715 ± 1690*	3775 ± 429†‡

PVR: pulmonary vascular resistance; All results expressed as mean ± standard deviation; p values by ANOVA

*:p<0.01 vs. BSL †:p < 0.01 vs. Control ‡:p < 0.05 vs. Prime-MP

Conclusions: MP given eight hours and immediately before surgery provides superior protection from the inflammatory response to CPB compared to no treatment and to addition of MP to the CPB circuit prime. These results suggest a simple, available, cost effective means of reducing early post-CPB morbidity, especially if used in selected high risk patients.

*By invitation

F5. CONTINUOUS PERFUSION OF DONOR HEARTS IN THE BEATING STATE EXTENDS PRESERVATION TIME AND IMPROVES RECOVERY OF FUNCTION.

Waleed H. Hassanein, M.D.*, Lambros Zellos, M.D.*, Tracey A. Tyrrell, B.A.*, Nancy A. Healey, B.S.*, Michael D. Crittenden, M.D.*, Vladimir Birjiniuk, M.D.* and Shukri F. Khuri, M.D.

West Roxbury, Massachusetts

Improving methods of donor heart preservation may permit prolonged storage and remote procurement of cardiac allografts. We **hypothesized** that continuous sanguineous perfusion of the donor heart in the beating working state may prolong myocardial preservation. We developed a simple, portable perfusion apparatus that may be used for donor heart preservation. Contractile, metabolic, and vasomotor functions were monitored simultaneously in an isolated pig heart. Metabolic function was monitored by myocardial tissue pH. Vasomotor function was assessed in isolated coronary ring chambers. Hearts were randomized into three Groups: I (n = 5) cardioplegic arrest, 12hr storage @ 4°C using Modified Belzer's Solution, and 2hr sanguineous reperfusion in the working state, or II (n = 6) 12hr continuous perfusion in the beating working state, 30min arrest (to simulate time needed for re-implantation), and 2hr reperfusion, as above. Group III (n = 7) served as coronary rings controls. **Results** (m ± SD): At 2 hours of reperfusion LV developed pressure in Group II was higher than in I (90+6, 53+15mmHg, *P = 0.005).* Significantly less myocardial edema was observed in Group II vs I (73 ± 4, 80 ± 1% H_20 content, *P = 0.01*). Significantly less myocardial acidosis was noted in Group II vs I during preservation (pH 7.3 ± 0.01, 6.1 ± 0.03, *P<0.001*) and reperfusion (pH 7.3 ± 0.008, 6.8 ± 0.05, *P<0.001*).Coronary

endothelial vasomotor function was better preserved in Group II vs I as evidenced by the dose response relaxation of coronary rings to 10-^{8}M Bradykinin (37%, 55%ˆ† baseline, $P = 0.01$). **Conclusion:** This new method of heart preservation extends the current preservation limit and avoids time dependent ischemic injury, thereby allowing for distant procurement of donor organs.

*By invitation

F6. TRANSGENE EXPRESSION FOLLOWINGADENOVIRAL-MEDIATED RETRANSFECTION OF RAT LUNGS IS INCREASED AND PROLONGED BY TRANSPLANTATION-LEVEL IMMUNOSUPPRESSION.

Stephen D. Cassivi, M.D.*, Mingyao Liu, M.D.*, Annette Boehler, M.D.*, Keith Tanswell, M.B.*, Arthur Slutsky, B.A.Sc., M.A.Sc., M.D.* and Shaf H. Keshavjee, M.Sc., M.D.*

Toronto, Ontario, Canada

Sponsored by: Thomas R.J. Todd, M.D., Toronto, Ontario, Canada

The potential benefits of gene therapy, in modifying donor organs to better withstand the process of transplantation, have yet to be fully realized. *In vivo* gene transfer using adenoviral vectors has had limited success due to a host immune response inducing severe inflammation, which limits both the amount and duration of transgene expression, and obviates effective retransfection. Since this transgene expression diminishes over time, the ability to achieve effective retransfection is essential to providing successful chronic gene therapy. Our recent studies have shown that by administering transplant-level immunosuppression, transgene expression, following initial adenoviral-mediated transfection, can be significantly increased and prolonged for up to 5 weeks. In our current studies, we have shown that by administering transplant-level immunosuppression consisting of cyclosporine, azathioprine, and methylprednisolone, effective and prolonged adenoviral-mediated retransfection of rat lungs can also be achieved. Male Lewis rats, randomly assigned to immunosuppressed or non-immunosuppressed (control) groups, received intratracheal delivery of a first-generation, replication-deficient adenovirus containing the reporter gene Î²-galactosidase at day 0 and again at day 35. Whereas control rats had virtually no detectable transgene expression following retransfection (2.8 ± l.lRLU/ng protein, 0.4 ± 0.1 RLU/ng protein, and 1.2 ±0.2 RLU/ng protein, at days 1, 7 and 14 respectively), immunosuppressed rats demonstrated significantly elevated transgene expression, peaking at 7 days and showing prolonged and elevated expression at 2 weeks (188.4 ±46.4 RLU/ng protein, 294.2 ±43.7 RLU/ng protein, and 183.8 ± 53.6 RLU/ng protein, at days 1, 7 and 14 respectively; $p < 0.005$ at all three time points). To confirm these quantitative chemiluminescence results, we performed chromogenic staining of lung tissue using the X-gal stain for the marker gene Î²-galactosidase, which showed a diffuse pattern of transgene expression in the lung tissue of immunosuppressed rats. Conversely, virtually no positive staining was observed in the control group. Chronic gene therapy, through effective gene retransfection, previously limited by a severe immune response, can therefore be achieved with transplant-level immunosuppression. Improved outcomes in lung transplantation may now be possible, through enhanced and prolonged genetic modification and gene therapy retreatment.

*By invitation

F7. ALTERED MYOCARDIAL GENE EXPRESSION FOLLOWING BRAIN DEATH.

Thomas Yeh, Jr., M.D, Ph.D.*, Andrew S. Wechsler, M.D., Laura J. Graham, L.V.T.*, Kathryn E. Loesser, Ph.D.*, Domenic A. Sica, M.D.*, Luke Wolfe, Ph.D.* and Emma R. Jakoi, Ph.D.*

Toronto, Ontario, Canada and Richmond, Virginia

Objectives: The depressed myocardial function observed in brain dead organ donors has been attributed to massive sympathetic discharge and catecholamine cardiotoxicity. Although physiologic and morphologic changes associated with brain death have been reported, the molecular mechanisms underlying the observed hemodynamic instability are poorly understood. Because chronic catecholamine administration alters gene expression in myocardial cell culture, and because humans with elevated systemic catecholamines manifest decreased expression of myocardial genes important in contractility, we tested the hypothesis that activation of the neuromyocardial axis modulates the expression of genes important for contractility.

Methods: In a balloon expansion model of increased intracranial pressure (†‘ICP) in rabbits (n = 23), sympathetic regulation of LV myocardial gene expression was analyzed. At timed intervals after brain death, mean arterial pressure, heart rate, ECGs and histologic myocardial injury were compared with sham-operated controls. Systemic catecholamine levels were correlated with changes in hemodynamic parameters in response to †‘ICP. Using RNA blot hybridization analysis, the effect of †‘ICP on levels of mRNA encoding myofilaments, adrenergic receptors, sarcoplasmic reticulum proteins, and stress induced genes [i.e. immediate early genes and heat shock protein 70 (hsp70)] was measured.

Results: In contrast with sham operated controls, †‘ICP resulted in several statistically significant changes, including an immediate pressor response that correlated temporally with diffuse ECG ST segment changes and histologic injury. A concurrent 5-8 fold increase in systemic epinephrine and norepinephrine levels dropped precipitously below baseline to near zero levels within 2 hours. Four hours after †‘ICP, levels of mRNA encoding skeletal and cardiac Î±-actins, egr-1, and hsp70 were selectively increased over sham operated controls.

Conclusions: This study of †‘ICP is the first systematic evaluation of multiple programs of myocardial gene expression in an *in vivo* model of brain injury, mRNAs encoding egr-1, hsp70 and Î±-actins are all significantly increased after †‘ICP, a pattern which differs from those typically associated with depressed myocardial contractility and chronic elevation of systemic catecholamines. Myocytes in experimental brain deal undergo a molecular transformation that may have important implications when such hearts are donated for transplantation.

*By invitation

F8. FACTOR IXai IS A SELECTIVE ANTICOAGULANT AGENT FOR CARDIOPULMONARY BYPASS WHICH PROVIDES EXTRACORPOREAL CIRCUIT ANTICOAGULATION WITH PRESERVATION OF SURGICAL HEMOSTASIS.

Talia B. Spanier, M.D.*, †Mehmet C. Oz, M.D., David M. Stern, M.D.*, Niloo M. Edwards, M.D.*, Eric A. Rose, M.D. and Ann Marie Schmidt, M.D.*

New York, New York

INTRODUCTION: The mechanism of activation of coagulation in the setting of cardiopulmonary bypass (CPB) remains to be delineated. We believe that activation of coagulation in the intravascular space/extracorporeal circuit is due to activation of the intrinsic pathway, while activation in the extravascular space / surgical wound is separately due to the extrinsic / tissue factor (TF) mediated pathway. An ideal anticoagulant agent in CPB, therefore, would be one which would SELECTIVELY inhibit CPB circuit / contact-mediated thrombosis, while preserving extravascular / TF-mediated hemostasis. We focused on Factor IX due to its unique location in the intrinsic pathway of the coagulation cascade and hypothesized that blockade of Factor IX would achieve this end.

METHODS: Active-site blocked Factor IXa (IXai), a competitive inhibitor of the assembly of IXa in the X activation complex, was prepared by enzymatic modification of IXa with dansyl-glutamyl, glycyl arginyl chlormethylyketone. CPB was performed in 12 baboons (7 with IXai (460μg/kg)/no reversal, 5 with heparin/protamine (H)). After 1 hour at 32°C, baboons were weaned from CPB and observed for 3 hours postoperatively. Intraoperative blood and tissue samples were taken for analysis of thrombin generation, macrophage procoagulant activity and immunohistochemistry.

RESULTS: Normal circuit pressures were maintained throughout CPB in both groups with no clot formation in the tubing. Scanning EM of arterial filters confirmed no differences in fibrin / platelet deposition. Blood loss was significantly less with IXai vs H (480 ± 75 vs 1150 ± 115ml, $p<0.05$). A modified bleeding time, used to assess extravascular hemostasis, revealed that there was no evidence of enhanced extravascular bleeding during CPB or postoperatively compared to baseline in IXai treated animals. In contrast, significantly increased extravascular bleeding was observed after infusion of H during CPB, which returned to baseline after H reversal with protamine ($p<0.05$). In order to delineate the sites and extent of thrombin generation in CPB, examination of plasma prepared from peripheral blood from animals treated with IXai compared to H revealed significantly increased levels of thrombin antithrombin III complex [TAT] and prothrombin activation complex (F1+2), with peak levels occurring on CPB, and then declining immediately after termination. Furthermore, retrieval of monocytes from baboon peripheral blood, arterial filter and CPB pump (representing the intravascular/ extracorporeal space) during the procedure revealed no demonstrable TF activity in IXai or H treated animals. In contrast, monocytes isolated from pericardial blood (representing the extravascular space) in IXai treated animals manifested significantly enhanced procoagulant activity. Immunohistochemical examination of the pericardium during CPB confirmed enhanced TF activity in IXai but not H treated animals with striking TF expression in the pericardial mesothelial cells, in macrophages, as well as in the vasculature and macrophages that pervade pericardial tissue.

CONCLUSIONS: The finding that overall thrombin generation is greater in baboons treated with IXai supports our hypothesis that separate mechanisms are responsible for activation of coagulation in CPB since, while effective antithrombotic effect is realized in the extracorporeal circuit with IXai, extravascular hemostasis is preserved. Activation of coagulation in the setting of CPB, therefore, is separately attributable to the intrinsic pathway in the bypass circuit/ intravascular space, while the extrinsic /TF pathway is predominantly responsible for that within the surgical wound. Selective anticoagulation with IXai is a novel anticoagulant for CPB which affords a mechanism by which to study activation of coagulation in CPB, and also selectively prevents intravascular/extracorporeal circuit thrombosis with preservation of surgical hemostasis.

†1994-96 AATS Robert E. Gross Research Scholar

*By invitation

9:00 a.m. PLENARY SCIENTIFIC SESSION

Ballroom, Hynes Convention Center

Moderators: Floyd D. Loop, M.D.

14. EXCELLENT LONG TERM FUNCTIONAL OUTCOME AFTER SURGERY FOR ANOMALOUS LEFT CORONARY ARTERY FROM THE PULMONARY ARTERY.

Andrew D. Cochrane, F.R.C.S.*, David M. Colmenan, M.B.Ch.B.*, Christian P.R. Brizard, M.D.* and Tom R. Karl, M.D.

Melbourne, Australia

Discussant: Yasuharu Imai, M.D., Shinjuku-ku, Japan

During 1980-1996, 20 patients underwent surgery for this condition. In 19 patients the left main coronary artery arose from the MPA or RPA, and in one infant the circumflex alone came from the MPA. The median age at presentation was 9 months (range 6 weeks to 26 years). Fourteen patients were severely symptomatic with heart failure at time of operation, at a younger median age of 6 months. Mitral regurgitation was present in all except two patients, usually moderate to severe in degree; one other patient had undergone a mitral valve replacement before the diagnosis of ALCAPA was made. One infant had LV anterior wall rupture and a hemopericardium due to transmural infarction. Treatment involved Takeuchi operation (n=12), direct reimplantation (n = 6), aorto-coronary bypass in an older patient after previous ligation (n = 1) and ligation of the isolated anomalous circumflex artery (n = 1). There were no operative deaths (0%, CL = 0-17%) and no late deaths. Although 5 patients required LVAD support, their outcome has been excellent with no increment in early mortality or late sequelae ($p>0.05$). There have been two late reoperations, one for supravalvar PA stenosis after Takeuchi repair, and one mitral valve replacement. Nine patients followed up at this hospital have undergone late studies, including three who required LVAD and one who had a late mitral valve replacement. On nuclear gated scan at a mean of 6 years from surgery the LVEF was 64 ± 3 % at rest and increased normally to 74 ± 3 % on exercise. First pass RVEF was normal at 65 ± 3 %. On echocardiography 6 patients have mild or no mitral regurgitation, while 3 patients have persistent moderate or severe regurgitation with one infant requiring medical therapy. The LV dimensions, fractional shortening and indices of systolic and diastolic function are no different between the patients and healthy age-matched controls ($p>0.05$). In the 7 children old enough to perform a treadmill exercise test, the exercise time and blood pressure response were normal. Long-term outlook and late LV function is good, even in infants with severely impaired LV function at presentation or in those whorequire LVAD support. Cardiac transplantation should rarely be necessary in this group, even in patients with initial severe impairment of LV function.
*By invitation

15. EFFECT OF DONOR AGE AND DONOR ISCHEMIC TIME ON INTERMEDIATE-TERM SURVIVAL AND SECONDARY ENDPOINTS AFTER LUNG TRANSPLANTATION.

Dan M. Meyer, M.D.*, Leah H. Bennett, Ph.D.*, Richard J. Novick, M.D. and

Jeffrey D. Hosenpud, M.D.*

Dallas, Texas; Richmond, Virginia; London, Ontario, Canada and Milwaukee, Wisconsin

Discussant: G. Alexander Patterson, M.D., St. Louis, Missouri

Objective: Pressure to expand the donor pool has required the use of lungs from older donors or from more distant procurement areas. The long-term consequences of this policy have not yet been

fully addressed. The effect of donor age and donor ischemic time on intermediate survival and important secondary endpoints after lung transplantation was therefore determined. *Methods:* A cohort of 1450 lung transplant recipients with complete 2 year followup, operated upon in the United States between April 1, 1993 and March 31, 1996, were studied. *Results:* As shown below, donor age when analyzed independently did not affect intermediate survival or the secondary endpoints ($p>0.05$ for all variables).

Outcome	Time point	Donor Age < 45 years (N=1258)	Donor Age 45-55 years (N=169)	Donor Age > 55 years (N = 23)
Patient Survival (%)	1 year	87.4	88.1	78.3
	2 years	77.8	82.4	70.4
Hospitalized for	1 year	26.6	15.8	21.9
Rejection (%)	2 years	21.8	12.1	20.4
Bronchiolitis	1 year	11.4	10.5	9.2
Obliterans (%)	2 years	26.6	15.5	20.4
FEV_1 (%)	1 year	63.5	59.2	55.3
	2 years	62.6	58.9	59.3
Bronchial	1 year	7.9	12.1	9.1
Stricture (%)	2 years	7.3	3.6	20.4
Hospitalized for	1 year	37.4	44.2	52.3
Infection (%)	2 years	31.8	26.4	51.0

Similarly, when the interaction between *ischemic time* and donor age was examined in all of the multivariate models, none of the secondary endpoints was found to be significantly influenced. However, the combined interaction between donor age and ischemia time demonstrated a significantly worse survival at two years ($p = 0.02$) at donor age of > 50 years and donor ischemic time > 7 hours. *Conclusions:* Donor age and donor ischemic time did not negatively influence survival or important secondary endpoints after lung transplantation. However, intermediate-term survival was affected by the use of older donors when combined with a prolonged ischemic time. The impact of this combination should be considered when attempting to expand the donor pool.

*By invitation

16. VOLUME-OUTCOMES STUDIES OF CARDIOVASCULAR PROCEDURES IN NEW YORK STATE (1990-1995).

Keith Reemtsma, M.D., Eileen P. Shields, B.A.*, Jonathan M. Chen, M.D.*, Patrick J. Roonan, M.S.* and Annetine C. Gelijns, Ph.D.*

New York and Albany, New York

Discussant: Jack M. Matloff, M.D., Los Angeles, California

Background: Although conventional wisdom holds that increased experience yields better outcomes, studies have shown varied patterns in the relationship between volume of surgical cases

and outcome. We have examined the relationship of hospital volume on in-hospital mortality in three patient cohorts: (1) adults undergoing coronary artery bypass graft (CABG) surgery without additional procedures (a high volume, State regulated procedure) (2) elective non-ruptured abdominal aortic aneurysm (AAA) repair (a low volume, unregulated procedure), and (3) pediatric patients undergoing cardiac surgery involving cardiopulmonary bypass (a low volume, regulated procedure). Methods: All cardiovascular procedures performed in New York State from 1990-1995 were evaluated in this study. Logistic regression Ï‡²analysis (with resultant odds ratios) was performed evaluating hospital case volume with in-hospital mortality. Patients in the pediatric cohort were subdivided by age into three strata for analysis: <30 days (neonates), 31 days-1 year, 1-12 years of age, 13 -20 years of age.

Results: 109,372 patients underwent CABG surgery, 9,981 patients underwent AAA repair, and 6,618 pediatric patients underwent open-heart procedures. No significant association ($p>0.05$) was demonstrated between hospital case volume and mortality for CABG patients. However, a significant inverse relationship was noted ($p = 0.0001$) for patients undergoing elective AAA surgery, and children less than one year of age undergoing cardiac surgery. Additionally, for neonates, hospitals with case volumes below 100 patients displayed mortality rates three-fold higher than in the highest volume hospital ($p = 0.0001$). Patients 1-20 years of age did not show a significant inverse relationship by logistic regression analysis ($p>0.05$).

In New York State, cardiac surgery is regulated by a certificate-of-need process to approximately 30 hospitals state-wide, and the median CABG case volume for 1990-1995 was 2501 operations. By contrast, surgery for abdominal aortic aneurysms was performed in 195 hospitals, and in 138 hospitals fewer than 50 aneurysm operations were done over the six year period. Only three hospitals had operative volumes exceeding 300 operations in the same time period (1990-1995).

Conclusions: Although no relationship was demonstrable between hospital volume and mortality for patients undergoing CABG surgery, a strong correlation was noted for adults undergoing elective AAA repair and children undergoing open-heart procedures, most notably neonates. Neither AAA nor pediatric cardiac procedures were concentrated in hospitals with high volumes. These data support the concept of concentrating high risk procedures (especially those of low volume) in a limited number of hospitals that can provide an acceptable annual volume of activity.

10:00 a.m. INTERMISSION - VISIT EXHIBITS

*By invitation

10:45 a.m. PLENARY SCIENTIFIC SESSION

Ballroom, Hynes Convention Center

Moderators: Floyd D. Loop, M.D.

James L. Cox, M.D.

17. CRYOPRESERVED ARTERIAL HOMOGRAFTS IN THE TREATMENT OF MAJOR VASCULAR INFECTION: A COMPARISON WITH CONVENTIONAL SURGICAL TECHNIQUES.

Paul Robert Vogt, M.D.*, Hans-Peter Brunner-LaRocca, M.D.*, Thierry Carrel, M.D.*, Ludwig K. von Segesser, M.D.*, Christian Ruef, M.D.*, Wolfgang Kiowski, M.D.* and Marko Turina, M.D.

Zurich, Switzerland

Discussant: Tirone E. David, M.D., Toronto, Ontario, Canada

Objectives: The results with cryopreserved heart valve homografts in the treatment of infectious endocarditis suggest that the use of cryopreserved arterial homografts may improve the outcome in patients with major vascular infections.

Methods: Between 1990 and 1997, 72 patients with mycotic aneurysm (n = 29) or infected vascular prostheses (n = 43) of the thoracic (n = 26) or abdominal aorta (n = 46) were treated either with conventional surgery (n = 38) or implantation of a cryopreserved arterial homograft (n = 34). Survival, survival free of reoperation, lengths of intensive care, hospitalization, antibiotic treatment and costs were assessed. In patients with homografts, computed tomography, magnetic resonance imaging-angiography, or intravenous digital subtraction angiography were performed after a mean follow-up of 27 ± 16 months.

Results:	Homografts (n=34)	Prosthesis (n=38)	p- value
30-day-mortality	5.9%	18.4%	0.16
Total mortality	11.8%	31.8%	0.07
Infection eliminated	91%	53%	0.001
Reoperation during first postoperative year	9%	34%	0.01
Reoperation during follow-up time	9%	45%	0.001
Mean (± SD) time on respirator postoperative (days)	3.5(6.1)	12.8(15.2)	0.001
Mean (± SD) time on intensive care (days)	5.6(9.7)	23.5 (29.5)	0.001
Mean (± SD) duration of hospitalization (days)	44.5 (28.6)	93.4(58.3)	0.001
Mean (± SD) duration of antibiotic therapy (days)	40.4(12.2)	159.7(138.3)	0.001
Cost of treatment per case	88,539 US$	310,979 US$	0.001

After 4 years, freedom from reoperation and death was 75 ± 10% for homografts and 34 ± 9% for prosthesis (p = 0.0018). Neither false aneurysm formation, stenosis, leakage, nor dilatation of homografts were observed.

Conclusions: The use of cryopreserved arterial homografts provides a safer, cheaper and more effective treatment for mycotic aneurysms and infected vascular prostheses than conventional surgical techniques.

*By invitation

18. NEUROPSYCHOLOGICAL OUTCOME FOLLOWING DEEP HYPOTHERMIC CIRCULATORY ARREST.

David L. Reich, M.D.*, M. Arisan Ergin, M.D., Suzan Uysai, Ph.D.*, Martin Sliwinski, Ph.D.*, JockN. McCullough, M.D.*, Jan D. Galla, M.D.*, Wayne Gordon, Ph.D.*, Mary Hibbard, Ph.D.* and Randall B. Griepp, M.D.*

New York, New York

Discussant: Richard A. Jonas, M.D., Boston, Massachusetts

Introduction: There is compelling evidence in the pediatric populations that prolonged periods of deep hypothermic circulatory arrest (DHCA) are associated with long term deficits in cognitive and some intellectual functions. Although DHCA is widely used in surgery of the thoracic aorta there is a paucity of such information in adult patients. Evaluation of neurological outcome traditionally has been limited to reports of the incidence of postoperative stroke. The incidence of and factors associated with long term neuropsychological dysfunction are unknown.

Methods: Under an IRB-approved protocol 122 patients undergoing elective cardiac or thoracic aortic surgery were evaluated preoperatively, 1 week, and 6 weeks postoperatively with a battery of neuropsychological tests. Seventy-two patients had routine cardiac surgery without DHCA (Group No DHCA), 50 patients had thoracic aortic surgery with varying periods of DHCA and were subdivided into those with 1-24 rain of DHCA (n = 26), and those with > 25 rain of DHCA (n = 24). The neuropsychological test battery consisted of 8 tests consolidated into 5 domains: attention, cognitive speed, memory, executive function, and fine motor function. Data were normalized to baseline values, and were analyzed using ANOVA, ANCOVA, and survival functions.

Results: Age was a significant predictor of impairment in memory, fine motor and executive function, therefore standardized scores were age-adjusted. Patients who could not be tested or had poor testing performance at 1 week postoperatively were more likely to perform poorly at 6-weeks (odds ratio 5.27, $p<0.01$). DHCA > 25 rain and to a lesser degree increasing age were significant predictors of impaired memory and motor function at 6 weeks postoperatively (Table 1), but not of attention, cognitive speed, or executive function. The decline (-0.48 ± 0.27) in memory function at 6 weeks (Table 2) roughly approximates a 20 point decrease in IQ. DHCA > 25 min (Odds ratio 4.0; $p = 0.02$) and increasing age (Odds ratio/5 years 1.23; $p = 0.06$) were determinants of prolonged hospital stay(‰¥21 days) (Table 1)

Conclusion: Prolonged DCHA (‰¥25 minutes) especially in older patients is associated with a subtle but important neuropsychological deficit in the domains of memory and fine motor function and also with prolonged hospital stay. The previously reported high incidence of temporary neurological dysfunction may be a clinical marker of this insidious neurological injury.

Table 1: Predictors of neuropsychological outcome (at 6 weeks) and hospital stay

	MEMORY	MOTOR	Hospital Stay (>21 days)
	Odds Ratio p	Odds Ratio p	Odds Ratio p
DHCA >25 min	3.52 0.04	7.25 0.004	4.01 0.02
AGE (/5 year incr.)	1.14 0.16	1.21 0.08	1.23 0.06

Table 2: Mean memory performance and change from baseline (age-adjusted Standard scores ± SEM)

	Baseline	6 Weeks Postop	Change

No DHCA	0.04 ±0.1 6	0.42 ±0.1 8	0.38
1-24 min DHCA	0.01 ±0.20	0.49 ± 0.24	0.48
> 25 min DHCA	-0.04 ± 0.24	-0.48 ±0.27*	-0.44*

* $p < 0.05$ compared with no DHCA at baseline, 6 weeks post-op, and change from baseline

11:25 a.m. C. WALTON LILLEHEI RESIDENT FORUM AWARD PRESENTATION
11:30 a.m. ADDRESS BY HONORED SPEAKER

A Practical Affair

Professor Ken Taylor, M.D., FRCS, London, England

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

12:10 p.m. CARDIOTHORACIC RESIDENT'S LUNCHEON

*By invitation

TUESDAY AFTERNOON, MAY 5, 1998

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A CONGENITAL HEART DISEASE
Ballroom A, Hynes Convention Center

Moderators: Frank L. Hanley, M.D.

Thomas L. Spray, M.D.

19. MULTIPLE VENTRICULAR SEPTAL DEFECTS: HOW AND WHEN SHOULD WE REPAIR?

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

San Francisco, California

Discussant: Constantine Mavroudis, M.D., Chicago, Illinois

Congenital heart lesions with multiple ventricular septal defects (VSDs) remain a surgical challenge. Difficult exposure of VSDs in the trabecular component of the septum, especially the apical and anterior portions, complicates closure of such defects. Traditional approaches often rely on either ventriculotomy for exposure or palliation with pulmonary artery banding (PAB). Both of these techniques have potential drawbacks, so it is important to limit their use to pts in whom they will provide optimal benefit. However, indications for closure vs palliation and for various approaches to surgical exposure are not clearly defined. Since 1992, 32 pts with multiple VSDs have undergone surgery for this condition. Median age was 86 days and all but 4 pts were infants. Associated lesions were present in 27 pts, including transposition of the great arteries (9), aortic coarctation (4), atrioventricular septal defect (3), interrupted arch (2), tetralogy of Fallot (1), patent ductus arteriosus (3), atrial septal defect (3), and other defects (3). Prior operations had been performed elsewhere in 8 pts, including PAB in 6. Each pt had ‰¥ 2 VSDs (median 3). VSDs were perimembranous in 13 pts, inlet in 6, outlet in 2, anterior muscular in 13, posterior muscular in 10, apical in 21, and mid-muscular in 15. "Swiss cheese" VSDs were present in 6 pts, 5 of whom had

prior PAB. Initially, 25 pts underwent complete repair, all through right atriotomy except 1 who also required aortotomy. Seven pts were palliated with PAB (5) or other procedures. No pt received a ventriculotomy. In the pts who underwent complete repair, a combination of patch and suture closure was used in 16, patch only in 6, and sutures only in 3. In pts with muscular VSDs, the left side of the septum was explored through an aortotomy or a larger VSD when possible, which aids in visualization and closure of defects in the trabecular septum. There was 1 early death in a pt who had staged repair of coarctation and multiple VSDs. 3 palliated pts underwent early reoperation for PAB revision due to failure to thrive (1), PAB revision and then VSD closure for failure to thrive (1), or PAB removal after spontaneous closure of the VSDs led to decompensation (1). Thus, 3 of 7 palliated pts required early reoperation. Pacemakers were placed for atrioventricular dissociation in 2 pts, but sinus rhythm returned in both. At median followup of 22 mos (4.49 mos), there was 1 death in a palliated pt and 1 other palliated pt had a heart transplant for cardiomyopathy. Another palliated pt had the PAB removed and multiple apical VSDs closed 8 mos later after outgrowing the band. No pts have hemodynamically significant residual VSDs or persistent rhythm abnormalities. In our experience, palliation of multiple VSDs is associated with greater morbidity than primary repair. Almost all cases of multiple VSDs can be repaired adequately in early infancy without ventriculotomy, though "swiss cheese" VSDs are still an indication for palliation.
*By invitation

20. TRANSATRIAL-TRANSPULMONARY TETRALOGY REPAIR IS EFFECTIVE IN THE PRESENCE OF ANOMALOUS CORONARIES.

Christian P.R. Brizard, M.D.*, Andrew D. Cochrane, F.R.A.C.S.*and Tom R. Karl, M.D.

Melbourne, Australia

Discussant: John W. Brown, M.D., Indianapolis, Indiana

Objectives: To analyze the outcome of transatrial-transpulmonary tetralogy repair in children with an anomalous coronary crossing the RV outflow tract. Methods: 34/548 (6.2%) of our tetralogy repairs were associated with surgically relevant coronary artery anomalies. The median age and weight at repair were 25.8 months (4.4-88) and 10 kg (5-20). All patients had a major branch crossing the right ventricular outflow tract. Anomalies included LAD from RCA (n = 19), RCA from LCA or LAD (n = 10), large RCA conus branch (n = 4), and single RCA (n = 1). Diagnosis was established preoperatively in 24/34 with angiography (n = 23) or echo (n=1). Transatrial-transpulmonary repair was successfully used in 31 patients, and 24 of whom required a limited transannular patch in the RVOT. Three additional patients had a RV-PA conduit due to proximity of the coronary branch to the PA annulus, and inability to relieve the right ventricular outflow obstruction adequately.

Results: There has been no early or late mortality. Mean RV-PA gradient at last follow-up was 18mm (SD=10.6), compared to 15mm (SD = 24) for patients with normal coronaries operated by a similar approach. Actuarial freedom from reoperation at 105 months was 95% (± 4.9%), which was also similar to results of transatrial-transpulmonary repair in children with TOP and normal coronaries ($p > 1.0$).

Conclusions: Surgically important coronary anomalies in TOP can be dealt with using the transatrial-transpulmonary approach in most cases, without major alterations in technique. Outcome is similar to that for other TOP patients. Thus, the presence of anomalous coronary has not imparted incremental risk following this surgical strategy.

*By invitation

21. SINGLE STAGE REPAIR OF AORTIC ARCH OBSTRUCTION AND ASSOCIATED INTRACARDIAC DEFECTS USING PULMONARY HOMOGRAFT PATCH AORTOPLASTY.

Christo I. Tchervenkov, M.D.*, Stephen A. Tahta, M.D.*, Marie J. Beland* and Luc C. Jutras*

Montreal, Quebec, Canada

Sponsored by: Ray C.-J. Chiu, M.D., Ph.D. Montreal, Quebec, Canada

Discussant: Claude Planche, M.D., Paris, France

Intracardiac malformations associated with coarctation and aortic arch hypoplasia have traditionally been repaired in two stages, with a high mortality. Recently, improved survival has been reported with a single-stage approach predominantly using an extended end-to-end anastomosis to repair the aortic arch. Herein, we review our experience with a single-stage biventricular repair of intracardiac defects associated with aortic arch hypoplasia using a pulmonary homograft patch aortoplasty. Between October 1988 and October 1997, 39 of 40 consecutive patients underwent a single-stage biventricular repair for aortic arch obstruction and associated intracardiac defects. The median age at operation was 17 days and the mean weight was 3.71 ± 1.09 kg. Nineteen patients had either d-transposition of the great arteries or the Taussig-Bing anomaly. Sixteen patients had multiple left-sided obstructive lesions (2 critical aortic stenosis, 3 subaortic stenosis and VSD, 11 class III hypoplastic left heart syndrome as defined by Kirklin). One patient had an associated complete atrioventricular septal defect. Four patients had only an associated ventricular septal defect. By median sternotomy, the hypoplastic aortic arch was enlarged with a pulmonary homograft patch in 36 patients. In 4 patients, an extended end-to-end anastomosis was performed.

There were 2 early deaths (5%) and 21 late deaths (5%). One late death was non-cardiac related. The median followup time was 25 months (range 1 month to 8 years). The recoarctation rate was 11% but excluding those patients with associated left-sided obstructive lesions, this decreases to 0%. No aneurysm formation in the aorta has occurred. The actuarial survival rate at 8 years is 89 ± 10%.

One-stage biventricular repair of aortic arch obstruction and ‘associated intracardiac defects can achieve excellent survival. We recommend the pulmonary homograft patch aortoplasty of the hypoplastic arch because it achieves complete relief of anatomical afterload with a tension-free anastomosis and low incidence of recoarctation.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

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

Ballroom A, Hynes Convention Center

Moderators: Frank L. Hanley, M.D.

Thomas L. Spray, M.D.

22. PRIMARY REPAIR IS SUPERIOR TO INITIAL PALLIATION IN CHILDREN WITH ATRIOVENTRICULAR SEPTAL DEFECT AND TETRALOGY OF FALLOT.

Hani K. Najm, M.D.*, Glen S. Van Arsdell, M.D.*, Stefan Watzka*, Lisa Hornberger, M.D.*, John G. Coles, M.D.* and William G. Williams, M.D.

Toronto, Ontario, Canada

Discussant: Hillel Laks, M.D., Los Angeles, California

Children presenting with atrioventricular septal defect in conjunction with tetralogy of Fallot (AVSD/TOF) represent an uncommon complex lesion. To explore the best management algorithm we undertook a review of this institution's experience. From March 1981 to August 1997, 38 children were referred to our division with the diagnosis of AVSD/TOF. Down's syndrome was present in 32 (84%). Twenty one children were initially palliated with a systemic to pulmonary artery shunt. Two (9.5%) died prior to repair. Complete repair has been performed on 31 children. Of these 14 underwent initial palliation with systemic to pulmonary artery shunt (mean age at shunt 20 ± 24 months). During complete repair the endocardial cushion defect was repaired with two patches in 29 children. Relief of the right ventricular outflow obstruction was by a transannular patch in 22 (70%), 12 (54%) of those had a monocusp inserted, and 4 required an infundibular patch. Operative mortality occurred in 2 (6.4%) children, one was previously palliated. Reoperations were performed in 11 children, 7 (58%) were for pulmonary arterioplasty. The incidence of reoperation was higher in the palliated versus the non palliated groups (64% vs. 12%, P= 0.007). The palliated group were repaired at an older age (78 ± 52 vs. 36 +19 months, P= 0.005), had longer ventilatory support (8 ± 11 rs. 4 ± 3 days, P= 0.05), longer inotropic support (8 ± 13 vs. 3.6 ± 1.5 days, P= 0.028), and longer hospital stay (23 ± 22 vs. 14 ± 5 days, P= 0.004). There was one late death related to a reoperation for pulmonary arterioplasty and residual ventricular septal defect in the palliated group. We conclude that repair of AVSD/TOF can be achieved with low mortality. In this series initial palliation with a shunt resulted in a more complex post-operative course and carried a higher reoperative rate, mainly due to pulmonary artery complications. Primary repair of AVSD/TOF at the time of symptomatic presentation is superior to initial palliation followed by repair.
*By invitation

23. THE MODIFIED NORWOOD PROCEDURE FOR HYPOPLASTIC LEFT HEART SYNDROME: EARLY TO INTERMEDIATE RESULTS OF 120 PATIENTS.

William J. Brawn, F.R.C.S.*Kozo Ishino, M.D.*, Oliver Stumper, M.D.*, Joseph V. De Giovanni, F.R.C.P.*, Eric D. Silove, F.R.C.P.*, John G.C. Wright, F.R.C.P.* and Babulal Sethia, F.R.C.S.*

Birmingham, England

Sponsored by: †Marc de Leval, M.D., London, England

Discussant: Roger B.B. Mee, M.B., Ch.B., Cleveland, Ohio

Background: Classical first-stage Norwood repair of hypoplastic left heart syndrome (HLHS) utilizes homograft patch enlargement to obtain an unobstructed aorta and coronary arteries. Because of possible disadvantages of the homograft such as lack of growth, degeneration and calcification, and homograft availability, we have tried to repair the aorta without patch supplementation.

Method and Results: Between February 1993 and September 1997, 120 patients, aged 0 to 47 days (median, 4 days) and weighing 1.7 to 4.4 kg (median, 3.1 kg) underwent first-stage palliation for HLHS. The ascending aortic diameter ranged from 1.5 to 8 mm (median, 3.0 mm). Eight patients had an anomalous right subclavian artery (ARSCA) arising from the descending aorta In 95 patients (Group I), all ductal tissue was excised, the descending aorta anastomosed to the aortic arch which had been opened back to the ascending aorta, then to this confluence was anastomosed the proximal main pulmonary artery. In the remaining 25 patients (Group II), continuity of the aortic arch was maintained and the repair was performed with a Damus anastomosis. The size of the systemic-to-pulmonary shunt was 3.5 mm (70), 3 nun (48), and 4 mm (2). Circulatory arrest time ranged from 19 to 105 Min (median, 54 Min). A homograft patch was necessary for the arch reconstruction in a total of 18 patients (15.0%); 9 Group I patients (9.5%) and 9 Group II (36.0%) ($p< 0.01$). There were 82 hospital survivors (68.3%); 69 Group I (72.6%) and 13 Group II (52.0%) ($p<0.05$); 71 patients without patch (69.6%) and 11 with patch (61.1%) (p = NS). By multiple logistic regression, risk factor for hospital death included ascending aortic diameter ‰¤ 2 MM ($p = 0.1018$) and the ARSCA ($p = 0.039$). There were 6 late deaths. Sixteen of 71 patients (22.5%) who underwent second-stage palliation had developed neo-aortic arch obstruction with a peak gradient > 10 mmHg; 14 Group I (19.4%) and 2 Group II (22.2%) (p = NS); 15 without patch (23.1%) and one with patch (16.7%) (p = NS). Overall survivals were 56.7% at 1 year and 55.0% at 2 years.

Conclusion: The modified Norwood procedure without the use of exogeneous material does not result in an increased incidence of neo-aortic arch obstruction and may allow better long-term growth of the neo-aorta.

†1973-74 AATS Graham Fellow

*By invitation

24. POST-ISCHEMIC HYPERTHERMIA EXACERBATES NEUROLOGICAL INJURY FOLLOWING DEEP HYPOTHERMIC CIRCULATORY ARREST.

Dominique Shum-Tim, M.D.*, Mitsugi Nagashima, M.D.*, Toshiharu Shin'oka, M.D.*, Jan Bucerius, M.D.*, Georg Nollert, M.D.*, Hart G.W. Lidov, M.D.*, Adre du Plessis, M.D.*, Peter C. Laussen, M.D.* and Richard A. Jonas, M.D.*

Boston, Massachusetts

Discussant: Julie A. Swain, M.D., Lexington, Kentucky

Background: Aggressive surface warming is common practice in the pediatric intensive care unit; also, fever is commonly seen after cardiopulmonary bypass (CPB). Recent rodent data emphasizes the protective effect of mild (2-3°C) hypothermia after cerebral ischemia. The purpose of this study was to evaluate different temperature regulation strategies after deep hypothermic circulatory arrest (DHCA) using a survival piglet model. **Methods:** Fifteen piglets (9.1 ± 1.1 kg) were randomized into 3 groups (n = 5/group) and a small temperature probe was inserted in the superficial temporal cortex. All groups underwent 100 min. of DHCA at 15°C and 40 min. of reperfusion with a Hct of 25% during CPB. Brain temperature was maintained at 34°C for 24 hr after coming off CPB in Gp I, 37°C in Gp II, and 39°C in Gp III, respectively. The cerebral redox state was determined by near infrared spectroscopy (NIRS) during and 3 hr after CPB. Neurobehavioral recovery was evaluated daily for 3 days after extubation in a blinded fashion by neurological deficit score (NDS; 0 = normal, 500 = brain death) and overall performance capacities (OPC; 1 = normal, 5 = brain death). Body weight gain and total body water content indicated by electrobioimpedance were evaluated

at 3 hr, 24 hr, and 4 days post-operatively. Brain was fixed in situ on day 4 and the hemisphere contralateral to the probe was examined and scored for ischemic injury (0 = normal, 5 = necrosis) in a blinded fashion. **Results:** All results are expressed as mean or percentage ± SD. P-value <0.05 is considered significant. One animal in Gp III died on day 3 due to severe neurological impairment. There were no differences in % body weight gain and bioimpedance after CPB. At 24 hr, Gp III animals had significant changes in weight line break (Gp 1= 109.3± 1.7%, Gp II = 113.6 ±6.3%, and Gp III = 117.9 ± 3.4%; p<0.05 Gp I vs III) and bioimpedance (Gp I = 83.9 ± 12.5%, Gp II = 74.0 ± 9.6%, and Gp III = 62.9 ± 10.8%; p<0.05 Gp I vs III) suggesting total body edema. There were no significant differences in NIRS signals throughout CPB. Recoveries of NDS, OPC, histology and NIRS were significantly worse in hyperthermic Gp III. There was a trend towards reduced injury in Gp I (see table).

Table	NDS	OPC	Histology (POD 4)			Oxy-Hb	Deoxy-Hb	Cyt. a,«3
			cortex	hippocampus	caudate	2hr off CPB	2hr off CPB	2hr off CPB
GpI	30.0±28.3	1.0±0.0	0.0 ± 0.0	0.0 ±0.0	0.8 ± 1.0	22.7±12.5	-28.7±9.0	3.9±0.6
GpII	87.0±59.4	1.4±06	1.0±1.2	0.0 ± 0.0§	0.8 ± 1.0	9.9±8.4	-10.1±20.6	05±2.1§
GplII	186.0±131*	2.8±1.3#	2.8 ± 1 8#	2.4 ± 0.9#	2.2 ±1 8	-13.5±12.1*	25.3±16.4*	-3.9±2.3*

p < 0.05 Gp I vs III by ANOVA and Bonferroni, #p< 0.05 Gp I vs III by Mann-Whitney u tests

§ p < 0.05 Gp II vs III

Conclusions: Mild post-ischemic hyperthermia significantly exacerbates functional and structural neurological injury after DHCA. Fever and active warming above 37°C should be avoided after DHCA. A mild degree of hypothermia may reduce cerebral injury after prolonged DHCA.

*By invitation

25. IS IT NECESSARY ROUTINELY TO FENESTRATE AN EXTRACARDIAC FONTAN?

LeNardo D. Thompson, M.D.*, Edwin Petrossian, M.D.*, Doff B. McElhinney, M.S.*, Natalia A. Abrikosova, M.S.*, Andrew J. Parry, M.D.*, V. Mohan Reddy, M.D.* and Frank L. Hanley, M.D.

San Francisco, California

Discussant: John E. Mayer, M.D., Boston, Massachusetts

Fenestration (FEN) of a Fontan connection has been proposed as a means of improving outcomes of single ventricle palliation by allowing partial decompression of the Fontan pathway. The benefit of FEN is likely to be greatest in the early postoperative period, when pts may experience increased pulmonary vascular resistance and decreased systolic and diastolic cardiac function due to the effects of cardiopulmonary bypass (CPB), aortic cross-clamping (ACC), and positive pressure ventilation. However, there are potential drawbacks to FEN, including arterial desaturation, complications of a right-left shunt, and the need for another procedure to close the communication, suggesting that routine use may not be in the best interest of all pts. The utility of FEN with the extracardiac conduit Fontan operation has not been determined. Since 1992, 60 pts have undergone a modification of the Fontan procedure in which an extracardiac inferior cavopulmonary conduit is used in combination with a previously staged bidirectional Glenn anastomosis. FEN was performed selectively in 32 pts (54%). Among the last 25 pts, FEN was used in only 6 (24%). In 7 pts, a FEN was placed or clipped in the early postoperative period without CPB. CPB time did not differ between the 2 groups (108 vs 122 rain), but significantly fewer non-fenestrated than fenestrated pts

had ACC (3 vs 8; $p<0.05$). There were no operative deaths. Prolonged (>2 wks) pleural drainage occurred in 12 pts, 8 with FEN and 4 without. Fontan and common atrial pressures 1 day after surgery did not differ between non-fenestrated and fenestrated pts (13.1 vs 12.6 mmHg and 4.8 vs 5.8 mmHg, respectively). At followup ranging to 5 yrs, there were 2 late deaths and no pts developed protein losing enteropathy. These data suggest that there is no difference in outcome between pts who undergo the extracardiac conduit Fontan procedure with and without FEN. The extracardiac Fortran operation has a number of advantages that minimize the need for and utility of FEN, including avoidance of ACC, shorter duration of CPB, and a more streamlined hydrodynamic connection. These factors help optimize ventricular and pulmonary vascular function in the early postoperative period, which contributes to improved hemodynamics. Moreover, when necessary, a FEN can be placed or revised without return to CPB, which allows for more accurate and practical assessment of the need for FEN after the Fontan connection has been completed. We conclude that FEN is of no benefit in the majority of Fontan pts when an extracardiac conduit technique as described is used, and therefore FEN should not be performed routinely.

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

Ballroom B, Hynes Convention Center

6:30 p.m. MEMBER RECEPTION

Museum of Science

*By invitation

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

Ballroom B, Hynes Convention Center

Moderators: Bruce W. Lytle, M.D.

Marko I. Turina, M.D.

26. DISENTANGLING PENETRATING ULCER AND INTRAMURAL HEMATOMA OF THE THORACIC AORTA FROM CLASSIC TYPE A AND B DISSECTIONS.

Michael A. Coady, M.D.*, John A. Rizzo, Ph.D.*, Graeme L. Hammond, M.D., Lee J. Goldstein, B.A.*, Gary S. Kopf, M.D. and John A. Elefteriades, M.D.

New Haven, Connecticut

Discussant: Nicholas T. Kouchoukos, M.D., St. Louis, Missouri

Classic type A and B aortic dissections have been well described, however, less is known about the natural history of penetrating atherosclerotic ulcers (PU) and intramural hematomas (IMH) of the thoracic aorta. PU are atherosclerotic plaques which disrupt the internal elastic lamina, burrow deeply through the intima into the aortic media, and cause a localized intramedial dissection. The diagnosis is made on CT scan by demonstration of a contrast-filled outpouching in the aorta in the absence of a dissection flap or a false lumen. Patients with IMH have a hematoma in the media but have no evidence of an atheromatous ulcer on imaging studies, and an intimal tear is not visualized. The objective of this study is to determine the natural history of PU and IMH and assess appropriate therapeutic correlates of the natural history. Data on 198 patients with aortic dissection (86 type A and 112 type B) treated at our institution from 1985 to 1997 were analyzed. The aortic dissection database includes 431 imaging studies (MRI, CT, and ECHO). Of the 198 patients, 27 individuals (13.6%) were found to have either a PU or IMH by CT/MRI imaging studies, intraoperative

findings, or pathology reports. For PU and IMH, 5 cases (18.5%) were located in the ascending aorta, and the remaining 22 (81.5%) were in the descending aorta.
A chart review was conducted in all patients with type A and type B dissections, PU, and IMH to evaluate rates of rupture and further define the natural history. The follow-up period ranged from 2 to 107 months, with a mean of 26.2 months. Sixteen of the 27 patients with PU or IMH (59.3%) had malignant courses (7 patients (25.9%) went on to require aortic replacement and another 9 (33.3%) died of aortic rupture prior to surgery). Results of univariate analyses are summarized in the table and figure below. Patients with PU and IMH, in comparison to type A or B thoracic aortic dissections, affect an older age population, present with larger aortic diameters, occur primarily in the descending aorta, are associated more often with a prior diagnosed or treated AAA. The risk of aortic rupture is higher in patients with PU and IMH (9/27, 33.3%) than in patients with type A (6/86, 6.97%) ($p<0.0003$) or type B (4/1 12, 3.57%) aortic dissection ($p<0.0001$). One year survival is less for patients with penetrating ulcers (71%), as opposed to patients with type A (90%) or B dissections (90%) ($p<0.05$).

	Penetrating Ulcer/ Intramural Hematoma	*Type Dissection*	*Type B Dissection*
Patients	27 (15m, 12 f)	86 (61m, 25 f)	1 12 (70 m, 42 f)
Age	73.9 years ± 8.2†*	52.7 years ±17.6†	67.1 years ± 13.3*
Initial Aortic Size	6.45 cm ± 1.9	5.63 cm ± 1.5	5.4 cm ± 1.6
Prior AAA	8 (29.6%)	8 (9.3%)**	35 (31.2%)**

Table reports means ± standard deviation, *p = 0.01, †p < 0.0001, **p = 0.001

We conclude that accurate recognition at initial presentation is critical for optimal clinical management of these patients. PU and IMH presenting acutely must be differentiated from classic acute dissections. For PU and IMH, the prognosis is more serious than with classic acute aortic dissection. Surgical treatment should be considered strongly at presentation.
*By invitation

27. RUPTURE OF CHRONIC TYPE B DISSECTING ANEURYSMS: A NATURAL HISTORY STUDY.

Tatu Juvonen, M.D.*, M. Arisan Erigan, M.D., Jan D. Galla, M.D.*, Steven L. Lansman, M.D., Jock N. McCullough, M.D.*, Khanh H. Nguyen, M.D.*, David Spielvogel, M.D.*, James J. Klein, M.D.*, Carol A. Bodian, Dr.PH.* and Randall B. Griepp, M.D.

New York, New York

Discussant: D. Craig Miller, M.D., Stanford, California

In an attempt to identify risk factors for rupture and to improve management of patients with type B dissection who survive the acute phase without operation, we have studied 59 patients using serial computer-generated 3-dimensional CT scans. Patients were included if they did not undergo surgery during the acute phase of a documented type B dissection, and had at least two CT scans a minimum of three months apart thereafter. The median duration of followup was 4.4 years (range 0.3-9.1), and the median interval between scans was 204 days (98-655) in the unoperated group and 258 (61-443) days in those who ruptured.
During followup, four patients died of causes unrelated to the aneurysm, and nine patients underwent elective aneurysm resection because of large aneurysm size, rapid expansion, or

development of symptoms: they are not considered further. Eleven patients (19%) experienced rupture, and 35 remain alive without operation or rupture. Possible risk factors for rupture and various dimensional parameters are shown below: the data for patients with rupture are from their last CT scans, and the data for patients still in nonoperative followup are from their penultimate studies. Data in the table are medians or percents; p values were derived using the Wilcoxon rank sum test for quantitative data, and Fisher's exact test for categorical data.

Risk Factors	***No Operation or Rupture*** *(n = 35)*	***Rupture*** *(n = 11)*	***p value***
Age (years)	67	73	0.05
Years since dissection	3.0	3.4	NS
Male(%)	63	46	NS
Pain(%)	26	55	0.14
COPD(%)	14	46	0.04
Smoking (%)	54	64	NS
Hypertension (%)	69	73	NS
False lumen open (%)	59	71	NS
Aortic Dimensions (3dCT)			
Descending diameter (cm)	4.7	5.4	0.05
Descending volume (cm^3)	268	285	NS
Abdominal diameter (cm)	3.7	4.7	0.02
Abdominal volume (cm^3)	97	171	0.03

Patients in whom aortic rupture occurred were significantly older, and they were significantly more apt to have a history of COPD when compared with those without rupture; they were also somewhat more likely to have complained of pain at the time of their last visit. In addition, the size and extent of the aneurysm appears to have significant predictive value: patients who subsequently experienced rupture had significantly larger descending aortic and abdominal aortic diameters and a significantly larger abdominal aortic volume than were observed in patients whose aneurysms remained intact. These data indicate that older patients with larger and more extensive aneurysms following acute type B dissections, especially if they have a history of COPD, are significantly more likely to experience rupture than younger patients with smaller chronic type B aneurysms. The presence of a persistently patent false lumen does not appear to increase the risk of rupture. Overall, our data from patients who have entered the chronic phase of type B dissection reveal a pattern which bears a striking resemblance to the natural history of patients with nondissecting aneurysms, suggesting that operative guidelines for patients with chronic type B dissections should be similar to those for patients with other types of chronic descending thoracic and thoracoabdominal aneurysms.

*By invitation

28. SURGERY FOR ASCENDING AORTIC AND ARCH ANEURYSM: ANTEGRADE AORTIC PERFUSION AVOID NEUROLOGICAL COMPLICATIONS.

Francesco Musumeci, M.D.*, Giovanni Casali, M.D.*, Frank Dunstan, Ph.D.*, Antonino Marullo, M.D.* and William J. Penny, M.D.*

Cardiff, Wales

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

Discussant: G. Michael Deeb, M.D., Ann Arbor, Michigan

Background. Femoral arterial cannulation is commonly used for cardiopulmonary bypass (CPB) with ascending aortic (AAo) and arch aneurysm. Retrograde cerebral embolization, retrograde aortic dissection and lower limb ischaemia are well recognized complications. In order to minimize these events, since 1991 we have used direct cannulation of the aneurysmal AAo for CPB. This paper reviews our experience and the effectiveness of antegrade aortic perfusion in reducing postoperative neurological events.

Methods. Between December 1991 and June 1997, 84 consecutive pts (50 males; age range 6 to 83 (median 54) yrs) were operated on. Twenty-four (26.4%) were older than 70 yrs. All pts were evaluated by preoperative CT or MRI and perioperative echocardiography. The aneurysm involved the AAo only in 39 pts and in 45 extended also to the aortic arch. When replacing the AAo, the aortic root was also replaced in 71 pts, 9 had root remodelling and 4 aortic valve replacement. In addition, 45 pts had arch replacement (partial 19, total 24, elephant trunk 2). Direct cannulation of the aneurysmal AAo and bicaval venous return was used for CPB. Cerebral protection was achieved with hypothermic circulatory arrest to a temperature of 15 to 28°C. Continuous retrograde cerebral perfusion (RCP) through the superior vena cava was used in 24 pts when circulatory arrest time was longer than 30 min. All pts had a short period of RCP for de-airing prior to re-establishing CPB. The arterial cannula was repositioned within the distal sutureline, proximal to the innominate artery. Median circulatory arrest, cross clamp and CPB time were 19 (range, 8 to 53) min, 89 (range, 48 to 1 64) min and 122 (range, 82 to 206) min, respectively.

Results. Two pts aged 74 and 76 died in hospital (2.5%) due to respiratory (1) and renal (1) failure. Post-operative complications were bleeding (3) and prolonged ventilatory support (4). No cerebral or peripheral embolic complications occurred. At a mean follow up of 29 (3 to 69) months, there were 3 late deaths. The estimated 4 yr survival rate is 93% (SE 7%).

Conclusion. Direct cannulation of the aneurysmal AAo is a simple and safe alternative to femoral artery cannulation. Antegrade flow throughout the procedure associated with a period of RCP at the end of circulatory arrest was not associated with neurological embolic events. These excellent results suggest that this technique is worthy of further evaluation in a prospective manner.

2.45 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION B ADULT CARDIAC SURGERY

Ballroom B, Hynes Convention Center

Moderators: Bruce W. Lytle, M.D.

Marko I. Turina, M.D.

29. MITRAL VALVE REPAIR FOR DEGENERATIVE DISEASE.

A. Marc Gillinov, M.D.*, Delos M. Cosgrove, M.D., John H. Arnold, M.D.*, Nicholas G. Smedira, M.D.*, Joseph F. Sabik, M.D.*, Patrick M. McCarthy, M.D.,

Craig R. Saunders, M.D.*, Bruce W. Lytle, M.D., Robert W. Stewart, M.D.* and Floyd D. Loop, M.D.

Cleveland, Ohio

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

Objective Degenerative mitral valve disease has become the most common cause of mitral insufficiency in the United States. Mitral valve repair is applicable to the majority of these patients and has become the procedure of choice. This study was undertaken to define the operative risk and to determine risk factors for late mortality and reoperation.

Patients and Methods From 1985 to 1996, 1,101 consecutive patients (pts) had primary isolated mitral valve repair for valvular insufficiency caused by degenerative disease. Mean age was 58 ±12 years; 64% were male. Preoperative degree of mitral regurgitation was 3.6 ± 0.9. *Results* There were 4 hospital deaths (0.4%). Long-term followup was available in 1,089 pts (99%), with 4,319 pt-years of follow-up available for analysis. Eight-year actuarial survival was 84% and 8-year freedom from cardiac death was 94%. Univariate analysis identified moderate or severe left ventricular dysfunction ($p<0.001$), older age ($p<0.001$), chordal shortening ($p<0.001$) and earlier year of surgery ($p<0.001$) and annulopasty alone ($p = 0.001$) as risk factors for late mortality. At 8 years, freedom from thromboembolism was 92%, from endocarditis 99%, from anticoagulant-related hemorrhage 99%, and from reoperation 94%. Of the 30 pts who required reoperation for late mitral valve dysfunction, 15 (50%) repairs failed secondary to progressive degenerative disease. Univariate analysis identified chordal shortening ($p<0.02$), earlier year of surgery ($p<0.001$), and annuloplasty alone ($p<0.001$) as risk factors for reoperation.

Conclusions For patients with degenerative mitral valve disease, valve repair is associated with (1) very low operative mortality rates, and (2) low rates of valve related morbidity and mortality. (3) Isolated annuloplasty and chordal shortening jeopardize late results.

*By invitation

30. IMPACT OF CORONARY ARTERY DISEASE AND AGE ON LATE SURVIVAL IN PATIENTS UNDERGOING BIOPROSTHETIC AORTIC VALVE REPLACEMENT.

Gideon Cohen, M.D.*, Tirone E. David, M.D., Christopher M. Feindel, M.D., Joan Ivano, MSc.*, Michael Borger, M.D.* and Susan Armstrong, MSc.*

Toronto, Ontario, Canada

Discussant: Robert W. Emery, M.D., Minneapolis, Minnesota

Prolonged lifespan in a progressively aging population will likely increase the incidence of aortic valve disease and the demand for aortic valve replacement (AYR) well into the new millennium. To ensure maximal event-free survival, age and lifestyle are routinely considered upon prosthetic valve selection (tissue vs. mechanical). The presence of concomitant coronary artery disease (CAD), however, may be overlooked in the decision-making process. METHODS: Data were prospectively collected on 670 patients undergoing bioprosthetic Hancock II AYR between 1982 and 1994. Follow-up was conducted during a four month period in 1996 by clinic visit or telephone interview. All events were verified. Longitudinal survival and freedom from reoperation were evaluated univariately by Kaplan-Meier analysis and multivariately by Cox regression.

RESULTS: Follow-up was 99.7% complete at 69 ± 40 mos (median 66 mos; range 0.1-168 mos) with 75% of patients having follow-up of at least 93 mos. Mean patient age was 65 ± 12 yrs (median: 68 yrs; range: 18-86 yrs). Survival was significantly different by Kaplan-Meier analysis for both age <65 (71 ± 4% at 12 yrs) vs age > 65 (36 ± 7%, p = 0.0004) and no CAD (65 ± 4% at 12 yrs) vs CAD (35 ± 8%, p<0.0001). When both variables were combined, patients <65 years of age with no CAD demonstrated the best long-term survival, patients <65 years with CAD *or* patients ‰¥ 65 years without CAD demonstrated intermediate long-term survival, and patients ‰¥ 65 years with CAD demonstrated the poorest long-term survival (p<0.0001). After adjusting for gender, the Cox regression coefficient for age ‰¥ 65 was 0.62 ±0.17 (risk ratio 1.86) and for CAD, 0.50 ±0.16 (risk ratio 1.86). Only 24 patients required reoperation for primary tissue failure (PTF). CAD did not univariately nor multivariately influence the need for reoperation. At 12 yrs, freedom from reoperation for PTF was 83 ± 5% for those <65 yrs and 98 ± 1% for those ‰¥ 65 yrs (p = 0.006). Age ‰¥ 65 was associated with a reduced risk for reoperation: Cox regression coefficient -1.24 ± 0.5 (risk ratio 0.29).

CONCLUSIONS: Long-term survival following AYR is highly dependent upon age and the presence of concomitant CAD. Thus, patients greater than 65 years of age with CAD are ideal candidates for a bioprosthetic valve. In patients greater than 65 years without CAD, the benefit of a bioprosthesis is less clear. Age has an independent effect on the durability of the porcine bioprosthesis. Although older patients with CAD are unlikely to require reoperation for bioprosthetic failure, older patients without CAD and all younger patients may require reoperation for this purpose.
*By invitation

31. CRYOPRESERVED HOMOGRAFT VALVES IN THE PULMONARY POSITION - RISK ANALYSIS FOR LATE FAILURE.

†Christopher J. Knott-Craig, M.D.*, Kazuo Niwaya, M.D.*, Ronald C. Elkins, M.D, Mary M. Lane, Ph.D.*, K. Chandrasekaran, M.D.* and Kent E. Ward, M.D.*

Oklahoma City, Oklahoma

Discussant: Gordon K. Danielson, Jr., M.D., Rochester, Minnesota

To examine their durability, and factors associated with late failure, we reviewed our entire experience (1986-1997) with 369 hospital survivors in whom cryopreserved homografts (aortic n = 37, pulmonary n = 332) were used in the "pulmonary" position. Mean age was 18.5 ± 15.7 years (range 2 days -62 years). Operations included Ross operation (n = 273), Tetralogy of Fallot (n = 48), truncus arteriosus (n = 22), transposition of great arteries (n = 16), others (n=10). Median follow-up was 3.3 years (range 0.1-10.8 yrs) and recent echocardiographic evaluation was complete for 97% pts. Reoperation occurred in 7.3% (27/369), and failure (reoperation, gradient ‰¥ 40mmHg) in 12.7% (47/369) pts. Freedom from reoperation at 8 years was 83 ± 4%, and freedom from failure was 72±4%; for aortic versus pulmonary homografts, this was 38 ± 11% vs 77 ± 14% (p = 0.001). For pts <3 yrs at operation (n = 41) this was 34 ± 12% vs 75±5% for older pts (p = 0.0002). Multivariate analysis identified (i) younger age of homograft donors, (ii) aortic homografts, (iii) non-Ross operation, and (iv) later year of operation as risk factors for homograft failure (all p<0.005).
Homografts are the replacement conduit of choice for the pulmonary position. Pulmonary homografts are more durable than aortic homografts, and both fail earlier in young children. Residual pulmonary vascular abnormalities adversely impact the long-term durability of the conduit.
†1989-90 AATS Graham Fellow

*By invitation

32. A 20-YEAR EXPERIENCE WITH CARDIAC RETRANSPLANTATION.

Ranjit John, M.D.*, Jonathan Chen, M.D.*, Alan Weinberg, M.S.*, ‡Mehmet C. Oz, M.D., Silviu Itescu, M.D.*, Eric A. Rose, M.D. and Niloo M. Edwards, M.D.*

New York, New York

Discussant: William A. Baumgartner, M.D., Baltimore, Maryland

Retransplantation is the only currently available treatment for severe myocardial dysfunction in cardiac allograft recipients. However, the allocation of scarce organs to patients who have previously received a transplant is a controversial issue. We, retrospectively, reviewed our experience with patients undergoing cardiac retransplantation. Between 1977 - October 1997, 952 patients underwent cardiac transplantation for the treatment of end stage heart disease. Of these, 43 patients underwent retransplantation for cardiac failure resulting from transplant coronary artery disease, rejection and early graft failure. The mean age of these patients at primary transplantation was 37.2 years (range 2 -58 years, SD ± 16.7) and mean age at retransplantation was 41.6 years (range 5-64 years, SD ± 16.6). The interval between primary and retransplantation ranged from < 1 day to 11.4 years (mean 4.4 years). There was no significant difference in actuarial survival by Kaplan Meier analysis at 1, 2 and 5 years between patients undergoing primary and retransplantation -76%, 71% and 60% versus 66%, 66% and 45% respectively ($p = 0.213$). However, the actuarial survival of retransplant patients from the time of the first transplant was 91%, 88% and 74% at 1, 2 and 5 years respectively. A univariate analysis of various risk factors affecting outcome after cardiac retransplantation was performed. The variables evaluated ‘included the decade of transplantation, ages at primary and retransplantation, indications for primary and retransplantation, interval between transplants, HLA matching, recipient and donor demographic variables and the presence of pretransplant anti-HLA IgG and IgM antibodies. A shorter duration between transplants ($p = 0.003$) and older age at transplantation ($p = 0.04$) were identified as risk factors for poorer outcome after retransplantation. In conclusion, cardiac retransplantation can be performed with low morbidity and mortality and satisfactory long term outcome. However, patient characteristics and preoperative variables should assist in the rational application of retransplantation to ensure optimal utilization of donor organs.

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

Ballroom B, Hynes Convention Center

6:30 p.m. MEMBER RECEPTION

Museum of Science

‡1994-96 AATS Robert E. Gross Research Scholar

*By invitation

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

Ballroom C, Hynes Convention Center

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

Victor F. Trastek, M.D.

33. RESECTION MARGINS, N2 STATUS AND CELL TYPE DETERMINE SURVIVAL IN TRIMODALITY THERAPY OF MALIGNANT PLEURAL MESOTHELIOMA (MPM): RESULTS IN 183 PATIENTS.

David J. Sugarbaker, M.D., Raja M. Flores, M.D.*, Michael T. Jaklitsch, M.D.*, William G. Richards, Ph.D.*, Malcolm M. DeCamp, M.D.*, Scott J. Swanson, M.D.*, Raphael Bueno, M.D.* and Steven J. Mentzer, M.D.

Boston, Massachusetts

Discussant: Larry R. Kaiser, M.D., Philadelphia, Pennsylvania

Objectives: We review our experience with extrapleural pneumonectomy (EPP) in the multimodality management of MPM. *Methods:* From 1980 to 1997, 183 consecutive patients underwent trimodality therapy involving EPP followed by adjuvant chemotherapy and radiotherapy. Standardized systematic pathologic analysis was undertaken. Results: The cohort included 43 women and 140 men with a mean age of 51yrs (range 31- 76) and a median followup interval of 13 months. Overall survival was 36% at 2 yrs and 14% at 5 yrs (median 17 mo). There were seven perioperative deaths (3.8% mortality). Factors affecting long-term survival were evaluated in 176 patients surviving surgery (among these, survival was 38% at 2 yrs and 15% at 5 yrs; median 19 mo). As indicated in the table below, lack of N2 nodal involvement, negative resection margins and epithelial histology were associated with improved survival. Factorial grouping by N2 status and resection margins significantly stratified survival among all patients surviving surgery ($p<0.02$), and among those with epithelial histology ($p<0.02$). Thirty-one patients with epithelial tumors, negative resection margins and without N2 involvement had a 51 mo median survival (68% 2-yr. 46% 5-yr). A clinico-pathologic staging system previously published stratified survival ($p< 0.05$; see table).

Epithelial	103	52%	21%	26	0.0001
Mixed/Sarcom.	73	16%	0%	13	
Margins (+)	110	33%	9%	15	0.02
Margins (-)	66	44%	25%	23	
N2(+)	40	23%	0%	14	0.004
N2(-)	136	42%	17%	21	
Epithel. N2(+)	21	38%	0%	20	0.052
Epithel. N2(-)	82	56%	24%	34	
Epithel. N2(+) Marg(+)	12	29%	0%	14	0.013
Epithel. N2(+) Marg(-)	9	43%	0%	22	
Epithel. N2(-) Marg(-)	51	49%	14%	22	
Epithel. N2(-) Marg(-)	31	68%	46%	51	
Stage I	66	53%	20% 14% 14%	25	0.048
Stage II	41	44%		20	
Stage III	69	17%		16	

These data support the following conclusions: 1) Multimodality therapy including EPP is feasible in selected patients with MPM, 2) Microscopic resection margins affect long-term survival,

pointing to the need for further investigation of local-regional control strategies, 3) Mediastinoscopy to evaluate N2 nodes is recommended, and 4) Patients with epithelial, margin-negative, N2-negative resection enjoy long-term survival.
*By invitation

34. INDUCTION CHEMOTHERAPY FOR T4 CENTRALLY LOCATED NON SMALL CELL LUNG CANCER.

Erino A. Rendina, M.D.*, Federico Venuta, M.D.*, Tiziano De Giacomo, M.D.*, A. Maria Ciccone, M.D.* Giorgio Furio Coloni, M.D.* and Costante Ricci, M.D.*

Rome, Italy

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

Discussant: L. Penfield Faber, M.D., Chicago, Illinois

We employed induction chemotherapy in a prospective, single institution clinical trial intended to achieve resectability in patients with centrally located, irresectable T4 NSCLC. Other types of T4 (pleural effusion, N3) were excluded. Between January 1990 and April 1996 we enrolled 57 patients with histologically confirmed NSCLC. They all underwent Computed Tomography (CT), bronchoscopy, bone scan and in selected cases Magnetic Resonance (MR). Eligibility criteria for T4 were: Clinical [Superior Vena Cava Syndrome (9 patients), vocal cord paralysis (6 patients), dysphagia from esophageal involvement (1 patient)]; Radiological (CT and MR evidence of infiltration - 10 patients); Bronchoscopic (tracheal infiltration - 11 patients); Thoracoscopic (histologically proved mediastinal infiltration - 20 patients). Mediastinoscopy was employed in 38 patients. N2 was histologically confirmed in 40 patients. After 3 cycles of cisplatin (120 mg/m^2), vinblastine (4 mg/m^2), mitomycin (2 mg/m^2) patients were reevaluated: 42 (73%) (36 males. 6 females: age range 42-75 years; mean 58 years) responded to therapy and underwent thoracotomy; 11 did not respond and 4 had major toxicity. Thirty-six patients (85%) had complete resection. We performed 4 exploratory thoracotomies, 6 pneumo-nectomies, 32 lobectomies (20 associated with reconstruction of hilar-mediastinal structures). Pathologically, 4 patients had T4, 6 had T3, 24 had T2, 3 had T1 and 5 had T0. Thirteen patients had N2, 15 had N1 and 14 had N0. Overall, 4 patients had no histological evidence of disease. We had 2 bronchopleural fistulas with 1 death, 5 other major complications and 9 cases of delayed lung reexpansion. Adjuvant chemo and/or radiotherapy was administered to N2 and N1 patients. After a follow up of 15 to 76 months (mean 26 months), 25 patients died of cancer and 2 died of unrelated causes: 14 are alive and free of disease and 1 is alive with disease. Survival at 1 and 4 years is 69% and 26% (median survival 25 months). In conclusion, of the initial group of 57 patients, 42 (73%) underwent exploration with a 4 year survival of 26%, and 36 (63%) had complete resection. Our data indicate that induction chemotherapy is effective for downstaging and surgical reconvertion of centrally located, irresectable T4 NSCLC. Survival is promising but it remains to be confirmed in larger series of patients and phase III trials.

*By invitation

35. THE PROGRESSION OF INTESTINAL METAPLASIA IN GASTROESOPHAGEAL REFLUX DISEASE.

Stefan Oberg, M.D.*, Jeffrey H. Peters, M.D.*, Peter F. Crookes, M.D.*, John J. Nigro, M.D.*, Jorg Theisen, M.D.*, Jeffrey A. Hagen, M.D.* and Tom R. DeMeester, M.D.

Los Angeles, California

Discussant: Victor F. Trastek, M.D., Rochester, Minnesota

It is currently suggested that Barrett's esophagus appears abruptly and once present does not increase in length. This widely quoted but unproven hypothesis is based on data from patients with traditional, long segment Barrett's esophagus with endstage gastroesophageal reflux disease (GERD). Increasingly it is recognized that shorter segments of intestinal metaplasia are part of the same underlying disease process. The aim of the present study was to relate physiologic abnormalities of GERD to increasing lengths of intestinal metaplasia.

One hundred thirty consecutive patients with symptoms suggestive of GERD and intestinal metaplasia of the cardia or esophagus on biopsy during upper endoscopy, were studied by esophageal motility and 24 hour pH and bilirubin monitoring. Duration of symptoms was documented. Standard measures of lower esophageal sphincter (LES) function, esophageal acid and bilirubin exposure, and lower esophageal peristaltic amplitude were assessed. Patients were divided into three groups based on the extent of intestinal metaplasia: Cardiac intestinal metaplasia (CIM) where the intestinal metaplasia was detected on biopsy in an endoscopically normal appearing cardia, short segment Barrett's Esophagus (SSBE) group with intestinal metaplasia in the tubular esophagus less than 3cm, and patients with long segment Barrett's Esophagus (LSBE).

	CIM (n = 30)	SSBE (n = 32)	LSBE (n = 68)
Duration of GERD symptoms (years)	10.0 (5-15)	10.0 (3-15)	17(10-23)*
% time esophageal pH < 4	6.5 (3.3-8.4)	10.0 (5.5-16.4)*	21.3(13.2-41.1)*
% time esophageal bilirubin abs. > 0.2	1.6 (0.0-12.8)	14.8 (1.7-25.4)	16.9 (4.3-30.5)*
LES resting pressure (mmHg)	6.6 (3.5-11.8)	5.2 (2.3-10.2)	3.2(1.2-7.4)†
LES abdominal length (cm)	1.0 (0.4-1.4)	0.6 (0.2-1.2)	0.2 (0.0-0.8)†
Prevalence of defective LES (%)	58.6	65.9	87.5*
Esophageal contraction amplitude (mmHg)	64.5 (39.2-98.5)	56.5 (38.8-83.7)	51.0 (35.0-74.0)*

Values expressed as medians (IQR) * $p < 0.05$ vs all other groups, †$P < 0.05$ vs CIM

Length of intestinal metaplasia was strongly correlated with the degree of esophageal acid exposure ($r = 0.71$, $p = 0.0001$), and inversely with the LES pressure ($r = -0.26$, $p = 0.003$). Patients with long segment Barrett's esophagus had a longer duration of symptoms than those with short segment or cardia metaplasia only. The LSBE group demonstrated the greatest degree of physiologic derangement and the CIM group the least.

The results of this study suggests that longer segments of IM are associated with longer duration of reflux symptoms and increased severity of reflux disease. Intestinal metaplasia at the cardia is characterized by the same physiologic abnormalities as traditional Barrett's esophagus, although of lesser degree. This raises the possibility that shorter segments of intestinal metaplasia may progress to longer segments as the antireflux mechanism deteriorates.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

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

Ballroom C, Hynes Convention Center

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

Victor F. Trastek, M.D.

36. PLEURAL SPACE IRRIGATION AND MODIFIED CLAGGETT PROCEDURE FOR THE TREATMENT OF EARLY POSTPNEUMONECTOMY EMPYEMA.

Farid Gharagozloo, M.D.*, Gregory D. Trachiotis, M.D.*, Andrew J. Wolfe, M.D.*, Kevin M. DuBree, P.A.C.* and James L. Cox, M.D.

Washington, DC

Discussant: Peter C. Pairolero, M.D., Rochester, Minnesota

The incidence of postpneumonectomy empyema is 5 - 10%. Approximately half of postpneumonectomy empyemas occur within 2 to 4 weeks of pneumonectomy. A bronchopleural fistula is found in more than 80% of the patients. The classic treatment of postpneumonectomy empyema includes parenteral antibiotics, drainage of the pleural space, removal of necrotic tissue, and open pleural packing for many weeks followed by obliteration of the empyema space with antibiotic fluid or muscle. This approach results in prolonged hospitalization, repeated operations, and significant morbidity. In a five year period we treated 22 patients with early postpneumonectomy empyema. All patients had a bronchopleural fistula. The patient profile and predisposing factors were:

Sex	Age	Pleural Space	Bronchiectasis	PreOp Radiation
16M/16F	62 ± 5	18R/4L	2	3
		Malnutrition	Steroid Therapy	Obstructive Pneumonitis
		2	3	12

All patients underwent emergent drainage of the pleural space followed by thoracotomy, debridement of necrotic tissue, closure of the bronchial stump with nonabsorbable monofilament suture, and pleural space irrigation. Pleural space irrigation consisted of two irrigation catheters placed at the apex of the chest and two chest tubes placed in dependent positions anteriorly and posteriorly. The pleural space was irrigated with 0.1% betadine solution (40 ml/hr) for days 1 through 7. On day 8, the betadine irrigation was discontinued and the pleural space was irrigated with normal saline (40 ml/hr) for 24 hours. On day 9 a gram stain of the chest tube drainage fluid was obtained. If the gram stain was positive for organisms or leukocytes, betadine irrigation was resumed and continued for days 9 through 15, followed by saline irrigation on day 16 and a repeat gram stain on day 17. This cycle was continued until a negative gram stain was obtained. With a negative gram stain the pleural space was filled with 2 liters of Dab's (Gentamycin 80mg/L, Neomycin 500 mg/L, Polymyxin B 100 mg/L) solution, and the irrigation and drainage catheters were removed. 20 patients had negative gram stains on Day 9, 2 patients had a negative gram stain on Day 16. The mean hospitalization was 12.9 ± 3.4 days. All patients were examined at one week, one month, and one year following discharge from the hospital. There was no recurrence of empyema or a bronchopleural fistula. Pleural space irrigation followed by obliteration of the pleural space with an antibiotic solution required one surgical procedure, and resulted in significantly shorter hospitalization, and decreased morbidity in patients with early postpneumonectomy empyema.

*By invitation

37. RESULTS OF REOPERATION ON THE UPPER ESOPHAGEAL SPHINCTER.

Gaetano Rocco, M.D.*, Claude Deschamps, M.D., Elyse Mattel, M.D.*, Andre Duranceau, M.D., Victor F. Trastek, M.D., Mark S. Allen, M.D., Daniel L. Miller, M.D.* and Peter C. Pairolero, M.D.
Rochester, Minnesota and Montreal, Quebec, Canada

Discussant: Mark B. Orringer, M.D., Ann Arbor, Michigan

Between September 1976 and February 1997, 37 patients (29 males and 8 females) underwent reoperation on the upper esophageal spinchter (UES) for recurrent or persistent obstructive symptoms. Median age was 69 yrs (range, 38-87). Original indication for surgery was a Zenker's diverticulum in 33 patients (89.2%), oculopharyngeal dystrophy in 3 (8.1%) and muscular dystrophy in 1 (2.7%), one prior UES operation had been performed in 26 patients (70.3%), two in 9 (24.3%) and three in 2 (5.4%). Median interval between the most recent operation and reoperation was 25 months (range, 1-217). All patients were symptomatic. Thirty-five patients (94.6%) had dysphagia, 23 (62.2%) had regurgitation and 12 (32.4%) had episodes of aspiration. Thirty of 33 patients (91.0%) with Zenker's diverticulum were found to have a recurrent or persistent diverticulum at reoperation. A diverticulectomy and cricopharyngeal myotomy was performed in 23 patients (62.2%), cricopharyngeal myotomy alone in 7 (18.9%), diverticulopexy and cricopharyngeal myotomy in 6 (16.2%), and diverticulectomy alone in 1 (2.7%). There was no operative mortality and 10 patients (27.0%) developed at least one complication. Followup was complete in 33 patients (89.2%) and ranged from 2 to 149 months (median, 38 mts.). Thirty-one patients (94.0%) were improved. Functional results were classified as excellent in 25 patients (75.7%), good in 2 (6.1%), fair in 4 (12.1%) and poor in 2 (6.1%). We conclude that reoperation for those patients who present with persistent or recurrent symptoms after surgery on the UES is associated with low morbidity and mortality. Resolution of symptoms occurs in the majority of patients.
*By invitation

38. IS RESECTION OF PULMONARY AND HEPATIC METASTASES WARRANTED IN COLORECTAL CANCER PATIENTS?

Brigitta J. Robinson, M.D.*, Scott A. Strong, M.D.* and Thomas W. Rice, M.D.

Cleveland, Ohio

Discussant: Mark S. Allen, M.D., Rochester, Minnesota

Isolated hepatic or pulmonary metastasis can be resected for a potential cure in patients with colorectal carcinoma; however, little data is available to determine the results of resection of both hepatic and pulmonary metastases. Therefore a retrospective study was undertaken to evaluate the results of resection of both pulmonary and hepatic metastases in colorectal carcinoma patients. Over a 16 year period, March 1979 to June 1995, 25 patients (pts) underwent resection of colorectal carcinomas and both hepatic and pulmonary metastases. Fifteen (60%) pts were female. Median age was 57 years (range 37 to 71). The stage of the colorectal carcinoma was I in 2 (8%) pts, II in 6 (24%), III in 11 (44%) and IV in 6 (24%). Synchronous resection was defined as resection of a metastasis within 3 months of a previous resection: 3 (12%) pts had synchronous resections of the primary and both metastatic sites and 7 (28%) had synchronous resection of the primary and one metastatic site followed by metachronous resection of the second metastatic site (6 primary and liver, then lung and 1 primary and lung, then liver). Fifteen (60%) pts underwent metachronous resections (10 primary, then liver, then lung and 5 primary, then lung, then liver). Chemotherapy was administered following colorectal resection in 12 (48%) pts and after metastatectomy in 4

(16%). Median follow-up was 42 months (range 15 to 201). Overall survival was 41% at 5 years with a median of 47 months (Fig. 1). The timing of resections did not influence survival (Fig.2).

This study indicates that if the colorectal carcinoma is or can be curatively resected, then resection of both hepatic and pulmonary metastases, regardless of the disease-free interval or the timing of resections, should be undertaken in patients with adequate cardiopulmonary reserve and metastases confined to the liver and lungs.
*By invitation

39. DIFFUSING CAPACITY PREDICTS OPERATIVE MORTALITY BUT NOT LONG-TERM SURVIVAL AFTER RESECTION FOR LUNG CANCER.

†Jun Wang, M.D.*, Jemi Olak, M.D.* and ‡Mark K. Ferguson, M.D.

Chicago, Illinois

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

Background Diffusing capacity predicts operative mortality after major lung resection. It is also a determinant of intermediate term pulmonary morbidity after lung cancer resection and of long-term survival in patients with emphysema. We sought to determine whether diffusing capacity also influences long-term survival after resection for lung cancer.

Methods We retrospectively reviewed the records of patients who underwent major lung resection (lobectomy, bilobectomy, or pneumonectomy) for non-small cell lung cancer from 1980 through June, 1997. Operative mortality was compared to the postoperative predicted diffusing capacity (expressed as a percent of predicted; ppoDLCO%) by chi-squared analysis. Long-term survival was calculated using the Kaplan-Meier method and comparisons between groups were made with the log-rank test using ppoDLCO% = 50 as a cutoff value.

Results There were 410 patients (242 men, 168 women) with a mean age of 62.3 years (range 35 to 87 years). There were 196 patients with squamous cell cancer, 186 with adenocarcinoma, and 14 each with large cell cancer or bronchoalveolar carcinoma. We performed 273 lobectomies, 35 bilobectomies, and 102 pneumonectomies. The pathological stage was I in 184 patients, II in 68, IIIa in 143, IIIb in 8, IV in 4, and unknown in 3 patients. There were 32 operative deaths (7.8%) that were closely related to ppoDLCO% (< 40 =19.6%; 40-50=14.6%; 50-60 = 4.4%; >60 = 3.5%; d.f. = 3, Ï‡^{2} = 18.01, p<0.0001). Examining only operative survivors, survival (mos) categorized by stage was not influenced by ppoDLCO%:

	ppoDLCO%‰¤50			ppoDLCO%>50			
Stage	Pts	Mean	Median	Pts	Mean	Median	p Value
I	22	98.6	110.0	116	101.6	89.0	0.516
II	12	52.4	26.6	35	52.0	32.0	0.631
IIIa	32	31.4	31.0	61	47.2	26.0	0.715

Conclusions The use of diffusing capacity to select candidates for operation should be tempered by the knowledge that a poor diffusing capacity does not adversely affect long-term survival. Efforts should be directed at improving the perioperative management of patients undergoing major lung resection to enable inclusion of more patients with reduced diffusing capacity in the candidate pool for surgery, thus maximizing the long-term survival of lung cancer patients.

4:35 p.m. EXECUTIVE SESSION (Limited to Members)
Ballroom B, Hynes Convention Center

6:30 p.m. MEMBER RECEPTION

Museum of Science

†1997-98 AATS Graham Fellow

‡1986-88 AATS Edward D. Churchill Research Scholar

*By invitation

WEDNESDAY MORNING, MAY 6, 1998

7:00 a.m. GENERAL THORACIC SURGERY

FORUM SESSION
Ballroom C, Hynes Convention Center

Moderators: Larry R. Kaiser, M.D.

David J. Sugarbaker, M.D.

F9. HUMAN PLEURAL MESOTHELIOMAS CONTAIN SIMIAN VIRUS-40 (SV40) REGULATORY AND LARGE TUMOR (T) ANTIGEN DNA SEQUENCES.

Harvey I. Pass, M.D., Jessica S. Donington, M.D.*, Peter Wu, M.D.*, Paola Rizzo, Ph.D.*, Ronald Kennedy, Ph.D.* and Michele Carbone, M.D, Ph.D.*

Detroit, Michigan; Washington, DC; Bethesda, MD;

Chicago, Illinois and Oklahoma City, Oklahoma

A cohort (20%) of mesothelioma (meso) patients will not have an exposure to asbestos. Recently, a DNA tumor virus (SV40) has been shown to cause hamster mesos, and we previously described SV40-like DNA amino terminus sequences in 29 of 48 mesos *(Oncogene* 9:1781-90, 1994). We analyzed an *additional* 42 mesos to determine (1) whether our initial observations were durable (2) if other regions of the SV40 genome were present and (3) whether meso patients exhibited an immune response to SV40. **METHODS:**Genomic DNA was extracted from snap frozen meso tumor samples and from the SV40-induced hamster meso tumor H9A. PCR primers were used to amplify various SV40 large T antigen (T-ag) regions including a 105 bp *amino terminus* fragment, a 281 bp *carboxy terminus* fragment, and a 310 bp fragment of the *enhancer promoter* region. Endonuclease digestions were used to verify the expected product. SV40 T-ag specificity and liter of human serum antibodies were examined in 31 mesothelioma patients using an enzyme-linked immunosorbent assay (ELISA). **RESULTS:** 30 of the 42 (71%) samples amplified T-ag amino sequences, and specificity was verified by Southern hybridization. Thirteen of 42 samples (31%) amplified the appropriate size fragment for the carboxy terminus, and digestion with BsaB I matched that of H9A. Twenty of 42 samples (48%) amplified SV40 regulatory sequences and Fok I digestion matched that of the hamster control tumor. Sequence analysis (4 patients) revealed 100% homology with the regulatory region of SV40 strain 776. Compared to non-SV40 exposed controls, the frequency of serum antibodies to T-ag in meso was significantly greater ($P_2 =$

0.045). **CONCLUSIONS:** These data suggest an association between the SV40 virus and human mesothelioma which could be exploited for diagnostic/therapeutic options including early detection and potential vaccination strategies.

*By invitation

F10. EVIDENCE THAT DIFFUSION CAPACITY LIMITS LUNG VOLUME REDUCTION SURGERY IN A RABBIT MODEL OF EMPHYSEMA.

John C. Chen, M.D.*, Dan L. Serna, M.D.*, Ledford L. Powell, M.D.*, Henry E. Aryan, B.S.*, Robert J. McKenna, M.D., Arthur Gelb, M.D.* and Matthew Brenner, M.D.*

Orange and Irvine, California

Purpose: Lung volume reduction surgery (LVRS) has been suggested to improve lung compliance and expiratory flows in patients with obstructive emphysema. Endpoints for optimal resection volumes are not known. Spirometric improvements may have to be weighed against reduction in gas exchange surfaces as larger volumes of lung tissue are removed. The purpose of this study was to evaluate the effects of LVRS on lung diffusion capacity (D_LCO), pulmonary compliance, and airway flow in a rabbit model of emphysema.

Methods: Forty-nine rabbits were induced with 15,000 units of elastase by aerosolization through an endotracheal tube. Emphysema was confirmed histologically 5 weeks following induction. Single breath D_LCO, static pressure volume relationships, and forced expiratory flows (FEF 33%) were measured at baseline prior to induction, preoperatively at 4 weeks following induction, and 1 week postoperatively following sham sternotomy (n = 10) or bilateral upper lobe LVRS (n = 39).

Results: Transpleural pressures with 60 cc insufflation above FRC increased with resection of larger volumes of lung tissue (Graph 1, ANOVA p = 0.000). Comparison of changes in D_LCO reveal diminishing diffusing capacity with increasing amounts of lung volume resected (Graph 2, p = 0.063). In contrast, early expiratory flow improved most in the rabbits with 2.3-3 grams of lung tissue resected (Graph 3, p = NS).

Conclusions: Static lung compliance continues to improve with escalating volumes of lung resected. Decreased compliance and increased airway flow following LVRS in this animal model parallel findings in clinical studies. These findings support the hypothesis that mechanisms of improved elastic recoil and airway resistance contribute to patient improvement. Our findings suggest that there exists an optimal resection volume range for improvement in early expiratory flow and D_LCO, which may help to explain clinical response. Supported by ALA #CI-030-N,CTRDRP #6RT-0158, DOE #FG03-91ER61227.

*By invitation

F11. ADJUVANT TREATMENT OF REFRACTORY LUNG TRANSPLANT REJECTION WITH EXTRACORPOREAL PHOTOPHORESIS.

C.T. Salerno, M.D.*, Soon J. Park, M.D.*, David M. Kulick, M.D.*, Nathan S. Kreykes, B.S.*, Marshall I. Hertz, M.D.* and R. Morton Bolman, III, M.D.

Minneapolis, Minnesota

Photophoresis (PPE) is a technique in which a patient's leukocytes, removed by apheresis, are exposed to ultraviolet-A light after pretreatment with 8-methoxypsoralen, a photosensitive drug activated by long-wavelength ultraviolet light. The irradiated mononuclear suspension is subsequently re-infused to the patient. PPE has been used for the treatment of autoimmune diseases, T cell lymphoma and the treatment of acute rejection in heart transplant recipients. PPE appears to induce specific suppression of both cellular and humoral rejection. The altered lymphocytes produce a suppressor response that targets unirradiated T cells of similar clones in a mechanism that is not yet elucidated. To date there have been few published reports detailing the use of PPE in lung transplant recipients. We have used PPE, in addition to standard anti-rejection chemotherapy in 7 lung transplant recipients with refractory rejection since 1992. All 7 patients had progressively worse graft function and were BOS grade 3 prior to the initiation of photophoresis. There were 3 women and 4 men. Two patients had a pre-transplant diagnosis of COPD, one a-1 antitrypsin deficiency, one cystic fibrosis, one bronchiectasis and two primary pulmonary hypertension. Prior to developing refractory rejection all patients had been treated with three drug immunosuppression, and anti T-cell therapy. The mean time from transplant to the initiation on PPE was 16 months (range 7-32). The mean number of treatments was 6 (range 3-12). RESULTS: Five of six patients subjectively improved after PPE therapy. In these 5 patients PPE was associated with a stabilization in the measured FEV_1. In two patients there was biopsy proven reversal of rejection following PPE. With a mean follow up of 28 months (range 2-53), all 7 patients are alive and well. Three patients required re-transplant at a mean period of 19 months (range 15-21) after completion of the PPE treatments. Four patients have remained stable post PPE with only subtle changes in their immunosuppressive therapy. There were no PPE related complications. We believe that this treatment is a safe option for patients with refractory lung allograft rejection when increased immunosuppression is contraindicated or ineffective.

*By invitation

F12. BILE ACIDS INDUCE CYCLOOXYGENASE-2 AND PROSTAGLANDIN SYNTHESIS: A POTENTIAL MECHANISMOF ESOPHAGEAL CARCINOGENESIS.

Fan Zhang, M.D., Ph.D.*, Andrew J. Dannenberg, M.D.* and Nasser K. Altorki, M.D.

New York, New York

Bile reflux has been implicated in the genesis of esophageal cancer in animals and humans but the underlying mechanism(s) remains unclear. A large body of data from a variety of experimental systems suggest that cyclooxygenase-2 (Cox-2), the inducible form of Cox, is important in tumor formation because it catalyzes the synthesis of prostaglandins (PCs) from arachidonic acid, inhibits apoptosis and bioactivates chemical carcinogens. We investigated, therefore, the effects of bile acids, known endogenous promoters of colon cancer, on the expression of Cox-2 and synthesis of PCs in a human Barrett's adenocarcinoma cell line. Treatment with the unconjugated dihydroxy bile acids, chenodeoxycholate (CD) and deoxycholate (DC), resulted in about a 10-fold increase in

the production of PGE_2 Enhanced synthesis of PGE_2 was associated with a marked increase in levels of Cox-2 mRNA (Fig. 1) and Cox-2 protein (Fig. 2) with maximal effects at 8-12 h and 12-24 h, respectively. In contrast, neither cholic acid nor conjugated bile acids affected levels of Cox-2 or synthesis of PGE_2.

Bile acid-mediated induction of Cox-2 was blocked by inhibitors of protein kinase C activity, including calphostin C and staurosporine. Bile acids were also potent inducers of Cox-2 in esophageal squamous cell carcinoma cell lines. These results may explain, in part, the mechanism by which bile reflux contributes to the pathogenesis of esophageal cancer.

*By invitation

F13. LIPOSOME-MEDIATED GENE TRANSFER IN RAT LUNG TRANSPLANTATION: A COMPARISON BETWEEN THE *IN VIVO* AND *EX VIVO* APPROACH.

Carlos H. Boasquevisque, M.D.*, Bassem Mora, M.D.*, Mariano Boglione, M.D.*, Jonathan S. Bromberg, M.D.*, Ronald K. Scheule, Ph.D.*, Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.
St. Louis, Missouri; Ann Arbor, Michigan;

Framingham, Massachusetts

Background: We have recently studied the pattern of *in vivo* and *ex vivo* liposome-mediated gene transfer to rat lung isografts using the reporter gene chlorarnphenicol acetyl transferase (CAT). We also demonstrated that *ex vivo* transfection of TGF(3-1 cDNA into rat lungs improved allograft function. In this study we compared the efficacy of *in vivo* and *ex vivo* transfection in rat lung transplantation. Methods: Animals were divided into two groups according to the transfected cDNA. (1) CAT: Fischer rats underwent isogeneic left lung transplant and were divided into 6 groups (n = 4). In group I grafts were infused *ex vivo* with 660 µg of liposome-CAT cDNA and transplanted immediately (1-1/2 hours of exposure at 10°C). In group II animals underwent the same procedure but the exposure time was 10 hrs. In group III *(in vivo),* donors received an intrajugular injection of 1320 µg of CAT cDNA. Left lungs were harvested after 1-1/2 hours at 10°C and transplanted. In group IV grafts were transfected *in vivo,* harvested after 10 hours and preserved for 10 hours at 10°C. In groups V and VI, grafts were transfected *in vivo* and *ex vivo* respectively with CAT cDNA not complexed to liposomes. The exposure time was 1 1/2 hours in both groups. In all groups recipients were sacrificed at two days post transplant for CAT assay. (2) TGFÎ²-1 Brown-Norway rats served as donors and Fischer rats as recipients. Animals were divided into 4 groups. In group I *(in vivo,* n = 7) donors received an intrajugular injection of 1320 ng of TGFÎ²-1 sense cDNA complexed to liposomes. Left lungs were harvested 3 hours later. In group II (n = 7) grafts were harvested and then infused *ex vivo* with 660 µg of TGFÎ²-1 sense cDNA complexed to liposomes. In group 111 (n = 5) grafts were harvested and then infused *ex vivo* with 660 µg of TGFÎ²-1 anti-sense complexed liposomes. In group IV (n = 5) lung grafts were harvested and transfected *ex vivo* with TGFÎ²-1 sense cDNA not complexed to liposomes. All grafts were preserved for 3 hours at 10°C prior to transplantation. Recipients were sacrificed on postoperative day 5 for arterial oxygenation and histologic assessment of rejection score. **Results:** (1) CAT - Transfection was clearly superior in the *ex vivo* subgroup. Transfection was similar for both exposure times. CAT cDNA alone was inefficient either *in vivo* or *ex vivo.* (2) TGFÎ²-1 oxygenation was superior in the *ex vivo* liposome-TGFÎ²-1 sense group in comparison to the *in vivo* liposome TGFÎ²-1 sense group and *ex vivo* liposome TGFÎ²-1 anti-sense group. Allograft

function was superior in both groups treated *ex vivo* and *in vivo* with TGFÎ²-1 sense-liposomes compared to TGFÎ²-1 sense cDNA alone. Rejection scores were not significantly different between the *ex vivo* liposome-sense versus *in vivo* liposome-sense subgroups. However rejection scores were superior after liposome-sense transfection compared to liposome-anti-sense and cDNA alone groups, **Conclusions:** (1) using current liposome technology the *ex vivo* approach is superior to the *in vivo* approach in experimental lung transplantation. (2) Infusion of cDNA not complexed to liposomes was sufficient to provide transgene expression but not at levels high enough to produce a functional effect.

*By invitation

F14. INDUCTION CHEMOTHERAPY, SURGICAL RESECTION AND RADIOTHERAPY IN PATIENTS WITH MALIGNANT PLEURAL EFFUSION, MEDIASTINO-SCOPY NEGATIVE(STAGE IIIB) NON-SMALL CELL LUNG CANCER.

Scott J. Swanson, M.D.*, Michael T. Jaklitsch, M.D.*, Steven J. Mentzer, M.D., Malcolm M. DeCamp, M.D.*, William G. Richards, Ph.D.*, Raphael Bueno, M.D.* and David J. Sugarbaker, M.D.

Boston, Massachusetts

Objective:The purpose of this study was to determine the feasibility, morbidity and mortality of multimodality therapy including extrapleural pneumonectomy in patients with malignant pleural effusion and negative mediastinal lymph nodes (by mediastinoscopy) with a diagnosis of non-small cell lung cancer (NSCLC) (stage IIIB).

Rationale:Stage IIIB NSCLC on the basis of malignant pleural involvement has a guarded prognosis with average survival of 3-6 months in most series regardless of therapy. A subset of these patients at presentation do not manifest hematogenous or mediastinal lymphatic metastases but only local regional pleural spread. Clinically, these patients can be confused with patients with malignant pleural mesothelioma for whom multimodality therapy may extend survival. Induction chemotherapy followed by surgery has been shown to have a survival benefit in several recent series of patients with stage IIIA disease. Surgical resection in early stage malignant mesothelioma has been shown to be feasible. Radiotherapy has been demonstrated to decrease local recurrence following surgery in NSCLC. A combination of the strategies used to treat malignant mesothelioma and stage IIIA NSCLC might be applicable to a select group of patients with stage IIIB (malignant pleural involvement) NSCLC.

Methods:Between 1994 and 1997, 12 consecutive patients with stage IIIB NSCLC on the basis of malignant pleural involvement were selected to undergo a multimodality treatment regimen. Patients who demonstrated adequate functional reserve (ECOG ps 0-2), who had no significant medical comorbidites and had adequate pulmonary reserve to tolerate a pneumonectomy (predicted postoperative FEV 1 ‰¥ 1) were screened for signs of metastatic disease. Each patient had a bone scan, head ct scan and chest ct scan that included the liver and adrenal glands. Patients without signs of hematogenous metastases underwent mediastinoscopy to rule out mediastinal nodal involvement. In those without N2 or N3 disease, induction chemotherapy was carried out with 3 cycles of a platinum-based regimen. Twelve patients without clinical or radiographic signs of progression went on to have an extrapleural pneumonectomy including prosthetic reconstruction of the hemidiaphragm and pericardium. Seven patients had post-operative external beam

radiotherapy. Perioperative data and followup information was gathered from our prospective thoracic database

Results: This cohort was made up of 6 males and 6 females with a median age of 61.5 years (range 41-69), median follow-up 8.5 months (range 3-25). Histological evaluation of the specimens revealed 9 adenocarcinomas and 3 squamous cell carcinomas. Median length of stay was 7.5 days (range 6-80). Seven patients (58%) received red blood cell transfusions (median 1.5 units, range 0-6). There were no perioperative deaths and 7 (58%) patients had complications (6- atrial fibrillation, 1- aspiration pneumonia, 1- vocal cord paresis). Median survival in the N1 node positive subgroup is 24 months (n = 5). The median survival has not been reached in the node-negative subgroup (n = 7), (p = NS).

Conclusion: Multimodality therapy including surgical resection for stage IIIB NSCLC is feasible in selected patients. This pilot study would suggest that aggressive local regional strategies may be the subject of larger clinical trials in patients with stage IIIB NSCLC.

*By invitation

F15. PIG LUNG PRE-PERFUSION PREVENTS LUNG HYPERACUTE REJECTION IN THE PIG-TO-HUMAN COMBINATION.

Paolo Macchjarini, M.D., Ph.D.*, Rafael Oriol, M.D.*, Robert Rieben, Ph.D.*, Nicolao Bovin, Ph.D.* and Philipe Dartevelle, M.D.

Le Plessis and Villejuif, France; Berne, Switzerland; Moscow, Russia

Background: The clinical use of pig lungs is currently limited by hyperacute rejection (HAR), triggered by the binding of human anti-Î±Gal xenoreactive natural antibodies (hXNA) to endothelial xenograft cells. *Objective:* To test the relative effects of pig organ pre-perfusion and specific depletion of anti-Î±Gal hXNA in preventing HAR in the pig-to-human lung combination. *Methods:* Large White pig (20-25 kg) left lungs were harvested and *ex-vivo* continuously ventilated and reperfused, using a neonatal oxygenating system, with either whole human blood pre-perfused trough donor pigs right lung (group I), liver (group II), spleen (group III) or human plasma filtrated on an immunoabsorbent column with Î±-galactose (GalÎ±l-3GalÎ²(CH$_2$)3NH$_2$; Bdi) (group IV). Each study group included 6 animals. *Results:* Pre-perfusion of human blood through pig organs or the Î±Gal column achieved a similar 89 ± 4% reduction of anti-Î±Gal hXNA in the four groups. All pre-perfusing organs displayed HAR after 15 ± 3 min. Following *ex-vivo* reperfusion, group I lung xenografts had a significantly ($p<0.001$) longer functional survival than groups II, III and IV xenografts, and were the only histologically normal 5 hours upon reperfusion.

Groups	2 min		30 min		60 min		180 min		Xenograft
	PVR	AVO_2	PVR	AVO_2	PVR	AVO_2	PVR	AVO_2	survival
Group I	15 ± 4	24 ± 2	25 ± 10	24 ± 4	18 ± 3	20 ± 2	17 ± 4	17 ± 2	300 ± 54
Group II	24 ± 7	18 ± 4	39 ± 20	16 ± 7	29 ± 11	12 ± 5	27 ± 8	13 ± 3	145 ± 40
Group III	15 ± 6	21 ± 6	48 ± 36	17 ± 7	39 ± 23	9 ± 1	-	-	50 ± 15
Group IV	28 ± 15	21 ± 8	61 ± 24	18 ± 9	47 ± 21	16 ± 7	-	-	78 ± 45

Data are presented as mean ± standard deviation; pulmonary vascular resistance

(PVR: mm Hg/ml/min.); arteriovenous oxygen difference (AVO_2: mlO_2/100 ml).

Human blood reperfusing group I xenografts had a significantly ($p<0.05$) lower: *i)* fall in white blood cells, clotting factors, total circulating immuno-globulins; *ii)* total (CH100) and membrane attack complex (C5b-9) complement activation; and *in)* hemolysis. *Conclusions:* We provide evidence that specific depletion of anti-Î±Gal hXNA alone incompletely protects pig lungs from HAR. It is speculated that the complete protection afforded by lung pre-perfusion relates to a better removal of other critical humoral or cellular elements provoking HAR.

*By invitation

F16. ADENOVIRAL-MEDIATED P53 GENE TRANSFERS TO NON-SMALL CELL LUNG CANCERS.

David Weill, M.D.*, Allan N. Shulkin, M.D.*, Juliette L. Wait, M.D.*, Milissa Tuzzolino, R.N.*, John A. Osborne, M.D, Ph.D.*, John J. Nemunaitis, M.D.* and Michael J. Mack, M.D.

Dallas, Texas

Introduction The p53 gene, a tumor suppressor gene, is often rendered nonfunctional in human non-small cell lung cancer (NSCLC) by mutation or deletion. By restoring the expression of p53, it is postulated that tumor growth could be suppressed. We performed a Phase I study using an adenoviral vector expressing wildtype p53 (Ad-p53) to demonstrate the feasibility of safely delivering the gene and to document any observed antitumor activity.

Materials and Methods A replication-defective adenoviral vector containing wildtype p53 gene was constructed for intralesional injection into NSCLC that had documented p53 mutations. All patients were refractory to conventional treatment including cisplatin chemotherapy (n = 8) or external beam radiation (n = 3). Patients were enrolled into one of two treatment arms: treatment with Ad-p53 alone or treatment with Ad-p53 plus cisplatin. The Ad-p53 injections were performed monthly in patients with bronchoscopically accessible tumors. The study treatments continued as long as there was no tumor progression or unacceptable adverse effects. A maximum of 6 treatments was given. Results The Ad-p53 was injected into 11 patients with primary NSCLC. Doses of Ad-p53 ranged from 1×10^6 to 1×10^{11} plaque forming units. Following the injections, p53 expression was documented in 8 of the 9 evaluable patients. Toxicity after injection of Ad-p53 was minimal and included minor complications associated with the endobronchial biopsy procedure. During the course of treatment, 2 patients developed pneumonia which was likely secondary to the obstructing tumors.

In 6 of the 9 evaluable patients, endobronchial obstruction was relieved as determined by serial bronchoscopic imaging. Disease remained stable in 2 patients and progressive in 1. The average survival of patients enrolled in the study was 139.3 + days (range 16-292+).

Conclusion We demonstrated that Ad-p53 can be safely delivered intralesionally to patients with NSCLC. Toxicity was minimal, and transgenic expression was achieved. Furthermore, apoptosis as measured by relief of endobronchial obstruction was observed in most patients.

*By invitation

F17. A SELECTIVELY REPLICATING ADENOVIRUS (ONYX-015) LYSES NON-SMALL CELL LUNG CANCER CELLS THAT LACK FUNCTIONAL p53.

David M. Jablons, M.D.*, Liang You, M.D, Ph.D.*, Adam Sampson-Johannes, M.S.*, David Kirn, M.D.* and Frank McCormick, Ph.D.*

San Francisco and Richmond, California

Sponsored by: Frank Hanley, M.D., San Francisco, California

Loss or mutation of p53 tumor suppressor gene is a common genetic abnormality in many human tumors including lung cancer. p53 mutations and loss of heterozygosity have been detected in more than 50% of lung cancers. p53 protein prevents replication of damaged DNA in normal cells and promotes apoptosis of cells with abnormal DNA. p53 mutations are frequently associated with poor prognosis in tumor patients. ONYX Pharmaceuticals has genetically designed a tumor-targeting adenovirus (a replication competent E1B-deleted) which only replicates in cells that lack functional p53 gene and therefore kills tumor cells specifically. In an in vitro study, this mutant adenovirus ONYX-015 has been shown to kill cervical carcinoma cells, colon carcinoma cells, glioblastoma ceils and pancreatic adenocarcinoma ceils lacking functional p53. It was also demonstrated that this virus caused a significant reduction in tumor size and caused complete regression of 60% of the tumors induced by p53-defi'cient human cervical carcinoma cells in nude mice. In this study, we tested the cytotoxicity of this mutant adenovirus against two non-small cell lung cancer (NSCLC) cell lines that lack functional p53 gene (NCI-H522 and NCI-HI703) using cytopathic effect (CPE) assays. NCI-H522 is a lung adenocarcinoma cell line with a missense mutation at codon 285 (GAG-»AAG) and NCI-HI703 is a squamous lung carcinoma cell with a single base deletion at codon 191 (CCT->CT) of p53 gene. Loss of heterozygosity of the p53 gene was found in both NCI-H522 and NCI-HI703 cells. We have demonstrated that the virus can lyse those cells but not NSCLC cells with functional p53 (NCI-H2304) or a mesothelioma cell line (MS-1). In replicate experiments, ONYX-015 virus lysed, in a dose-dependent fashion, NCI-H522 and NCI-HI703 cells. Five days after infection, 50% of the cells were lysed at multiplicities of infection (MOI) of 1 plaque-forming unit (PFU) per cell; 9 days after refection, at the MOI of 1 and 0.1, 100% and 50% of the cells were lysed respectively. No signs of cell lysis were noticed in NCI-H2304 and MS-1 at MOI of 0.1 and 1. PFU per cell 9 days after infection. Wild-Type adenovirus served as control for effective infection. We are currently in the process of testing this virus in combination with chemotherapy in these NSCLC cell lines as well as in fresh human lung tumor cells. It is hoped that with subsequent pre-clinical studies ONYX-015 can be advanced to phase I clinical trials for the treatment of patients with advanced lung cancer.

*By invitation

F18. MDM-2 EXPRESSION: AN ALTERNATIVE MECHANISM FOR p53 INACTIVATION IN ESOPHAGEAL ADENOCARCINOMA.

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

New York, New York

Several immunohistochemical studies have shown that the p53 protein is expressed in 60-70% of esophageal adenocarcinomas. Since mutations of this tumor suppressor gene are present in approximately 40% of tumors, it is presumed that p53 stabilization and expression may develop as a result of mechanisms other than gene mutation. Amplification and expression of

the mdm-2 gene, a known regulator of p53 activity, can result in inactivation of the p53 gene with stabilization of its protein product and loss of its tumor suppressor function.

In this study we evaluated the incidence of p53 abnormalities as well as the expression of the mdm-2 protein product in 16 esophageal adenocarcinomas. Routine immunohistochemical studies were performed following standard antigen retrieval methods and interpreted using a previously described immunoreactive score. Tumor DNA was obtained by microdissection, amplified and sequenced for mutations in the p53 hot spot regions (exons 5-8). P53 expression was observed in 12 of 16 (75%) cases while p53 mutations were detected in 7 of 16 (43%). This does not exclude the possibility of mutations in exons other than 5-8. Overall, mdm-2 expression was present in 8 of 16 tumors. Moderate or strong expression of mdm-2 was observed in 4 tumors none of which had detectable p53 mutations.

We conclude that in esophageal adenocarcinoma (1.) Discordance of p53 immunohistochemistry and mutations occurs in 20-30% of cases and (2.) mdm-2 expression may be responsible for loss of p53 tumor suppressor function in 25% of esophageal adenocarcinomas.

*By invitation

7:00 a.m. CARDIAC SURGERY FORUM SESSION

Ballroom B, Hynes Convention Center

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

Edward D. Verrier, M.D.

F19. AN EXPERIMENTAL MODEL OF SMALL INTESTINE SUBMUCOSA AS A GROWING VASCULAR GRAFT FOR CONGENITAL CARDIAC SURGERY.

†MonicaC. Robotin-Johnson, M.D., F.R.A.C.S.*, Paul E. Swanson, M.D.*, David C. Johnson, M.D., F.R.A.C.S.* and James L. Cox, M.D.

St. Louis, Missouri and Washington, DC

The ideal vascular graft for use in children with congenital heart defects should not only be biocompatible, nonthrombogenic and present no infectious risk, but should grow at the same rate as the recipient.

We have tested autologous small intestine submucosa (SIS) as a superior vena cava (SVC) interposition graft in 11 piglets, with a mean weight of 10.7 kg. The grafts were prepared from segments of autologous jejunum, rendered nonthrombogenic by bonding with heparin. The SVC from the level of the azygous vein to the SVC-right atrial junction, measuring a mean length of 9.9 mm and circumference of 25.9 mm was replaced. There was one early and one late death, not related to the SVC replacement. At 90 days the 9 long term survivors had a mean weight increase of 630% and the grafts increased in length by 147% [$p<0.03$] and in circumference by 184% [$p<0.001$], paralleling the growth of the native cava. All 11 grafts were patent and free of thrombus at the time of explantation. Cavograms showed no anastomotic stricture or aneurysm formation in 7 of 9 cases. The luminal surface of all grafts was smooth, shiny and indistinguishable from that of the native cava. The light microscopy showed a loosely textured cellular collagen framework, with a dense capillary network and complete luminal coverage by cells resembling endothelial cells. Electron microscopy confirmed that a complete endothelial cell layer was present.

In conclusion, SIS provides a collagen framework that becomes remodelled, keeps up with the growth of the recipient and acquires a nonthrombogenic endothelial surface. This makes it

potentially very well suited as material for pulmonary artery reconstruction or Fontan operations in small children.

†1996-97 AATS Graham Fellow

*By invitation

F20. MEMANTIN IS A POTENT EXCITATORY AMINO ACID BLOCKER FOR PREVENTING SPINAL CORD INJURY IN A RABBIT MODEL.

Marek P. Ehrlich, M.D.*, Erich Knolle, M.D.*, Ruxandra Ciovica, M.D.*, Peter Boeck, M.D.*, Martin Grubenwoger, M.D.*, Ricarda Konetschny, M.D.*, Fabiola Cartews-Zumelzu, M.D.*, Edvin R. Turkof, M.D.*, Ernst Wolner, M.D. and Michael Havel, M.D.*

Vienna, Austria

Introduction: This study was conducted to investigate the effect of memantin, a noncompetitive N-methyl-D-aspartate receptor antagonist, on the neurological outcome of spinal cord ischemia and reperfusion after aortic occlusion.

Materials and methods: New Zealand white rabbits (3 - 4.5 kg) were anesthetized and spinal cord ischemia was then induced for 40 minutes by infrarenal aortic occlusion. Animals were randomly assigned into three groups. Group A animals (n = 8) received intraaortic memantin infusion (20 mg/kg) after aortic clamping. Group B animals (n = 8) were pretreated with memantin infusion (20 mg/kg) 45 minutes prior to aortic occlusion. Group C (n = 8) was the control group, in which no pharmacological intervention was applied. Neurological status was assessed at 12, 24, 36 and 48 hours after operation and scored using the Tarlov system (4 normal, 0 paraplegia). Lumbar spinal root stimulation potentials (SRS) as well as electrical transcranial motor evoked potentials (MEP) from lower limb muscles were monitored pre-, intra- and postoperatively. After sacrifice at 48 hours, the spinal cord was fixed and studied histopathologically.

Results: All measured potentials disappeared shortly after aortic cross-clamping. MEP did not correlate with clinical findings. Quantitative analysis of SRS showed, that potentials in both memantin treated groups regained activity earlier compared to the placebo group. Histologic examination of spinal cords from group A and B rabbits revealed only a few abnormal motorneurons, whereas spinal cords from the control group had evidence of extensive cord injury with prominent lysis of Nissl substance, destruction of nuclear chromatin and vacuolization of anterior horn motorneurons. Median values for neurological observations from each group are reported below:

Variable	12 Hours	24 Hours	36 Hours	48 Hours
Control (n= 8)	0	0	0	0
Intraaortal(n = 8)	2	2.5	3	3
Systemic (n= 8)	3	4	4	4
control vs. intraaortal	P = 0.001	P = 0.0002	P = 0.0006	P = 0.0006
control vs. systemic	P = 0.00022	P = 0.0002	P = 0.0002	P = 0.0002

Conclusion: Memantin significantly reduced neurological injury secondary to spinal cord ischemia and reperfusion after aortic occlusion at 40 minutes in the rabbit model.

F21. MODULATION OF MYOCARDIAL PERFUSION AND VASCULAR REACTIVITY BY PERICARDIAL bFGF: IMPLICATIONS IN THE TREATMENT OF INOPERABLE CORONARY ARTERY DISEASE.

Roger J. Laham, M.D.*, Motohisa Tofukuji, M.D., Ph.D.*, Michael Simons, M.D.* and Frank W. Sellke, M.D.

Boston, Massachusetts

Endothelium-dependent relaxation and perfusion are reduced in the collateral-dependant myocardium. To examine the modulating effects of pericardial fluid basic-fibroblast growth factor (bFGF) on endothelium-dependent responses and expression of inducible (iNOS) and endothelial (eNOS) nitric oxide synthases in the collateral-dependant myocardium, ameroid occluders were placed around the left circumflex (LCx) artery of pigs. After 3 weeks, LCx occlusion was confirmed with angiography. Saline containing 30 μg (n = 6) or 2 mg (n = 6) bFGF or no bFGF (control, n = 6) was injected into the pericardial space. Four weeks later myocardial blood flow was determined with colored microspheres, endothelium-dependent coronary microvascular responses to ADP (10 μM) and endothelium-independent responses to nipride (100 μM) were examined, and protein and gene expressions of iNOS and eNOS were determined (Western analysis, PCR) in the ischemic LCx and non-ischemic LAD regions. Vascular response = % relaxation of precontract diameter.

Group	**ADP**	**Nipride**	**Blood Flow**	(ml/min/g)
LAD-control	60 ± 6*	82 ± 6	1.7 ± 0.2*	*p<0.05 vs LCx-control
LCx-control	25 ± 7	82 ± 4	0.9 ± 0.1	(ANOVA, Scheffe's test)
LCx-bFGF-30ug	44 ± 6*	86 ± 4	1.2 ± 0.1*	
LCx-bFGF-2mg	70 ± 7*	82 ± 3	1.3 ± 0.1*	

Both iNOS protein and mRNA were increased 3.3 ± 1 fold in the LCx compared to the LAD territory, whereas eNOS expression was similar in both regions. This suggests that the decreased endothelium-dependent relaxation in the collateral-dependent circulation may be due to increased iNOS expression and NO-induced inhibition of eNOS. bFGF improves endothelium- dependent relaxation and blood flow in the collateral-dependent myocardium. These findings may have implication regarding the cause of altered blood flow regulation in chronically ischemic myocardium and in the treatment of patients with inoperable coronary disease with the injection of bFGF into the pericardial space.

*By invitation

F22. ANGIOGENESIS ACCOMPANIES IMPROVED PERFUSION IN REGIONS OF HIBERNATING MYOCARDIUM FOLLOWING TRANSMYOCARDIAL LASER REVASCULARIZATION.

G. Chad Hughes, M.D.*, Alan P. Kypson, M.D.*, James D. St. Louis, M.D.*, Brian H. Annex, M.D.*, Timothy R. DeGrado, Ph.D.*, R. Edward Coleman, M.D.*, James E. Lowe, M.D. and Kevin P. Landolfo, M.D.*

Durham, North Carolina

Background. The mechanism for clinical improvement following transmyocardial laser revascularization (TMR) is unknown, although preliminary work in normal myocardium suggests

that angiogenesis may play a role. The purpose of this study was to describe the quantity and nature of the neovascularization accompanying improved myocardial perfusion following TMR in a model of hibernating myocardium.

Methods. Five adult mini-swine (n = 5) underwent left circumflex coronary artery (LCx) occlusion to reduce resting blood flow by 90% as documented by ultrasonic flow probe measurement. Two weeks post-occlusion, the animals underwent positron emission tomography (PET) to document the presence of ischemic, viable myocardium in the LCx distribution. TMR was then performed using a holmium:YAG (n = 3) or CO_2 (n = 2) laser with 20 channels placed at 1 cm intervals in the ischemic region. Six months post-TMR, repeat PET was performed. Animals were then sacrificed, and their hearts harvested for histology as well as immunohistochemical analysis to identify the presence of endothelial cells.

Results. Mean myocardial blood flow by PET in the ischemic LCx distribution increased from 0.37 ± 0.16 to 0.60 ± 0.13 ml/g/min ($p<0.05$) at 6 months post-TMR. There was no change in control septal flow from pre- to post-TMR (0.73 ± 0.38 to 0.64 ± 0.15 ml/g/min). Histologic examination of the lased LCx area revealed many neovessels located predominantly at the periphery of the TMR channels. These vessels were present in a highly disorganized pattern consistent with angiogenesis. The presence of endothelial ceils within the neovessels was confirmed with the endothelial cell specific antibodies anti-von Willebrand factor and anti-human tie-2 (TEK). Quantitative analysis revealed the lased ischemic regions contained a mean of 26.0 ± 8.8 microvessels per 200x field, compared with a mean of 5.1 ± 2.0 in control non-lased regions ($p = 0.0003$).

Conclusions. Angiogenesis occurs in the channel regions following TMR in hibernating myocardium and accompanies improved regional perfusion. These findings strongly suggest that angiogenesis is the mechanism of increased blood flow following TMR in ischemic myocardium.

*By invitation

F23. TIME-COURSE OF FUNCTIONAL RECOVERY AFTER CORONARY BYPASS SURGERY IN PATIENTS WITH CHRONIC LEFT VENTRICULAR ISCHEMIC DYSFUNCTION.

Jean-Louis J. Vanoverschelde, M.D., Ph.D.*, Christophe Depre, M.D., Ph.D.*, Bernhard L. Gerber, M.D.*, Marcel Borgers, Ph.D.*, William Wijns, M.D.*, Jacques A. Melin, M.D., Ph.D.* and Robert A. Dion, M.D.*

Brussels, Belgium

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

Background Chronic left ventricular (LV) ischemic dysfunction, a condition often referred to as myocardial hibernation, is associated in humans with ultrastructural alterations of the myocytes, including the loss of myofilarnants and the accumulation of glycogen. Given the severity of these structural changes, contractile function is unlikely to resume immediately upon revascularization.

Methods and results We studied 32 patients with coronary disease and chronic LV ischemic dysfunction undergoing bypass surgery. Dynamic Positron Emission Tomography with ^{13}N-ammonia and ^{18}F-deoxyglucose to assess myocardial perfusion and glucose metabolism was performed in 29 patients. Coronary bypass surgery was subsequently performed with the use of the left internal mammary artery to graft the left anterior descending coronary artery. All other co-diseased vessels were also revascularized. On average, each patient received 2.9 anastomoses, of which 1.3 were constructed with arterial conduits. In every patient, a peroperative transmural

biopsy was harvested from the center of the dysfunctional area, to quantify the increase in extracellular matrix and the presence of structurally altered cardiomyocytes. LV function was serially measured by digitized 2D-echocardiography before and again 10 days, 2 months and 6 months after revascularization. The time-course of recovery of regional function was estimated from the monoexponential decrease in dysfunctional wall motion score. At the 6 months followup, 19 patients had improved LV function while 13 patients showed persistent dysfunction. Before revascularization, reversibly dysfunctional segments had higher myocardial blood flow (83 ± 29 versus 60 ±21 $ml \cdot (min \cdot 100g)^{-1}$, $p<0.01$), higher glucose uptake (40 ±14 versus 21 ±9 $\mu mol \cdot (min \cdot 100g)^{-1}$, $p<0.05$) and less increase in extracellular matrix (25 ± 17 versus 46 ± 17%, $p = 0.01$) than segments with persistent dysfunction. The extent of functional recovery correlated with myocardial blood flow ($r = 0.84$) and the increase in extracellular matrix ($r = -0.60$). In patients with reversible dysfunction, the return of segmental function was progressive and followed a monoexponential time-course with a time constant of 23 days (range 6-78 days). The rate of recovery correlated best with the proportion of altered cardiomyocytes in the biopsy ($r = 0.83$).

Conclusions. The present study indicates that the recovery of regional and global LV function after successful revascularization is progressive and follows a monoexponential time-course which is influenced by the extent and severity of the structural changes affecting the cardiomyocytes.

*By invitation

F24. MYOCYTE ENDOTHELIN EXPOSURE DURING CARDIOPLEGIC ARREST EXACERBATES CONTRACTILE DYSFUNCTION WITH REPERFUSION.

R. Brent New, M.D.*, Jeffrey S. Mandel, M.D.*, Angela C. Sampson, B.A.*, Christopher A. Kerr, B.S.*, Rupak Mukherjee, Ph.D.*, Fred A. Crawford, Jr., M.D. and Francis G. Spinale, M.D., Ph.D.*

Charleston, South Carolina

Background: While transient left ventricular (LV) dysfunction can occur following cardioplegic arrest (CA), the contributory mechanisms for this phenomenon remain incompletely understood. Institution of cardiopulmonary bypass and CA results in neurohormonal system activation which includes increased release of the vasoactive peptide, endothelin (ET). Past studies have suggested that ET can influence LV myocyte contractility. Therefore, this project tested the hypothesis that exposure of LV myocytes to ET during simulated CA, would have direct effects on contractile processes with subsequent reperfusion.

Methods: LV porcine myocytes were randomly assigned to 3 groups: (1) Control: Normothermic (37°C) cell media (n=156); (2) Cardioplegia: simulated CA (K^+ 24 mEq/L, 4°C x 2hrs) followed by reperfusion and rewarming with cell media (n = 73); (3) Cardioplegia and ET: simulated CA in the presence of ET (200 pM) followed by reperfusion with cell media and ET (n = 54). Myocyte contractility was measured following reperfusion by videomicroscopy.

Results: Myocyte shortening velocity was reduced following simulated CA compared to controls (62 ±3 vs 80 ± 3 μm/s, respectively $p<0.05$) and was further reduced with CA and ET exposure (49 ± 3 μm/s, $p<0.05$). Myocyte velocity of relengthening, which reflects sarcomere cross-bridge release rates and calcium resequestration, was reduced after CA compared to controls (51 ± 3 vs 77 ± 3 μm/s, respectively, $p<0.05$) and was reduced to a greater degree with CA and ET exposure (41 ± 3 μm/s, $p<0.05$).

Conclusions: The unique findings of the present study demonstrated that exposure of the LV myocyte to ET during simulated CA, directly contributed to contractile dysfunction following reperfusion. A contributory molecular mechanism for the effect of ET with CA may be alterations in myocyte active relaxation processes. These findings suggest that increased ET levels which occur in the setting of cardiac surgery directly influence myocyte contractility, which in turn contributes to the transient LV dysfunction following cardioplegic arrest.

*By invitation

F25. ROLE OF ENDOTHELIN-1 AND ENDOTHELIN-1 RECEPTOR BLOCKADE IN PLACENTAL DYSFUNCTION AFTER FETAL CARDIAC BYPASS.

Doff B. McElhinney, M.D.*, V. Mohan Reddy, M.D.*, †Hiranya A. Rajasinghe, M.D.*, John R. Liddicoat, M.D.*, Karen Hendricks-Munoz, M.D.*, Jeffrey R. Fineman, M.D.* and Frank L. Hanley, M.D.

San Francisco, California

Fetal cardiac bypass (FCB) causes placental dysfunction, characterized by increased placental vascular resistance (PVR), decreased placental blood flow (PBF), hypoxia, and acidosis. A variety of factors have been found to contribute to the development of this placental dysfu nction, but its exact mechanisms remain unclear. Vasoactive factors produced by the vascular endothelium, such as nitric oxide and endothelin-1 (ET-1), are important regulators of placental vascular tone. To study the role of the vascular endothelium in placental dysfunction related to FCB, we studied 3 groups of fetal sheep. In the first (n:7), we determined placental hemodynamic responses pre- and post-FCB to an endothelium-dependent vasodilator (acetylcholine), an endothelium-independent vasodilator (sodium nitroprusside), and ET-1, a vasoactive polypeptide produced by the endothelium. Controls (n = 7) received the same vasoactive substances but were not exposed to FCB. In the second group (n = 5), an ET-1 receptor blocker (PDQ123) was administered and placental hemodynamics were measured before and after FCB. Results were compared with control fetuses that did not receive PDQ123 in order to assess the effect of ET-1 receptor blockade. In the third group (n = 5), no medications were given and plasma ET-1 levels were measured before and after FCB, then compared with controls that did not undergo FCB. Before FCB, exogenous ET-1 decreased PBF by 8.7% and increased PVR by 9.3%. After bypass, however, ET-1 decreased PBF by 47% and increased PVR by t06%. In addition, there was a significant attenuation of the placental vascular relaxation response to acetylcholine after FCB, with a decrease in PVR of 14%, compared with 20% pre-FCB. The response to sodium nitroprusside was not significantly altered by FCB. In fetuses that received the ET-1 blocker, PVR increased from 0.32 ± 0.03 U pre-FCB to 0.41 ± 0.07 after bypass, which was significantly less than in control animals (0.31 ± 0.04 pre-FCB, 0.51 ± 0.14 post-FCB). Similarly, PBF decreased significantly more in control animals (from 172 ± 29 ml/min/kg to 116 ± 36) than in fetuses receiving ET-1 receptor blocker (from 168 ± 31 to 140 ± 35). Plasma ET-1 increased significantly in fetuses exposed to FCB, but did not change in control animals. This study demonstrates that FCB causes a significant increase in plasma ET-1 and impairs the placental vascular response to endothelium-dependent modulators of vascular tone. ET-1 receptor blockade substantially reduces post-FCB placental dysfunction. This and other pharmacologic or physiologic interventions aimed preserving endothelial function may be effective means of optimizing fetal outcome after FCB.

†1995-97 TSFRE Research Fellow

*By invitation

F26. PHOSPHODIESTERASE INHIBITORS PREVENT THE SYSTEMIC INFLAMMATORY RESPONSE SYNDROME.

Koh Takeuchi, M.D.*, Pedro J. del Nido, M.D, Dimitrios N. Poutias, B.S.* and Francis X. McGowan, Jr., M.D.*

Boston, Massachusetts and Bethesda, Maryland

The systemic inflammatory response syndrome (SIRS) is an important cause of multiple organ dysfunction after cardiopulmonary bypass; its magnitude and consequences are particularly great in neonates and infants. We and others have previously found that certain phosphodiesterase inhibitors (PDI) interfere with inflammatory signaling pathways at several points. The present study tested the hypothesis that two clinically used PDIs, amrinone (AMR) and vesnarinone (VES), would decrease the SIRS.

A well-characterized, severe SIRS model of rabbit endotoxemia was used. Rabbits received *S. lyphi* endotoxin (lipopolysaccharide, LPS; 0.2 mg/kg iv bolus); LPS + AMR (1.0 mg/kg bolus + 10 mg/kg/min infusion); LPS + VES (3 mg/kg bolus only-t½ =~8 hours); or vehicle alone. Systemic effects were assessed by mortality, fever, and acidosis. Indices of the inflammatory response included plasma cytokine and myeloperoxidase (MPO) concentrations. Pulmonary involvement was assessed by changes in pulmonary vascular resistance (PVR) and lung MPO. Myocardial function was quantified in excised, Langendorff-perfused hearts; myocardial calcium cycling and contractile protein calcium sensitivity were measured using fluorescence spectroscopy. Plasma hepatocellular enzyme concentrations (SGPT, SGOT) served as markers of liver injury. Myocardial protein kinase C (PKC) and inducible nitric oxide synthase (iNOS) activity were used as indices of cytokine signaling. Measurements were made 0, 1, 2, and 6 hours after LPS. Results are summarized in the Table as mean ± sem, N = 6-8 each. *P<0.05 vs control; #P<0.05 vs LPS alone.

	Mortality (%)	Tmax	HCO_3-	MPO	TNF	iNOS	PDF
Control	0	37 ± 0.6	21 ± 3	155 ± 21	< 10	< 25	88 ± 7
LPS	60*	39 ± 0.2*	10 ± 1*	350 ± 55*	480 ± 30*	140 ± 30*	57 ± 8*
LPS+AMR	20	40 ± 0.6*	14 ±3*	220 ± 40#	210± 25#	60 ± 15#	97 ± 12#
LPS+VES	9#	38 ± 0.1#	18±3#	200 ± 30#	50 ± 10#	30 ± 5#	92 ± 7#

Tmax (^{0}C; HCO_3^-, MPO, U/ml; TNF, pg/ml; iNOS, pmol/hr/mg protein; POP, peak developed LV pressure, mmHg

Thus, VES, which has unique ion channel and gene expression effects unrelated to its PDI properties, was particularly effective in SIRS suppression. VES also protected against LPS-induced increases in cytokines, hepatocellular enzymes, myocardial PKC activity, and PVR; the protective effects of AMR upon these indices were significant, but less than VES. VES prevented LPS-induced reductions in myocardial calcium-activated contractile force, diastolic relaxation, and diastolic calcium removal. Neither VES nor AMR exacerbated LPS-induced hypotension. VES also prevented LPS-induced TNF production in cultured macrophages and cytokine-stimulated iNOS production in cultured neonatal cardiomyocytes. The beneficial effects of VIES and AMR in vivo were not shared by dobutamine, and the in vitro effects of VES were not mimicked by a cell-permeable cyclic AMP analog. These results indicate that certain phosphodiesterase inhibitors have multiple and potent effects upon inflammatory activation and signaling pathways. The mechanism does not appear to be due to elevations in cyclic AMP. These compounds, which are used clinically

for their inotropic and vasodilating properties, may be useful to limit inflammatory activation and signaling cascades during pediatric CPB, as well as other states that are associated with inflammatory cytokine production.

*By invitation

F27. IN VIVO IMAGING OF APOPTOSIS DURING CARDIAC ALLOGRAFT REJECTION USING RADIOLABELED ANNEXIN V.

Patrick W. Vriens, M.D.*, Francis G. Blankenberg, M.D.*, Eric R. Davis, M.D.*, Gerald J. Berry, M.D.*, Bruce A. Reitz, M.D., H. William Strauss, M.D.* and Robert C. Robbins, M.D.*

Stanford, California

Introduction We hypothesized that technetium 99m labeled annexin V, a new radioactive tracer of apoptotic cell death, can be utilized to monitor cardiac allograft rejection. Annexin V is a human protein that binds to phosphatidyl serine, a phospholipid that is selectively expressed on the cell surface, during the early stage of apoptosis.

Methods Untreated ACI rats served as recipients of heterotopically placed, allogeneic PVG rat, or syngeneic ACI rat cardiac grafts. Function of grafts was assessed by daily palpation. Annexin V was derivatized with technetium 99m hydrazinonicotinamide (99mTcHYNIC), and injected i.v. one hour prior to imaging at day 3, 4, and 5 after transplantation. Region of interest image analysis was used to quantify uptake of the radiopharmaceutical. Histopathologic analysis and TUNEL staining were performed at each time point after imaging. To investigate whether uptake of 99mTcHYNIC-annexin V decreased after treatment of rejection, recipients of allogeneic grafts were treated daily with Cyclosporin A (CSA, oral, 10 mg/kg), starting at day 4, and imaged at day 4, 7, and 11 after transplantation.

Results Allogeneic PVG cardiac grafts showed a readily visualizable 50% increase in uptake of 99mTcHYNIC-annexin V at day 3, a 160% increase at day 4 and a 274% increase at day 5 after transplantation in untreated ACI rats (Fig. 1) (n = 6 for each time point), compared to syngeneic hearts (Fig. 2) (n = 3 for each time point) ($P<0.01$). Histopathology showed grade 1, grade 1.5 and grade 2.3 rejection at these time points, respectively. Apoptotic nuclei were identified by TUNEL staining in myocytes and mononuclear infiltrates of rejecting allogeneic grafts, but not of syngeneie grafts. Palpable contractions of allogeneic grafts ceased at day 7, indicating complete rejection (n = 6). When CSA treatment was started at day 4, rejection could be reversed, and allogeneic grafts remained functional. Uptake of 99mTcHYNIC-annexin V in treated animals decreased to 62% at day 7 and 0% at day 11 (n = 6).

Conclusion Uptake of radiolabeled annexin V correlates with histopathological grades of acute rejection in cardiac allografts. When rejection is reversed by CSA treatment, uptake decreases to levels comparable to syngeneie grafts. Imaging of apoptosis using radiolabeled annexin V may enable detection of acute cardiac allograft rejection, and may provide a new tool to monitor rejection.

*By invitation

F28. SKELETAL MUSCLE VENTRICLES FROM LEFT VENTRICULAR APEX TO AORTA, EXPERIENCE UP TO 37WEEKS: STEP TOWARDS CLINICAL APPLICATION.

Frank A. Baciewicz, Jr., M.D.*, Gregory A. Thomas, M.D.*, Kevin A. Greer, M.D.*, Robert L. Hammond, Ph.D.*, Huiren Lu, M.D.*, Steven Bastian, M.S.* and Larry W. Stephenson, M.D.

Detroit, Michigan

Skeletal muscle ventricles (SMVs) were constructed from the latissimus muscle and lined with autogenous pericardium in 6 dogs. After 3 weeks of vascular delay and 6 weeks of electrical conditioning, SMVs were connected from the left ventricular apex with a valved conduit and then from the SMV to the descending aorta with a second-valved conduit. The SMV was stimulated during diastole at a 1:2 ratio with the heart. SMV contraction increased femoral pressure by 23% at 33 Hz and 28% at 50 Hz, resulting in 32% and 33% of the total cardiac output being pumped by the SMV. Data at implant (n = 6) is shown below:

	FAP_{aug}	FAP_{dia}	CAP_{aug}	CAP_{dia}	Q_{smv}	Q_{AV}	Q_{total}
OFF	59 ± 4	51 ± 5	62 ± 3	55 ± 3	604 ± 173	2976±434	3580 ± 556
33Hz	73 ± 8*	61 ± 5*	72 ± 5*	61 ± 5*	1147 ± 223*	2395 ±319*	3542 ± 454
50Hz	76 ± 7*	62 ± 5*	75 ± 4*	67 ± 4*	1162 ± 212*	2247 ± 308*	3454 ± 408

(FAP & CAP. fenorai and carotid arterial pressure, aug = augmented, dia = diastolic, Q_{SMV}: flow through S_{MV}, Q_{AV} flow through aortic valve, $Q_{total} = Q_{smv} + Q_{AV}$,* = P<0.05 by ANOVA).

The dogs survived 6, 17, 30, 72 and 263 days in circulation; one dog is alive at 160 days. Three deaths, including that at 263 days were related to complications of indwelling monitoring devices and not directly to the SMV. In the two longest surviving dogs, SMV function remained stable over time. During propranolol induced heart failure, SMV contraction augmented diastolic pressure 89% at 33Hz and 96% at 50 Hz. At a 1:2 contraction ratio with the heart, SMV assist increased systemic perfusion (Q_{smv} Q_{AV}) 23% at 33 Hz and 64% at 50 Hz. At a 1:1 contraction ratio, systemic perfusion was increased further by 25% at 33 Hz and 114% at 50Hz. This model of skeletal muscle assist is the most hemodynamically effective that we have tested, and now appears capable of functioning long-term.

*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION A-2 CONGENITAL HEART DISEASE

Ballroom A, Hynes Convention Center

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

John A. Waldhausen, M.D.

40. VENO-VENOUS MODIFIED ULTRAFILTRATION AND CIRCULATING CYTOKINES: A PROSPECTIVE RANDOMIZED STUDY.

Ugursay Kiziltepe, M.D.*, Azhar Hossain, M.D.*, Daniel Remick, M.D.*, Samuel Barst, M.D.*, Jeffrey P. Gold, M.D. and Hani A. Hennein, M.D.*

Atlanta, Georgia; New York, New Hyde Park and Bronx, New York; Ann Arbor, Michigan

Discussant: J. William Gaynor, M.D., Philadelphia, Pennsylvania

Background: Cardiopulmonary bypass (CPB) is associated with the production of both proinflammatory (IL-6, IL-8 and TNFcc) and antiinflammatory (IL-10) cytokines, and a resultant systemic inflammatory response. Arterio-venous modified ultrafiltration has been shown to be associated with a reduction in total body water and improved hemodynamic and hemostatic functions. Veno-venous modified ultrafiltration (VVMUF) is a further modification of the technique with the potential advantage of not reducing effective cardiac output. We tested the hypothesis that VVMUF reduces extracellular body water and removes circulating cytokines following CPB.

Methods: Thirty-seven children undergoing open surgical correction of congenital heart defects were randomly assigned to VVMUF or controls. Inferior and superior vena cava cannulas were used as the inflow and outflow of the VVMUF circuit. VVMUF was performed for 10 min after weaning from CPB. IL-1P, IL-6, IL-8, IL-10 and TNF-a plasma levels measured at seven time points before, during and after CPB.

Results: Both groups were similar in terms of age (6.48 ± 5.38 years in VVMUF vs. 6.27 ± 5.37 years in control, p: not significant (n.s.)). Mean cross-clamp times were 604-35 minutes (rain) in VVMUF and 41 ± 23 rain in control group, p:n.s. Mean bypass duration was 98 ± 56 min in VVMUF and 92 ± 50 min in control group, p:n.s. Minimum temperatures were 28.8 ±2.1 C in VVMUF and 28.4 ±4.4 C in control group, p:n.s. VVMUF resulted in a significant removal of extracellular fluid (980 ± 601 cc) and rise in hematocrit levels (from 28.27 ± 3.64% to 34 ± 3.64%, $p<0.05$). There was no mortality in either group. All 5 cytokines measured rose during and following bypass in both groups. A significant reduction of IL-lÎ² levels (from 11.3 ± 39.7 pg/ml to 4.4 ± 20.9 pg/ml) followed VVMUF. Changes in cytokines other than IL-lÎ² could not be demonstrated.

Conclusions: Veno-venous modified ultrafiltration is a safe and effective

method of removing extracellular volume following CPB. VVMUF is

associated with a significant reduction in IL-lÎ² levels and may therefore

reduce the deleterious effects of CPB by diminishing systemic inflammatory

response.

*By invitation

41. TOTAL REPAIR OF PULMONARY ATRESIA WITH VENTRICULAR SEPTAL DEFECT AND MAJOR AORTOPULMONARY COLLATERALS: AN INTEGRATED APPROACH.

Adriano Carotti, M.D.*, Roberto M. Di Donate, M.D.*, Cosimo Squitieri, M.D.*, Paolo Guccione, M.D.* and Glauco Catena, M.D.*

Rome, Italy

Sponsored by: Aldo R. Castaneda, M.D., Guatemala City, Guatemala

Discussant: Frank L. Hanley, M.D., San Francisco, California

Background: Predicting postrepair ratio of the peak systolic pressure in the right ventricle (pRV) to that in the left ventricle (pLV) may be of absolute prognostic value for patients undergoing total repair of pulmonary atresia, ventricular septal defect and major aortopulmonary collaterals (PA.VSD.MAPCAS). To this purpose, we currently rely on 2 novel parameters: 1) preoperative total neopulmonary index (TNPAI = CAI [total MAPCA index] + PAI [pulmonary artery index]); 2) mean pulmonary artery pressure changes during an intraoperative flow study, according to Reddv M. et al.

Patients and methods: Since January 1994, 15 patients (age mean±SD: 64 ± 54 months) with PA.VSD.MAPCAS were managed according to TNPAI: a preoperative value of ‰¥ 150 mm^2/m^2 was indication for total repair. Seven patients with hypoplastic pulmonary arteries and TNPAI < 150 mm^2/m^2 first underwent palliative conduit right ventricular outflow tract reconstruction (RVOTR), followed by secondary one-stage, midline total unifocalization and VSD closure. The other 8 patients with preoperative TNPAI > 150 mm^2/m^2 (absent pulmonary arteries m 2 cases) underwent single-stage unifocalization and repair. The VSD was closed in all cases. In 9, the decision to close the VSD was based on an intraoperative pulmonary flow study. In one case the VSD had to be reopened due to hypersystemic pRV.

Results: The 7 patients who initially underwent RVOTR had a significant increase of PAI (from 46 ± 26 to 194 ± 74 mm^2/m^2 $p<0.0001$) within 22 ± 6 months from the palliation. Of the total group of 15 patients, repair was successful in 14, with a postrepair pRV/pLV ratio of 0.47 ± 0.1. There was one hospital death due to hypersystemic pRV, despite a reassuring intraoperative pulmonary flow study. Accuracy of this test in predicting the postrepair mean pulmonary artery pressure was 89% (70% CL : 68 - 98%). At a mean follow-up of 12 ± 11 months, all patients arc in NYHA I, on no medications. Their mean pRV assessed by 2DD-ECHO is 40 ± 8 mmHg.

Conclusion: The integrated approach to total repair of PA.VSD.MAPCAS by preoperative calculation of TNPAI, RVOTR (when required) and intraoperative flow study, has led to optimal one-year results with low pRV/pLV ratios.

*By invitation

42. LONG-TERM OUTCOME OF INFANTS WITH SINGLE VENTRICLE AND TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION.

J. Williams Gaynor, M.D.*, Margaret H. Collins, M.D.*, Jack Rychik, M.D.* and Thomas L. Spray, M.D.

Philadelphia, Pennsylvania

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

Since 1984, 50 infants (28 male/22 female) with functional single ventricle (SV) and total anomalous pulmonary connection (TAPVC) have undergone palliative surgery at our institution. Heterotaxy syndrome was present in 33 patients and Hypoplastic Left Heart Syndrome (HLHS) in 9. TAPVC was supracardiac in 28 patients, cardiac in 7, infracardiac in 10 and mixed in 5. Obstructed TAPVC was present in 15 patients. Initial palliation (median

age 5 days, range 1 day-2.5 years) included aortopulmonary shunt (22), the Norwood procedure (17), cavopulmonary connection (5), pulmonary artery band (2), pulmonary valvotomy (1) and the Fontan procedure (1). Repair of TAPVC alone (2) or with other procedures was performed at the initial operation in 29 patients. Overall hospital mortality for initial palliation was 44% (22/50). Mortality was 59% (17/29) in patients undergoing TAPVC repair compared to 24% (5/21) in patients whom TAPVC repair was not performed, $p<0.02$. Sixteen of the 28 survivors have subsequently died and 1 has been lost to follow-up. TAPVC repair was performed at a subsequent operation in 3 patients. Only 8 patients have successfully undergone the Fontan procedure. Overall actuarial survival was 58 ± 7% at 6 months, 47 ± 7% at 1 year, 36 ± 7% at 2 years and 26 ± 7% at 5 years. Cardiac TAPVC was associated with improved survival compared to other forms of TAPVC (86 ± 13% vs. 42 ± 8% at 1 year and 64 ± 21% vs. 21 ± 6% at 5 years, $p = 0.01$). Actuarial survival for survivors of initial palliation was 81 ± 8% at 1 year, 6 ± 10% at 2 years, and 47 ± 10% at 5 years. By univariate analysis using Cox's proportional hazard model; repair of TAPVC, younger age at the time of initial surgery and non-cardiac TAPVC were predictors of mortality, $p<0.05$. Heterotaxy syndrome, HLHS, obstructed TAPVC, and performance of the Norwood procedure were not predictors of mortality, $p>0.1$. Time of TAPVC repair (initial vs. subsequent operation) did not alter survival, $p>0.1$. Lung tissue from 12 patients (median age 20 days, range 2 days-8.8 months) was available for histologic examination. TAPVC was supracardiac in 7 of these patients, infracardiac in 3 and mixed in 2. Obstructed TAPVC was present in only 3 patients. In all 12 patients the pulmonary veins were dilated with increased wall thickness for age. Increased muscularization of the arteries was present in 10 patients.

The long-term outcome for infants with SV and TAPVC is poor except for infants with cardiac TAPVC. Mortality at the initial palliative procedure is high and there is a continuing risk of late death. Repair of TAPVC is associated with a worse outcome. Development of the pulmonary vasculature, particularly the pulmonary veins, is abnormal even in infants without clinical evidence of obstructed TAPVC.

*By invitation

43. SURGICAL TREATMENT OF SUBAORTIC STENOSIS: A 17-YEAR EXPERIENCE.

†Alain Serraf, M.D.*, Joy Zoghby, M.D.*, FranÃ§ois Lacour-Gayet, M.D., Remi Houel, M.D.*, Emre Belli, M.D.*, Lorenzo Galletti, M.D.* and Claude Planche, M.D.

Le Plessis Robinson, France
Discussant: Michel N. Ilbawi, M.D., Chicago, Illinois

Left ventricular outflow tract (LVOT) obstruction due to subaortic stenosis (SAoS) covers a wide range of anatomic features from the localized discrete fibrous stenosis to the more complex multiple left-sided obstructive lesions. Several surgical techniques have been proposed to reestablish a widely patent LVOT, each of which being adapted to the anatomical form.

From 1980 to 1997, 160 pts with SAoS underwent surgical repair. Pts with isolated aortic valve (AoV) stenosis, supravalvar stenosis, those with VSD and SAoS or who developed SAoS after VSD closure were excluded. Preoperatively, 126 pts were in NYHA classes I-II and 34 in classes 1II-IV. Morphological and hemodynamic assessments were performed by angiography in 109 pts and echocardiography alone in 51. Localized discrete fibrous stenosis was present in 28 pts, fibromuscular stenosis in 90, localized hypertrophic obstructive cardiomyopathy in 12, diffuse subaortic stenosis in 23 and in 7 the SAoS was due to accessory mitral valve (MV) tissue or

abnormal insertion of mitral papillary muscle. AoV stenosis was associated in 24 and 16 had MV stenosis. In 13, there were multiple left-sided obstructive lesions (Shone's complex). Aortic coarctation was present in 25 pts. The mean preoperative peak systolic gradient across the LVOT was 80 ± 34.8 mmHg. Thirty two pts had previous surgery: 25 coarctation repair, 5 AoV commissurotomy and 2 repair of partial AV canal. The median age at repair was 10 years (Ranges: 0.1-30 years). Subaortic membrane excision was performed in 134 pts, associated to septal myotomy in 101, to septal myectomy in 24. Concomitant AoV commissurotomy was performed in 17 pts. Two had a Konno procedure, 5 had resection of abnormal MV tissue or papillary muscle and 2 had apical conduit insertion. AoV replacement was associated in 6 pts, MV replacement in 2 and mitral valvuloplasty in 4.

There were 5 early deaths (3.1%; 70% CL: 1.5-5.2%): in 2 Shone's complex, 2 diffuse SAoS and 1 with preoperative NYHA class IV. Permanent pace-maker insertion was mandatory in 5 pts. Twenty-five reoperations with 2 post operative deaths were performed in 19 pts in a mean delay of 67.1 ±55.7 months for recurrent LVOT obstruction in 18 and AoV endocarditis in 1. Ten of these pts initially presented with a diffuse SAoS. It was surgically addressed by new septal myectomy in 10, Ross procedure in 2, Konno operation in 2, an apical conduit in 3 and 2 had AoV replacements which was associated in 1 with a Manougian procedure. Statistical analysis revealed that diffuse SAoS with or without AoV stenosis was associated to higher risk for mortality and reoperation ($p = 0.002$). A median followup of 13.3 years (Ranges: 1.2-17.9 years) was achieved in 90% of survivors. There were 2 late deaths, all other patients were asymptomatic with a mean gradient across the LVOT of 20 ± 13 mmHg. Actuarial survival and freedom from reoperation rates at 15 years were 94.25 ± 1.34% and 85.3 ± 6%, respectively. In conclusion, SAoS covers a wide range of anatomical forms in which diffuse SAoS with or without AoV involvement carries the higher operative mortality and reoperation rates. A more aggressive initial operation should be performed in those patients.

10:50 a.m. INTERMISSION

†1993-94 AATS Graham Fellow

*By invitation

11:10 a.m. SIMULTANEOUS SCIENTIFIC SESSION A-2 CONGENITAL HEART DISEASE

Ballroom A, Hynes Convention Center

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

John A. Waldhausen, M.D.

44. LONG-TERM CLINICAL AND HEMODYNAMIC EVALUATION OF PORCINE VALVED CONDUITS IMPLANTED FROM THE RIGHT VENTRICLE TO THE PULMONARY ARTERY.

Gerard L. Champsaur, M.D., Jacques A. Robin, M.D.*, Francois Tronc, M.D.*, Alain Curtil, M.D.*, Catherine Vedrinne, M.D.*, Francois Sassolas, M.D.* and Jean Ninet, M.D.*

Lyon, France

Discussant: Ivan M. Rebeyka, M.D.,

Edmonton, Alberta, Canada

To evaluate the long-term results of valvular prosthetic conduits implanted as a new right ventricular outflow tract (RVOT), a retrospective study was conducted in a consecutive series of 127 patients presenting with complex ventriculo-pulmonary discontinuity and operated on between 1973 and 1996. The mean age was 5 ± 3.48 years (range 2 months-42 years). Initial pathology was tetralogy of Fallot in 42 cases, D-transposition of the great arteries in 25, truncus arteriosus in 21, double outlet right ventricle in 20, L-transposition of the great arteries in 18, and double outlet left ventricle in 1 case. Conduit sizes ranged from 14 to 25 mm, with a 20 mm size used in 38.5% (70% CL: 31-45%) and a 22 mm size used in 22.04% (70% CL: 14-29%) of the cases. Early mortality rate was 19% (24 patients). A post-operative evaluation was performed in 103 operative survivors who were followed up from 1 to 21.6 years, with a mean followup of 8.38 ± 6.2 years. The followup was over 10 years in 33 patients, (32%, 70% CL: 24-40%) and over 15 years in 28 (27%, 70% CL: 19-35%). There were 16 late deaths (RV failure and/or pulmonary hypertension in 7 cases, at various types of redo surgery in 3, sudden death in 1, acute conduit obstruction in 1, neurological in 1, infectious in 1, and miscellaneous in 2). The actuarial survival, including early mortality, was 72.91 ± 0.04% at 5 years, 63.16 ± 0.05% at 10 years, and 58.17 ± 0.05% at 15 years, where 20 patients were still exposed. A total of 74 hemodynamic studies were performed post-operatively, 50 patients having undergone at least one cardiac catheterization during their followup period. The mean peak systolic gradient across the RVOT was plotted as a function of time showing a gradual increase and a significant step-up after the 8th year.

Post-operative year	**0-4**	**4-8**	**8-12**	**>12**
Peak systolic gradient, mmHg	22 ± 20*	43 ± 36	69 ± 19*	73 ± 8*

*p < 0.05, ANOVA

Reoperation was required in 25 cases (24%, 70% CL: 15-33%) for progressive conduit obstruction between 1.1 and 17.7 years after implantation (mean 7.4 ± 4.8 years) in patients with generally very few symptoms or residual VSD in 3 cases. Conduits were replaced by either a RVOT patch (10 cases), a new porcine conduit (7 cases) or a non-valved conduit (5 cases). Freedom from reoperation was 79.52 ± 0.05% at 10 years and 65.81 ± 0.07% at 15 years. More readily available, porcine conduits may represent a valuable alternative to biological substitutes with similar long-term results in large (18 to 20 mm) sizes. Given the few symptoms, progressive conduit stenosis after the 8th postoperative year imposes a yearly noninvasive evaluation during their followup.

*By invitation

45. SURGICAL MANAGEMENT OF PROGRESSIVE PULMONARY VENOUS OBSTRUCTION FOLLOWING REPAIR OF TOTAL ANOMALOUS PULMONARY VENOUS DRAINAGE.

FranÃ§ois Lacour-Gayet, M.D., Joy Zoghby, M.D.*, †Alain Serraf, M.D.*, Emre Belli, M.D.*, Lorenzo Galletti, M.D.*, Jacqueline Bruniaux, M.D.* and Claude PlanchÃ©, M.D.

Le Plessis Robinson, France

Discussant: Thomas L. Spray, M.D., Philadelphia, Pennsylvania

Out of 158 patients who underwent correction of total anomalous pulmonary venous drainage (TAPVD) over the past fifteen years, 13 patients developed a progressive pulmonary venous (PV) obstruction. All included patients had satisfactory initial repair, documented by postoperative echo-Doppler, with biphasic flow and maximal velocity less than 1.5 m/s. The anatomical types were: 7 supra cardiac, 4 infra cardiac, 1 intra cardiac and 1 miscellaneous. At

initial correction, the common pulmonary vein trunk was anastomosed to the left atrium using nonresorbable suture. A progressive pulmonary venous obstruction occurred in a mean interval of 4 months ranging from 2 weeks to 12 years. Eleven patients required 15 reoperations; 1 patient is awaiting surgery and the last patient was judged inoperable. Two different anatomic lesions were frequently associated: - an anastomotic stenosis with inflammatory retraction of the suture line and - a PV ostial stenosis made of inflammatory intimal hyperplasia developing along the extra cardiac segment of the PV, involving one or several PV on either side. Two patients had isolated anastomotic stenosis, 3 patients had isolated PV ostial stenosis and 8 patients had both. PV ostial stenosis involved: 4 veins in 2 pts, 3 veins in 3 pts, 2 veins in 4 pts, 1 vein in 2 pts; the lesions were bilateral in 7 pts and unilateral in 4. The reoperations consisted in: 10 patch enlargement of the anastomotic stenosis, 5 patch enlargement of PV, 1 intra-operative stenting of PV and 6 intra pericardia! tunnelisation of PV. This last technique consisted in resection of the right pulmonary vein stenosis. The proximal normal PV are incised up to the pericardial reflection. The PV drain passively into the left atrium through a pericardial pouch made of a vascularized pericardial flap. No suture material is used on the PV walls. A similar technique is used on the left PV. There were 5 deaths (38%), all occurring in patients with bilateral PV stenosis. There was no death among the patients with isolated anastomotic stenosis. In the 11 patients who had PV ostial stenosis, 4 of the 6 survivors were successfully treated by the intra pericardial tunnel technique with normal pulmonary artery pressure and no residual gradient at a mean follow up of 15 months.

Conclusion: progressive PV stenosis following TAPVD repair is generally considered as an unpredictable and often lethal complication. It has been successfully treated in 4 patients in this series, using passive drainage of the pulmonary veins into the left atrium through an intra pericardial vascularized tunnel.

†1993-94 AATS Graham Fellow

*By invitation

46. SUPERIOR OUTCOME FOLLOWING SURGERY FOR PULMONARY ATRESIA AND INTACT VENTRICULAR SEPTUM.

Jack Rychik, M.D.*, Hara Levy, M.D.*, J. William Gaynor, M.D.*, William M. DeCampli, M.D.* and Thomas L. Spray, M.D.

Philadelphia, Pennsylvania

Discussant: Mohan V. Reddy, M.D., San Francisco, California

Pulmonary atresia and intact ventricular septum (PA/IVS) is an anatomically heterogeneous anomaly with a variety of possible surgical management strategies. In a recent multi-institutional study, survival was found to be only 64% at 48 months. Since 1981, 67 patients with PA/IVS have had surgery at our center with 51 survivors (median followup 51 months, range 1 month to 25 years). Surgical strategy was determined, not by formal protocol, but by subjective assessment of right ventricular (RV) size and coronary anatomy in each individual patient. Overall actuarial survival was 80 ± 5% at 1 year, 75 ± 6% at 5 years and 75 ± 6% at 8 years. Initial procedure in infancy consisted of an aorto-pulmonary shunt alone in 33 (group I), shunt with RV decompression in 32 (group H), and primary heart transplant in 2 (group III). In group I, 3 patients (9%) died after shunt placement without further intervention; Fontan operation was performed in 21 (64%) with 2 early deaths (no late deaths), late RV recruitment in 4 (12%) with no deaths, and transplant in 3 (9%) with 1 early death (no late deaths). Two patients have had superior cavopulmonary connection and are awaiting Fontan completion. In

group II, 7 infants (22%) died early after RV decompression, 2 of these had dysplastic tricuspid valves with massively dilated RV and 2 had associated aortic/subaortic stenosis; 16 (50%) completed separation into a biventricular circulation with 1 death, 3 (9%) underwent superior cavo-pulmonary connection with maintenance of antegrade RV flow ("½ ventricle repair") with 0 deaths, 2 (6%) had transplant with 1 early death, and 1 (3%) had successful Fontan operation. Three patients (9%) are pending separation into a biventricular circulation. In group III, 1 patient is alive after transplant and 1 died early after organ graft failure. One and 5 year actuarial survivals for group I (91 ± 5%, 80 ± 7%) versus group II (71 ± 8%, 71 ± 8%) were not statistically different. Coronary angiographic data was available in 60 patients; 35 (58%) had no abnormalities, 17 (28%) had RV-to-coronary fistulas alone, and 8 (13%) had fistulas with important coronary stenoses/interruption. Actuarial survival at 1 year was similar for these groups: 83 ± 6%, 93 ± 7%, 75 ± 15%, respectively. Superior long-term outcome is achieved for PA/IVS when the surgical strategy is targeted to the specific anatomical substrate present.

12:10 p.m. ADJOURN

*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION B-2

ADULT CARDIAC SURGERY

Ballroom B, Hynes Convention Center

Moderators: Karl H. Krieger, M.D.

Hartzel V. Schaff, M.D.

47. CORONARY BYPASS SURGERY IN PATIENTS WITH PREVIOUS MEDIASTINAL RADIATION THERAPY.

Nobuhiro Handa, M.D.*, Christopher G.A. McGregor, M.B., F.R.C.S., Gordon K. Danielson, M.D., Thomas A. Orszulak, M.D., Charles J. Mullany, M.D.*, Richard C. Daly, M.D.*, Joseph A. Dearani, M.D.*, Betty J. Anderson, R.N.* and Francisco J. Puga, M.D.

Rochester, Minnesota

Discussant: Bruce W. Lytle, M.D., Cleveland, Ohio

Between January 1976 and December 1996, 72 patients with previous mediastinal radiation therapy (MRT) underwent combined cardiac surgery. Forty-seven of these 72 patients who had CABG without valve surgery were reviewed for this study. The mean age was 63.5 ± 12.8 years (range 31 to 82 years). Indications for MRT were breast cancer (n = 26), lymphoma (n = 18) and lung cancer (n = 3). The mean interval between MRT and CABG was 15.1 ± 9.8 yrs (range 1.1 to 37.8 yrs). Preoperative NYHA class III or IV and CAC class III or IV were 93.6 and 89.4, respectively. Fifty-one percent of patients had a history of previous MI. The number of distal anastomosis performed was 2.9 ± 0.9. The left internal mammary artery was used as a LAD graft in 29.8% of patients. Associated cardiac procedures included coronary endarterectomy (2), LV aneurysmectomy (1), and pericardiectomy (2). Operative mortality was 4 cases (8.5%). Complications included prolonged ventilatory support 4 (8.5%), sternal wound infection or mal-union 4 (8.5%), IABP insertion 2 (4.3%), bleeding 2 (4.3%), renal insufficiency 1, gastric perforation 1, and infective endocarditis 1. Total followup was 293.7

patient-years (mean 6.2 ±5.1 yrs). There were 17 late deaths (malignancy 9, heart failure 5, stroke 1, unknown 2). Twelve of these 43 discharged patients developed valvular disease echocardiographically during follow-up (MR+ TR 4, AS+MS 5, AS+AR 2, mild AS 1). Nine patients required late reoperation (CABG 3, AVR+CABG 1, MV repair 1, heart transplant 1, wound repair 2, AICD/pacemaker placement 2). The mean interval between the original CABG and the six open cardiac procedures was 4.4 ± 2.8 years (range 1.1 to 8.6 yrs). Actuarial survival of the whole group (n = 47) was 71.6% (95% confidence interval 58.7%, 86.9%) at five years. These results indicate that increased early mortality, sternal wound complications and reoperation can be expected in this patient population. As progressive valvular disease developed in 27.9% of discharged patients at followup, and 25 of the original 72 patients required concomitant valve and coronary surgery, careful assessment of valvular lesions is important at the time of initial CABG. Careful followup, including echo, is recommended.
*By invitation

48. VALUE OF DOBUTAMINE ECHOCARDIOGRAPHY FOR PREDICTION OF FUNCTIONAL RECOVERY, SYMPTOMATIC IMPROVEMENT AND LONG-TERM SURVIVAL IN PATIENTS WITH CHRONIC LEFT VENTRICULAR ISCHEMIC DYSFUNCTION UNDERGOING CORONARY BYPASS SURGERY.

Jean-Louis J. Vanoverschelde, M.D., Ph.D.*, Agnes Pasquet, M.D.*, Jacques A. Melin, M.D., Ph.D.*, Philippe Noirhomme, M.D.*, Gebrine El Khoury, M.D.*, Robert Verhelst, M.D.* and Robert A. Dion, M.D.*

Brussels, Belgium

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

Discussant: Hillel Laks, M.D., Los Angeles, California

Background Most control studies comparing bypass surgery (CABG) to medical treatment in patients with poor left ventricular (LV) function have demonstrated the superiority of CABG in alleviating symptoms, preventing reinfarction and prolonging survival. Yet, not every patient with poor LV function does benefit from CABG, perhaps because it continues to entail significant immediate risks. In this study, we examined the possible contribution of dobutamine echocardiography (DbE) to the risk stratification of these patients.

Methods. Seventy-six consecutive patients (age, 60 ± 10 years) with coronary disease and chronic LV ischemic dysfunction (ejection fraction: 35 ± 11%) underwent DbE prior to CABG. CABG was performed with the use of the left internal mammary artery to graft the left anterior descending coronary artery. All other co-diseased vessels were also revascularized. On average, each patient received 2.9 anastomoses, of which 1.4 were constructed with arterial conduits. After CABG, patients were followed for 3 years, starting from the date of CABG and ending with a cardiac fatal event or on the most recent date in survivors. In each patient, the recovery of LV function was evaluated by echocardiography 5.3 ± 2.4 months after CABG. Survival and symptomatic status at follow-up were obtained from review of visit and hospital records or by telephone contact.

Results. Functional recovery was evaluated on both a segmental and an individual patient basis. On a segmental basis, pre-operative DbE correctly identified 74% of the segments that improved and 86% of those that remained dysfunctional after CABG. Overall accuracy was 81%. In individual patients, assessment of residual contractile reserve, defined as a decrease in wall motion score by > 3.5 during low-dose DbE, correctly identified 84% of those who improved LV ejection fraction by > 5% after CABG (n = 37) and 73% who failed to do so (n = 33). Overall accuracy was 79%.

During followup, 11 patients (15%) died of cardiac causes, 4 in hospital (5%) and 7 later on (6 sudden deaths, 1 progressive pump failure). Kaplan-Meier survival curves indicated that 3-year survival was significantly better in the 40 patients who exhibited residual contractile reserve pre-operatively (95%) than in the 36 patients who did not (75%, $P<0.01$). Among survivors, symptoms of heart failure (as reflected by the NYHA functional class) improved only in those with residual contractile reserve (from 1.9 ± 0.9 to 1.2 ± 0.4, $P<0.01$) but not in those without (from 1.9 ± 0.9 to 1.8 ± 0.9, P = ns).

Conclusions. The present study indicates that DbE allows for a comprehensive evaluation of patients with chronic LV ischemic dysfunction and permits accurate prediction of functional recovery and symptomatic improvement, as well as both short- and long-term survival.

*By invitation

49. TWELVE YEAR EXPERIENCE WITH THE RIGHT GASTROEPIPLOIC ARTERY GRAFT.

Hisayoshi Suma, M.D., Taiko Horii, M.D.*, Tadashi Isomura, M.D.* and Tetsuya Ichihara, M.D.*

Kamakura, Japan

Discussant: Robert A. Dion, M.D., Brussels, Belgium

Since 1986, the right gastroepiploic artery (GEA) graft has been used in 800 CABGs. Clinical and angiographic late outcome is reported.

Material and method: There were 682 men and 118 women with a mean age of 61 years. Single, double , triple and left main diseases were noted in 6 , 134, 522 and 138 patients, respectively. Previous MI was noted in 474 (60%) patients and 68 (9%) patients had previous CABG.

Sites of GEA grafting were 68 LAD, 7 diagonal, 131 CX and 616 RCAs with 22 sequential and 26 free grafts. Combined grafts were 768 (96%) ITAs (123 double ITAs), 45 (6%) inferior epigastric and 65 (8%) radial arteries. Saphenous vein was combined in 389(49%) patients.
Angiographic restudy was performed in 618 patients within one year, 682 patients after I to 5 years and 23 patients from 5 to 10 years.
Consecutive 189 patients (163 men and 26 women, with a mean age of 59 years) who were operated between 1986 to 1992 with single ITA+GEA ± SV graft were followed for more than 5 years (mean 7.7 years) and their late outcomes were evaluated.

Results: Operative death, new Q wave and postoperative IABP were noted in 16 (2%), 12 (1.5%) and 19 (2.4%) patients, respectively.

Angiographic restudy showed the patency rates of GEA graft at early (2 months), mid-term (2 years) and late (7 years) periods were 94%, 89% and 83% respectively. Late closure of GEA was mainly noted in case of less critically stenosed RCA and focal stenosis was rare in late period.
In the late followup group, 5 and 10 year survival rates were 88.4% and 85.0%, cardiac death-free rate was 94.5% and 93.4%, and cardiac event-free survival was 85.6% and 80.1%, respectively.

Conclusion: GEA graft can be used safely and effectively. Angiographic late result was favorable and the late outcome of single ITA + GEA is comparable with the reported double ITA grafts.

*By invitation

50. INTRAVENOUS T_3 IMPROVES MYOCARDIAL FUNCTION AND REDUCES MORBIDITY AFTER CORONARY BYPASS SURGERY: RESULTS OF A DOUBLE-BLIND RANDOMIZED TRIAL.

Samantha L. Mullis-Jansson, M.D.*, Michael Argenziano, M.D.*, Steven J. Corwin, M.D.*, Sunichi Homma, M.D.*, Eric A. Rose, M.D. and Craig R. Smith, M.D.

New York, New York

Discussant: Andrew S. Wechsler, M.D., Philadelphia, Pennsylvania

Background: Thyroid hormone is known to exert profound effects on myocardial function. Although triiodothyronine (T_3) deficiency has been described after cardiopulmonary bypass, preliminary studies examining the role of T_3 in the management of cardiovascular performance following CABG surgery have yielded conflicting results. In order to further define the clinical role of T_3 in cardiac surgery, a double-blind, randomized, placebo-controlled study was undertaken.

Methods: 130 consecutive patients undergoing elective CABG were enrolled. Emergency surgery, age greater than 85 years, and a history of thyroid disease or amiodarone therapy were exclusionary criteria. Upon removal of the aortic crossclamp, patients were randomized to intravenous T_3 (0.4 mcg/kg bolus + 0.1 mcg/kg infusion for 6 hours, n = 66) or placebo (n = 64). Outcome measures included perioperative hemodynamics and inotrope/pressor requirements at several time points (mean ± SEM), perioperative morbidity (arrhythmia/infarction), and mortality.

Results: Despite similar baseline characteristics, patients randomized to T_3 had a higher cardiac index and lower inotropic requirements postoperatively (table). In addition, patients randomized to T_3 demonstrated a significantly lower incidence of postoperative myocardial infarction (4.5% vs. 15.6%, $p < 0.05$) and pacemaker dependence (15.2% vs. 31.3%, $p < 0.05$). T_3 and placebo patients had equivalent rates of atrial fibrillation. Five patients in the placebo group required postoperative mechanical assistance (3 IABP, 2 LVAD), compared to none in the T_3 group ($p = 0.02$). There were 2 deaths in the placebo group and no deaths in the T_3 group.

	Cardiac Index (L/min/m^2)			Dopamine dependence (% of pts)	
Group	Preop	Post-CBP	Postop hr 12	Postop hr 6	Postop hr24
T_3	2.49±0.66	2.55±0.55	3.00±0.60	10.6%	4.6%
Placebo	2.43±0.57	2.17±0.55	2.78±0.60	19.4%	16.1%
p value	NS	<0.001	<0.005	0.09	<0.05

Conclusions: Parenteral T_3 given after crossclamp removal during elective CABG significantly improved postoperative ventricular function, reduced dependence on inotropic agents and mechanical devices, and decreased the incidence of myocardial infarction. While the incidence of atrial fibrillation was not affected, T_3 reduced the requirement for postoperative pacemaker support.

10:50 a.m. INTERMISSION

*By invitation

11:10 a.m. SIMULTANEOUS SCIENTIFIC SESSION B-2 ADULT CARDIAC SURGERY

Ballroom B, Hynes Convention Center

Moderators: Karl H. Krieger, M.D.

Hartzel V. Schaff, M.D.

51. LONG TERM IMPLANTED LEFT VENTRICULAR ASSIST DEVICES FUNCTION AS IMMUNE-INFLAMMATORY ORGANS.

Talia B. Spanier, M.D.*, †Mehmet C. Oz, M.D., David M. Stern, M.D.*, Silviu Itescu, M.D.* and Ann Marie Schmidt, M.D.*

New York, New York

Discussant: D. Glenn Pennington, M.D., Winston-Salem, North Carolina

Successful long-term use of implantable left ventricular assist devices (LVAD) is dependent on improved understanding of host-device interactions. The textured surface LVAD was designed to promote the time-dependent cellular population of the device and creation of a biologic lining. We examined the procoagulant and proinflammatory consequences of implantation of this mechanical device and found evidence of sustained thrombin generation (elevated thrombin-antithrombin III complex and prothrombin fragment 1+2) and fibrinolysis (d-dimers and fibrinogen degradation products) as well as clinically-significant immunological alterations with polyclonal B cell activation and autoantibody production. Significant elevations in anti-HLA antibodies were found to directly correlate with difficulty in identification of a negative cross match for cardiac transplantation (anti-MHC class I antibodies), and increased frequency/severity of cellular rejections post transplantation (anti-MHC class II antibodies). In addition, an increased incidence of anti-phospholipid antibodies was recognized in the LVAD population, often associated with clinically-apparent thrombocytopenia. A qualitative T cell defect with an overall increased susceptibility to opportunistic infections (25% incidence of candidal infections over first 100 days awaiting cardiac transplantation vs 2% in UNOS I heart failure controls, $p = 0.026$) supports the overall immunoregulatory dysfunction associated with LVAD implantation. We believe that circulating blood cells which are selectively adsorbed to the LVAD surface became activated causing the LVAD to become an immune-inflammatory organ with these significant proinflammatory/procoagulant consequences. Analysis of the cells populating the LVAD surface (35 days postop and beyond) support this contention revealing dendritic-type $CD34^+$ cells, as well as cells bearing monocyte (CD14)/macrophage (CD68) markers, and T (CD4/CD25/CD3) and B (CD20) cell markers. Dendritic-type cells also strongly express Vascular Cell Adhesion Molecule-1 (VCAM-1) and Intercellular Adhesion Molecule-1 (ICAM-1), consistent with their capacity to further recruit other circulating cells. RT-PCR confirms cellular activation on the LVAD surface, with transcripts for proinflammatory Interleukin (IL)-la, IL-2 and Tumor Necrosis Factor-a, in addition to VCAM-1 and ICAM-1. RT-PCR also shows that the activated T cells on the LVAD have a TH2 cytokine profile (mRNA was detected for IL-10 and CD40 Ligand but not for gamma interferon, IL-4 or IL-5), consistent with their promotion of an autoimmune phenotype. Furthermore, LVAD recipients demonstrate a reduced proliferative response to candidal antigens compared with heart failure controls (mean stimulation index 1.4 vs 2.0), and are anergic on intradermal challenge with candidal and control antigens. Together, these observations suggest that selective adsorption and activation of circulating dendritic-type cells and monocytic cells by the

LVAD surface contribute to the development of an overall immunoregulatory dysfunction in LVAD recipients. The LVAD therefore emerges as an implanted immune-inflammatory organ providing a sustained pro-inflammatory and pro-thrombotic stimulus with significant impact on the host beyond its mechanical function as a pump.

†1994-96 AATS Robert E. Gross Research Scholar

*By invitation

52. THE COX-MAZE III PROCEDURE: PARALLEL NORMALIZATION OF SINUS NODE DYSFUNCTION, IMPROVEMENT OF ATRIAL FUNCTION AND RECOVERY OF THE CARDIAC NERVOUS SYSTEM.

Miralem Pasic, M.D., Ph.D.*, Onnen Grauhan, M.D.*, Michele Musci, M.D.*, Takeo Teodoriya, M.D.*, Barbara Edelmann, M.S.*, Roland Hetzer, M.D., Ph.D. and Hendryk Siniawski, M.D.*

Berlin, Germany

Discussant: James L. Cox, M.D., Washington, DC

OBJECTIVE: The Cox-maze III procedure, the technique of choice for the management of atrial fibrillation, includes isolation of the pulmonary veins and multiple incisions in both atria in what corresponds to partial autotransplantation and partial denervation of both parasympathetic and sympathetic systems of the heart. In contrast, transplanted heart is completely denervated and, therefore, without efferent parasympathetic or sympathetic innervation. The aim of this prospective longitudinal study was to identify physiological effects of reinnervation on changes in heart rate at rest and in response to various stimulations, and atrial function following the Cox-maze procedure and orthotopic heart transplantation.

PATIENTS AND METHODS: Thirty adult patients with combined the Cox-maze III procedure and mitral valve surgery (PD-partially denervated maze group) and 15 heart-transplant recipients (CD-completely denervated Tx group) were prospectively followedup at 1, 3, 6, and 12 months after surgery. The results were compared to normal probands (control group, n = 12). Power spectral analysis of heart rate variability, exercise testing, rapid positional changes, Valsalva maneuver, 24-hour Holler monitoring and standard electrocardiogram were used to assessed dysfunction of the autonomic nervous system, ability of the sinus node to accelerate in response to internal physiological chronotropic stimuli, and heart rate variability. The atrial function was assessed by transesophageal echocardiography.

RESULTS: At 1 and 3 months, both groups exhibited the physiological effects of denervation with no differences in cardiac autonomic activity between the two groups as demonstrated by very low values in power spectral analysis of heart rate variability, inhibited cardiac autonomic activity, and no response after sympathetic stress (LF, HF, LF/HF: at 0 degree 12.5, 32.5, 0.38; at 60 degrees 1.2, 16.4; and 0.07, respectively). At 6 months, the maze group but not the Tx group demonstrated evidence of autonomic reinnervation. Recovery of cardiac autonomic activity was documented at one year in the maze group (LF, HF, LF/HF: at 0 degree 206.7, 403.4, 051; at 60 degress 9.6, 25.6, 0.37, respectively) but not in the Tx group. Inappropriate heart rate responses during physical exercise were clearly evident in both groups after 1 and 3 months, with progressive improvement seen between 6 and 12 months only in the maze group. Atrial function after the maze procedure improved parallel to the recovery of the sinus node function.

CONCLUSION: Progredient improvement of sinus node function and atrial contractions with functional normalization one year after the Cox-maze procedure corresponded to functional reinnervation and recovery of autonomic nervous system.

*By invitation

53. MANAGEMENT OF VASODILATORY SHOCK AFTER HIGH- RISK CARDIAC SURGERY: INDENTIFICATION OF PREDISPOSING FACTORS AND USE OF A NOVEL PRESSOR AGENT.

Michael Argenziano, M.D.*, Jonathan M. Chen, M.D.*, Asim F. Choudhri, B.S.*, Donald W. Landry, M.D.*, Alan D. Weinberg, M.S.*, Craig R. Smith, M.D., Eric A. Rose, M.D. and †Mehmet C. Oz, M.D.

New York, New York

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

Background: Cardiopulmonary bypass (CPB) may induce significant peripheral vasodilatation after cardiac surgery, often requiring perioperative catecholamine pressor support. Although arginine vasopressin (AVP) normally has no vasoactive properties in doses as high as 0.26 U/min, pressor effects have been described for lower doses in hypotensive patients unresponsive to traditional pressors. We investigated the incidence and clinical predictors of vasodilatory shock (VS) in high-risk cardiac surgical patients and the effects of low-dose AVP in the treatment of this syndrome.

Methods: In Phase I, 122 patients undergoing CPB for cardiac surgery (CABG, AYR, MVR, LVAD, heart transplant) were studied. Preoperative ejection fraction (EF), medications, and perioperative hemodynamics were recorded, and post-bypass serum AVP levels measured. VS was defined as mean arterial pressure (MAP) <70 mmHg, cardiac index (CI) >2.5 L/min/m^2 and norepinephrine (NE) dependence. In Phase II, patients who developed VS after heart transplantation (HTx) or left ventricular assist device (LVAD) placement were identified and treated with AVP infusions (0.1 U/min) and hemodynamic responses recorded.

Results: In Phase I, 14 of 122 patients (11%) met criteria for post-bypass VS (MAP 65 ± 2 mmHg and NE dose 7.4 ± 1.8 mcg/min). By multivariate analysis, EF < 0.35 and diuretic use were identified as independent predictors of post-bypass VS (relative risk of 7.4 and 3.8, respectively). VS was associated with inappropriately low serum AVP concentrations (12.9 ± 3.1 pg/ml), and the severity of vasodilatation was correlated to the degree of AVP deficiency (r = 0.81). In Phase II, 25 patients (14 HTx, 11 LVAD) developed post-bypass VS (MAP 59.5 ± 2.0 mmHg and NE dose 19.8 ± 4.1 mcg/min) and responded to low dose AVP infusions, with rapid increases in MAP (to 82.2 ± 3.2 mmHg, p<0.0001) and reductions in NE dependence (to 8.6 ± 3.2 mcg/min, p = 0.02). Ten patients (40%) were weaned completely off NE within 30 minutes.

Conclusions: Heart failure and diuretic dependence are risk factors for post-bypass vasodilatory shock, which may be associated with vasopressin deficiency. In patients exhibiting this syndrome after high-risk cardiac surgery, physiologic replacement infusions of AVP stabilize hemodynamics by improving blood pressure and reducing or eliminating catecholamine pressor requirements.

12:10 p.m. ADJOURN

†1994-96 AATS Robert E. Gross Research Scholar

*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION C-2

GENERAL THORACIC SURGERY
Ballroom C, Hynes Convention Center

Moderators: Mark K. Ferguson, M.D.

G. Alexander Patterson, M.D.

54. LONG-TERM FOLLOWUP AFTER SURGICAL TREATMENT OF PRIMARY SOFT TISSUE SARCOMAS OF THE LUNG.

John Langenfeld, M.D.*, Nael Martini, M.D., Michael Burt, M.D., Ph.D., Manjit S. Bains, M.D., Robert J. Downey, M.D.*, Valerie W. Rusch, M.D. and Robert Ginsberg, M.D.

New York, New York

Discussant: Cameron D. Wright, M.D., Boston, Massachusetts

Objective: Primary soft tissue sarcomas of the lung are rare, representing 0.4% of all lung malignancies, and data evaluating the results of treatment are sparse. Because of this, we reviewed our experience with this uncommon entity.

Methods: Retrospective review of medical records. Resectability, tumor size, and histological cell type were analyzed as predictors of survival. Survival was calculated by Kaplan-Meier method; survival differences compared by log rank.

Results: There were 42 patients; age ranged from 2 - 78 yr (median 53). There were leiomyosarcomas, 13 spindle cell sarcomas, 5 rhabdomyosarcomas, 4 malignant fibrous histiocytomas, 2 fibrosarcomas, and 1 malignant hemangiopericytoma. Of the 42 patients, 39 were explored with 19 complete resections (CR), 12 incomplete resections (IR), and 8 biopsy (Bx) only. There were 2 perioperative deaths (1 IR, 1 Bx). The overall actuarial five year survival was 25% (median 17 mos). The 1, 3, and 5 year survival for those having CR was 89%, 72% and 58%, respectively, which was significantly ($p<0.0001$) greater than those having an IR (1, 3, 5 yr survival = 42%, 8%, 0%; median 8 mos) or those having a Bx (1, 3 yr survival = 18%, 0%; median 7 mos). Eighteen patients (43%) developed distant metastases (11 to the contralateral lung, 7 bone, 3 brain and 1 liver). Regional metastases to mediastinal or hilar lymph nodes were identified in 12 patients (28%). Of the 19 patients having a CR, 10 (53%) developed a recurrence; 3 recurred distantly, 4 locoregionally, and 3 both distantly and locoregionally. The median time to the development of a recurrence following complete resection was 10 months. Tumor size did not impact on survival.

Conclusion: The best predictor for long-term survival in patients with primary soft tissue lung sarcomas is a complete resection. However, despite complete resection, patients remain at risk for the development of distant metastases, which occur predominately in lung and bone. As with other soft tissue sarcomas, adjuvant therapies should continue to be investigated.

*By invitation

55. BRONCHOALVEOLAR CARCINOMA: CLINICAL, RADIOLOGICAL, PATHOLOGICAL FACTORS AND SURVIVAL.

Kenichi Okubo, M.D.*, Eugene J. Mark, M.D.*, Douglas Flieder, M.D.*, John C. Wain, M.D.*, Cameron D. Wright, M.D., Ashby C. Moncure, M.D., Hermes C. Grille, M.D. and Douglas J. Mathisen, M.D.

Gifu, Japan and Boston, Massachusetts

Discussant: Mark S. Allen, M.D., Rochester, Minnesota

Bronchoalveolar carcinoma (BAG) is the least common of the non-small cell lung carcinoma variants. The tumors typically spread along airways or by aerogenous routes and may be multifocal. Prognostic factors and patterns of survival have not been clearly defined.

We studied 119 patients with pathologically confirmed BAG, of which 114 underwent thoracotomy (107 resection, 7 biopsy only). Clinical, radiological and pathologic factors were examined and factors affecting survival were analyzed.

Results

		Operation		Stage	
Asymptomatic	71.4%	Lobectomy	73	I	74.3%
Symptomatic	28.6%	Bilobectomy	6	II	6.4%
		Pneumonectomy	7	III	3.7%
Radiographic Mass	79.0%	Segmentectomy	8		
Single	67.2%	Wedge	13	Operative Mortality=2.4%	
Multiple	11.8%	Lung biopsy	7		
Infiltrate	21.0%				

Pathologic Findings

BAC%			**Miotic Index**			**Lymphocyte Grades**	
Aerogenous spread	95.0%	50%	16.8%	0	57.1%	0	5.0%
Mucinous	21.8%	60%	19.3%	1	34.5%	1	31.9%
Non Mucinous	68.9%	70%	11.8%	2	0.0084%	2	34.5%
Sclerosing	80.7%	80%	18.5%	**Nuclear Grade**		3	18.5%
Association with scar	60.5%	90%	19.3%	Well Differentiated			17.6%
		100%	14.3%	Moderately differentiated			59.7%
				Poorly Differentiated			12.6%

Positive Effect	p	No Effect
Mass vs. Infiltrate	0.0037	Single vs. Multiple
Sclerosing vs. Non	0.0010	< 3 cm vs. > 3 cm
Scar vs. No Scar	0.0156	Mucinous vs. Nonmucinous
50-90% BAC vs. 100%BAC	0.022	Nuclear grade
Grade 3 lymphocyte vs. 0,1,2	0.0001	

N0 vs. N 1-2	0.0008	
Complete Resurrection vs. Incomplete	0.0001	

We have identified favorable prognostic factors that predict long term survival in BAC.
*By invitation

56. LONG-TERM RESULTS OF PROSTHETIC CHEST-WALL RECONSTRUCTION.

Claude Deschamps, M.D., Ramin Darbandi-Tonkabon*, Mehmet B. Tirnaksiz, M.D.*, Victor F. Trastek, M.D., Daniel L. Miller, M.D.*, Mark S. Allen, M.D., Phillip G. Arnold, M.D.* and Peter C. Pairolero, M.D.

Rochester, Minnesota

Discussant: Richard H. Feins, M.D., Rochester, New York

Between January 1977 to December 1992, 137 patients (77 males and 60 females) underwent chest wall (CW) resection and reconstruction with prosthetic material. Median age was 57 years (range, 11-86). Indication for resection was recurrent malignancy in 46 patients (33.6%), contiguous lung or breast carcinoma in 44 (32.1%), primary CW malignancy in 37 (27.0%) and others in 10 (7.3%). Three patients (2.2%) presented with an open draining wound. Fifty patients (36.5%) were smokers, 39 (28.5%) had preoperative chemotherapy and/or radiation, and 19 (14.0%) were on oral corticosteroids. This study covers two time periods. Fifty-three patients (38.7%) were reconstructed with Prolene mesh (PM) during the period from 1977 to 1986. Eighty-four patients (61.3%) were reconstructed with Gore-tex (GT) soft tissue patch from 1984 to 1992. Soft tissue coverage with transposed muscle or omentum was used in 75 patients (54.7%); the remainder had coverage with local tissue only. There were 2 deaths (1.5%) and 48 patients (35%) had at least one complication. Median hospitalization was 19 days (range, 4-142). Followup was complete in all 135 operative survivors and ranged from one to 144 months (median, 11 months). Ninety-five patients (70.4%) had well-healed wounds and were asymptomatic. An additional 36 patients (26.7%) initially had well healed wounds but developed a local recurrence. Local recurrence was greatest in those patients with breast cancer. Four PM and none of the GT had to be removed. Seromas occurred in eight patients; seven were small and resolved while the last (GT) required wound exploration. Late wound infections occurred in seven patients (5 PM and 2 GT; P0.05). Four of the five PM had to be removed. The wounds in the remaining three patients (2 GT and 1 PM) were all opened and cleaned with eventual healing in all three. While late wound infection occurred more commonly in patients with PM, this may reflect our early inexperience of placing prosthetic material in contaminated wounds. We conclude that CW resection and prosthetic reconstruction will yield satisfactory results in most patients.

57. PREVALENCE AND LOCATION OF NODAL METASTASES IN DISTAL ESOPHAGEAL ADENOCARCINOMA CONFINED TO THE WALL.

John J. Nigro, M.D.*, Steven R. DeMeester, M.D.*, Jeffrey A. Hagen, M.D.*, Stefan Oberg, M.D.*, Jeffrey H. Peters, M.D.*, Milton Kiyabu, M.D.*, Peter F. Crookes, M.D.*, Cedric G. Bremner, M.D.* and Tom R. DeMeester, M.D.

Los Angeles, California

Discussant: Thomas W. Rice, M.D., Cleveland, Ohio

Background: Barrett's surveillance programs have identified an increasing number of patients with early esophageal cancer. The purpose of this study was to characterize the prevalence and location of lymph node metastases in patients with invasive adenocarcinoma confined to the esophageal wall.

Methods: Esophagogastrectomy combined with systematic mediastinal and abdominal lymphadenectomy was performed on 34 patients with Barrett's adenocarcinoma confined to the esophageal wall. Patients who had preoperative chemotherapy, radiotherapy, or previous esophageal resection were excluded. Followup was complete in 33 of 34 patients at a mean of 34 months (median 22 months).

Results: The depth of tumor invasion was limited to the mucosa in 15 patients, the submucosa in 9 patients, and the muscularis propria in ten patients. Histologically positive nodes were found in 12 of 34 patients (35.3 %), and the prevalence of nodal metastases increased progressively with depth of invasion.

The location of each positive node is presented in the following table, grouped by the number of involved nodes per patient.

1 Node (n = 6)	2 Nodes (n = 4)	3 Nodes (n = 1)	> 4 Nodes (n = 1)
Lesser Curve (3)	Lesser Curve (1)	Lesser Curve (1)	Hiatal and Splenic (1)
Celiac (2)	Lesser Curve And Azygos		
Hiatal (1)	Azygos and Greater Curve (1)		
	Hiatal (1)		

Actuarial survival for the entire group was 63% at 5 years. Recurrence was identified in 5 of the 34 patients (15 %). There have been no recurrences in patients with intramucosal carcinoma. However, systemic recurrence was identified in 2 of 9 patients with submucosal and 3 of 10 patients with muscular invasion.

Conclusions: The prevalence of nodal metastases correlates with depth of wall invasion, and once the muscular wall is penetrated the majority of patients have involved nodes. Although most metastases are found along the lesser curvature, lymph node metastases do occur in areas not routinely removed by trans-hiatal resection. A low recurrence rate and good long term survival can be achieved in these patients emphasizing the importance of total esophagectomy with lymphadenectomy to include all potentially involved nodes.

10:50 a.m. INTERMISSION

*By invitation

11:10 a.m. SIMULTANEOUS SCIENTIFIC SESSION C-2

GENERAL THORACIC SURGERY
Ballroom C, Hynes Convention Center

Moderators: Mark K. Ferguson, M.D.

G. Alexander Patterson, M.D.

58. HEART-LUNG VERSUS DOUBLE-LUNG TRANSPLANTATION FOR SUPPURATIVE LUNG DISEASE.

Clifford W. Barlow, M.D.*, Robert C. Robbins, M.D.*, Marc R. Moon, M.D.*, James Theodore, M.D.* and Bruce A. Reitz, M.D.

Stanford, California

Discussant: Thomas M. Egan, M.D., Chapel Hill, North Carolina

Heart-lung (HLTx) and double-lung (DLTx) transplantation have both been advocated for patients with end-stage suppurative lung disease. We reviewed our experience of all patients with cystic fibrosis (CF) or bronchiectasis who underwent HLTx or DLTx between 1/88 and 8/97 to obtain medium-term follow-up and comparative results. Twenty-two patients (13 male, 21 CF) had HLTx and 24 patients (8 male, 19 CF) had DLTx. 'Domino[1] heart transplantation took place in all HLTx patients with suitable cardiac function. The mean age of the HLTx group was 26 ± 13 years (range 9 to 49) and of the DLTx group was 28 ± 14 years (range 10 to 53). There were no differences in weight, pre-operative creatinine, bilirubin, CMV status, maintenance immunosuppression, and medical management between the 2 groups. Patients all received induction therapy with monoclonal (OKT3) (9 HLTx, 3 DLTx) or polyclonal (RATG) antibody (13 HLTx, 21 DLTx). Sixteen of 24 patients had DLTx after 1/95 while 13 of 22 patients had HLTx before 1/91. Mean waiting time for DLTx was 3614 ± 47 days and for HLTx was 270 ± 52 days. Actuarial analyses of outcomes for the groups are as follows:

		3 months	1 year	3 years	5 years	p value
Survival (%)	HLTx	95	81	81	64	NS
	DLTx	95	95	81	-	
Freedom from obliterative bronchiolitis (OB) death (%)	HLTx	100	100	95	86	NS
	DLTx	100	100	84	-	
Freedom from OB (%)	HLTx	95	76	60	44	NS
	DLTx	100	84	73	-	
Freedom from lung rejection (%)	HLTx	43	37	21	21	NS
	DLTx	61	56	48	-	
Freedom from heart rejection (%)	HLTx	80	75	69	61	-
	DLTx	NA	NA	NA	NA	
Freedom from graft atherosclerosis (GCAD) (%)	HLTx	100	100	85	85	-
	DLTx	NA	NA	NA	NA	
Linearized infection rate (events/100 patients days)	HLTx	2.05	0.11	0.36	0.09	NS
	DLTx	2.34	0.29	0.18	-	

Thirty day survival was 100 % for HLTx and 96 % for DLTx. There were 7 late HLTx deaths (3 OB, 2 infection, 0 GCAD, 2 Other) and 2 late deaths in the DLTx group (2 OB).

These data show that HLTx and DLTx for suppurative lung diseases have comparable short and medium-term survival, OB, rejection and infection rates. While heart rejection episodes and GCAD may be experienced in HLTx, they have not resulted in retransplantation or death.

We continue to consider DLTx or HLTx with ‘domino' heart transplantation for patients with end-stage suppurative lung disease.

*By invitation

59. EFFECT OF CMV AND DEVELOPMENT OF HLA ANTIBODIES ON LUNG TRANSPLANT SURVIVAL AND BRONCHIOLITIS ON OBLITERANS SYNDROME.

†Michael A. Smith, M.D.*, Sudhir Sundaresan, M.D.*, T. Mohanakumar, Ph.D.*, Elbert P. Trulock, M.D.*, John P. Lynch, M.D.*, Donna L. Phelan, B.A, C.H.S.*, Joel D. Cooper, M.D. and G. Alexander Patterson, M.D. *St. Louis, Missouri*

Discussant: R. Morton Bolman, III, M.D., Minneapolis, Minnesota

The long term survival of lung transplant recipients continues to be limited by the development of bronchiolitis obliterans syndrome (BOS). A retrospective analysis was done to identify factors which may effect long-term survival and play a role in the development of BOS.

Methods: Of 295 consecutive lung transplants (LT) performed from July 1988 to December 1995, 264 survived at least 3 months and form the basis for this analysis. There was a minimum followup period of 12 months. BOS was defined as a decline in spirometry (FEV1 < 80% of baseline) and/or histologic presence of obliterative bronchiolitis. Variables analyzed included cytomegalovirus (CMV) antibody status and post-transplant development of lymphocytotoxic antibodies against human leukocyte antigens (HLA antibodies). Recipient sera were tested by microcytotoxicity assays. Those having cytotoxicity to greater than 10% of a known panel of HLA alleles were identified as positive for HLA antibodies. Variables were subjected to Kaplan-Meier analysis for effects on overall survival and freedom from BOS.

Results: Overall survival was 78%, 55%, and 39% at 3, 5, and 7 years, respectively. At 3 and 5 years 39% and 23% of patients, respectively, remained free of BOS. There appeared to be worse survival of CMV seronegative patients who received allografts from a seropositive donor compared to seronegative recipient/donor combinations and seropositive patients receiving allografis from seronegative donors ($p = 0.074$). There was also a trend toward greater frequency of development of BOS in seronegative patients who received allografts from seropositive donors ($p = 0.116$). Most strikingly, the development of HLA antibodies after transplantation significantly correlated with the development of BOS ($p = 0.048$). Further, these patients who developed HLA antibodies had poorer survival ($p = 0.063$).

Conclusion: These data suggest that BOS is the result of an immune mediated process in which HLA antibodies may play a significant role. The effect of CMV on the development of BOS and long term survival remains uncertain.

†1998-99 TSFRE Research Fellow

*By invitation

60. BILATERAL SEQUENTIAL LUNG TRANSPLANT WITHOUT STERNAL DIVISION ELIMINATES POST TRANSPLANT STERNAL COMPLICATIONS.

Bryan Meyers, M.D.*, Sudhir Sundaresan, M.D.*, Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.

St. Louis, Missouri

Bilateral sequential single lung transplantation (BLT) is the procedure of choice for bilateral lung replacement. This procedure is usually performed through a bilateral anterolateral thoracosternotomy ("clam-shell") incision. Unfortunately the "clam-shell" is associated with complications such as sternal malunion, dehiscence, osteomyelitis and migrating hardware in as many as 50% of patients. From 1989 to the present we have performed 228 adult BLT. During the past 16 months, 35 of 47 BLT were conducted without sternal division. Thirty-one were performed through bilateral anterolateral fourth interspace thoracotomies, three through left posterolateral and right anterolateral thoracotomy and one through sequential posterolateral thoracotomies. The recipient diagnoses were COPD in 11 patients, alpha-1 antitrypsin deficiency emphysema in seven patients, cystic fibrosis in seven patients, bronchiectasis in five patients, pulmonary fibrosis in three patients, bronchiolitis obliterans (retransplant) in one patient and primary pulmonary hypertension in one patient. Three patients were placed on cardiopulmonary bypass electively to permit transplantation. The intact sternum did not impede ascending aortic and right atrial cannulation. Early in our experience with this technique two patients underwent sternal division for institution of emergent cardiopulmonary bypass to repair an atrial laceration in one patient and a left atrial suture line defect in another patient. These two patients were the only deaths in this experience (5.7%). There were no incisional complications among operative survivors. With experience BLT can be safely conducted without sternal division thereby avoiding the frequent complications associated with transverse sternotomy.

12:10 p.m. ADJOURN

*By invitation

GEOGRAPHICAL ROSTER

NECROLOGY

Michael E. Burt, M.D..... New York, New York
Forest D. Dodrill, M.D..... Baltimore, Maryland
Richmond L. Moore, M.D..... Lynchburg, Virginia
George C. Morris, M.D..... Shepherd, Texas
Thomas F. Nealon, Jr., M.D..... New York, New York
Edward F. Skinner, M.D..... Forest City, Arizona
Chester M. VanAllen, M.D..... Bikaner, India
George W. Wright, M.D..... Denver, Colorado

The American Association for Thoracic Surgery

(Listed by Countries, States, Provinces and Cities)

Geographical - UNITED STATES
1997-1998

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Kirklin, John W
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McPhail, Jasper L
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Copeland, Jack G, III
Sanderson, Richard G
Sethi, Gulshan K
ARKANSAS
Little Rock
Campell, Gilbert S
Read, Raymond C
CALIFORNIA
Anaheim
Main, F Beachley
Berkeley
Young, J Nilas
Bonita
Gonzales-Lavin, Lorenzo
Burlingame
Ullyot, Daniel J
Capistrano Beach
Plynn, Pierce J
Chico
Becker, Ronald M
Coronado
Silver, Arthur W
Covina
Wareham, Ellsworth E
Del Mar
Fosburg, Richard G
El Macero
Andrews, Niel C
Escondido
Mannix, Edgar P, Jr
Flintridge
Penido, John R F
Fresno
Evans, Byron H
Hanford
Kerth, William J
Indian Wells
Carter, P Richard
Salyer, John M
Inglewood
Lee, Myles E
Irvine
Connolly, John E
Wakabayashy, Akio
La Canada
Meyer, Bertrand W
La Jolla
DeLaria, Giacomo, A
Hutchin, Peter
Loma Linda
Bailey, Leonard L
Gundry, Steven R

Long Beach
Bloomer, William E
Stemmer, Edward A
Los Angeles
Buckberg, Gerald D
Chaux, Aurelio
Cohen, Robbin G
DeMeester, Tom R
Holmes, E. Carmack
Kay, Jerome H
Khonsari, Siavosh
Laks, Hillel
Lindesmith, George G
Longmire, William, Jr
Maloney, James V, Jr
Mandal, Ashis K
Matloff, Jack M
McKenna, Robert J, Jr
Mulder, Donald G
Starnes, Vaughn A
Trento, Alfredo
Waters, Paul F
Los Osos
Aronstam, Elmore M
Martinez
Guernsey, James M
Montebello
Lui, Alfred H F
Oakland
Ecker, Roger R
Iverson, Leigh I G
Orange
Gazzaniga, Alan B
Oxnard
Dart, Charles H, Jr
Palo Alto
Cohn, Roy B
Jamplis, Robert W
Peters, Richard M
Wilson, John L
Palos Verdes Estates
Stiles, Quentin R
Pasadena
Hughes, Richard K
Ingram, Ivan N
Newman, Melvin M
Pebble Beach
Miller, George E, Jr
Ramsay, Beatty H
Portola Valley
Fogarty, Thomas J
Redding
Moreno-Cabral, Ricardo J
Sacramento
Benfield, John R
Berkoff, Herbert A
Follette, David M
Harlan, Bradley J
Hurley, Smeloff, J
Edward A Edward
San Bernardino
Misbach, Gregory A
San Diego
Baronofsky, Ivan D
Chambers, John S
Daily, Pat O
Dembitsky, Walter P
Jamieson, Stuart W
Lamberti, John J
Miller, Fletcher A
Mountain, Clifton F
Trammer, Max J
San Francisco
Ellis, Robert J
Grimes, Orville F
Hanley, Frank L
Hill, J Donald
Leeds, Sanford E
Roe, Benson B
Thomas, Arthur N
Turley, Kevin
San Jose
Oakes, David D
San Marino
Tsuji, Harold K
Santa Ana
Pratt, Lawrence A
Santa Barbara
Higginson, John F
Jahnke, Edward J
Love, Jack W
Santa Cruz
Fishman, Noel H
Santa Monica
Fonkalsrud, Eric W
Morton, Donald L
Nelson, Ronald
J Robertson, John M
Santa Rosa
Neville, William E
Sausalito
Zaroff, Lawrence I
Sonoma
Richards, Victor
Spring Valley
Long, David M, Jr
St Helena
Dugan, David J
Stanford
Mark, James B. D
Miller, D. Craig
Oyer, Philip E
Reitz, Bruce A
Shumway, Norman E
Pomerantz, Marvin
Rainer, W. Gerald
Englewood
Kovarik, Joseph L
Littleton
Pappas, George
Vail
Fuller, Josiah
CONNECTICUT
Bridgeport
Rose, Daniel M
Cheshire
Blumenstock, David A
Essex
Jaretzki, Alfred, III
Hartford
Kemler, R Leonard
New Haven
Elefteriades, John A
Glenn, William W. L
Hammond, Graeme L

Stinson, Edward B
Tiburon
Heydorn, William H
Torrance
Carey, Joseph S
Cukingnan, Ramon A
Moore, Thomas C
State, David
Valley Village
Davis, Lowell L
Victorville
Jurado, Roy A
Walnut Creek
May, Ivan A
COLORADO
Beulah
Bartley, Thomas D
Denver
Brown, Robert K
Campbell, David N
Clarke, David R
Eiseman, Ben
Grover, Frederick L
Grow, John B, Sr
Harken, Alden H
Hopeman, Alan R
Paton, Bruce C
Kopf, Gary S
New Milford
Okinaka, Arthur J
Wilton
Pool, John L
Woodbridge
Lindskog, Gustaf E
Stern, Harold
DELAWARE
Newark
Lemole, Gerald M
Wilmington
Norwood, William I
Pecora, David V
DISTRICT OF COLUMBIA
Washington
Cox, James L
Gomes, Mario N
Katz, Nevin M
Keshishian, John M
Lefemine, Armand A
Midgley, Frank M
Simmons, Robert L

FLORIDA
Atlantic Beach
Stranahan, Allan
Bal Harbour
Grondin, Pierre
Belleair
Lasley, Charles H
Boca Raton
Seley, Gabriel P
Clearwater
Wheat, Myron W, Jr
Coconut Grove
Center, Sol
Coral Gables
Cooke, Francis N
Delray Beach
Shumacker, Harris B, Jr
Gainesville
Alexander, James A
Jacksonville
Edwards, Fred H
Koster, J Kenneth, Jr
Stephenson, Sam, Jr
Jupiter
Gerbasi, Francis S
Lakeland
Brown, Ivan W, Jr
Marathon
Mangiardi, Joseph L
Miami
Bolooki, Hooshang
Daughtry, Dewitt C
Greenberg, Jack J
Jude, James R
Kaiser, Gerard A
Papper, Emanuel M
Ripstein, Charles B
Thurer, Richard J
Wilder, Robert J
Miami Beach
Reis, Robert L
Mundth, Eldred D
Smyth, Nicholas P D
Orlando
Scott, Meredith L
Ponte Vedra Beach
Gilbert, Joseph, Jr
Punta Gorda
Taber, Rodman E
St Petersburg
Daicoff, George R
Tallahassee
Kraeft, Nelson H
Tamarac
Mendelssohn, Edwin
Tampa
Angell, William W
Robinson, Lary A
Seller, Hawley H
Winter Haven
Maurer, Elmer P R
Winter Park
Sherman, Paul H
GEORGIA
Atlanta
Craver, Joseph M
Gott, John P
Guyton, Robert A
Hatcher, Charles R, Jr
Hopkins, William A
Jones, Ellis L
Kanter, Kirk R
King, Richard
Lee, Arthur B, Jr
Mansour, Kamal A
Miller, Joseph I, Jr
Rivkin, Laurence M
Symbas, Panagiotis
Williams, Willis H
Augusta
Ellison, Robert G
Rubin, Joseph W

Spear, Harold C
Subramanian, S

Naples
Battersby, James S
Linberg, Eugene J
MacGregor, David C

Marietta
Netterville, Rush E

Savannah
Yeh, Thomas J

St Simons Island
Taylor, Frederick H

HAWAII

Honolulu
Ching, Nathaniel P
Gebauer, Paul W
McNamara, J. Judson

IDAHO

Boise
Herr, Rodney H

ILLINOIS

Burr Ridge
Blakeman, Bradford P

Chicago
Amato, Joseph J
Backer, Carl L
Barker, Walter L
Barwinsky, Jaroslaw
Breyer, Robert H
Campbell, Charles D
Ebert, Paul A
Faber, L. Penfield
Ferguson, Mark K
Fullerton, David A
Goldin, Marshall D
Hanlon, C. Rollins
Head, Louis R
Hunter, James A
Ilbawi, Michel N
Karp, Robert B
Kittle, C Frederick
Mavroudis, Constantine
Michaelis, Lawrence
Montoya, Alvaro
Najafi, Hassan
Raffensperger, John
Replogle, Robert L
Shields, Thomas W
Tatooles, C, J
Thomas, Paul A, Jr
Vanecko, Robert M
Warren, William H

Downers Grove
Leininger, Bernard J

Chickamauga
Hall, David P

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

Elk Grove Village
Sullivan, Henry J

Evanston
Fry, Willard A

Glencoe
Rubenstein, L H

Harvey
Norman, John C

Maywood
DeLeon, Serafm Y
Mason, G. Robert
Pifarre, Roque

Oak Brook
Javid, Hushang
Jensik, Robert J
Nigro, Salvatore L

Park Ridge
Baffes, Thomas G
Weinberg, Milton, Jr

Peoria
DeBord, Robert A

Springfield
Wellons, Harry A, Jr

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
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
McFadden, P Michael
Mills, Noel L
Moulder, Peter V
Ochsner, John L
Pearce, Charles W
Schramel, Robert J
Webb, Watts R

MAINE

Portland
Bredenberg, Carl E
Morton, Jeremy R

Prairie Village
Holder, Thomas M
Shawnee Mission
Adelman, Arthur
Mayer, John H, Jr
Padula, Richard T
Wichita
Tocker, Alfred M
KENTUCKY
Lexington
Crutcher, Richard R
Mentzer, Robert M, Jr
Swain, Julie A
Todd, Edward P
Louisville
Austin, Erie H, III
Gray, Laman A, Jr
Mahaffey, Daniel E
Ransdell, Herbert, Jr
LOUISIANA
Alexandria
Knoepp, Louis F
Baton Rouge
Berry, B Eugene
Beskin, Charles A
Jonesboro
Bloodwell, Robert D
Metairie
Ochsner, Alton, Jr
New Orleans
Blalock, John B
DeCamp, Paul T
Ferguson, T Bruce, Jr
Hartz, Renee S
Hewitt, Robert L
Lindsey, Edward S
Daly, Benedict D T
Del Nido, Pedro J
Ellis, F. Henry, Jr
Frank, Howard A
Gaensler, Edward A
Grille, Hermes C
Hilgenberg, Alan D
Johnson, Robert G
Jonas, Richard A
Lazar, Harold L
Levitsky, Sidney
LoCicero, Joseph, III
Mathisen, Douglas J
Mayer, John E
Mentzer, Steven J
Moncure, Ashby C
Rheinlander, Harold F
Sellke, Frank W
Shemin, Richard J
Starkey, George W B
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
Burlington
Shahian, David M
Rockport
Swenson, Orvar
Windham
Hiebert, Clement
MARYLAND
Baltimore
Attar, Safuh
Baker, R. Robinson
Baumgartner, William A
Blair, Emil
Cameron, Duke Edward
Gott, Vincent L
Haller, J. Alex, Jr
Hankins, John R
Krasna, Mark J
McLaughlin, Joseph S
Michelson, Elliott
Salomon, Neal W
Watkins, Levi, Jr
Fort Detrick
Zajtchuk, Rostik
Glenarm
Turney, Stephen Z
Reisterstown
Heitmiller, Richard F
Worton
Walkup, Harry E
MASSACHUSETTS
Boston
Akins, Cary W
Austen, W. Gerald
Bougas, James A
Buckley, Mortimer J
Burke, John F
Cohn, Lawrence H
Collins, John J, Jr
Daggett, Willard M
Shrewsbury
Moran, John M
Springfield
Engelman, Richard M
Rousou, John A
Vineyard Haven
Malm, James R
Wellesley Hills
Cleveland, Richard J
West Newton
Neptune, Wilford 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
Boiling, Steven F
Bove, Edward L
Deeb, G. Michael
Gago, Otto
Greenfield, Lazar J
Kirsh, Marvin M

Cambridge
Berger, Robert L
Malcolm, John A
Chestnut Hill
Laforet, Eugene G
Dover
Black, Harrison
Falmouth
McElvein, Richard B
Framingham
Bernhard, William F
Schuster, Samuel R
Medford
Desforges, Gerard
Methuen
Wilson, Norman J
North Andover
Cook, William A
Morris, Joe D
Neerken, A John
Orringer, Mark B
Prager, Richard L
Sloan, Herbert E
Beverly Hills
Timmis, Hilary H
Detroit
Arbulu, Agustin
Pass, Harvey I
Silverman, Norman A
Steiger, Zwi
Stephenson, Larry W
Wilson, Robert F
Grand Rapids
Harrison, Robert W
Rasmussen, Richard A
Tomatis, Luis A

St Joseph
Levine, Frederick H
West Bloomfield
Arciniegas, Eduardo
MINNESOTA
Coon Rapids
Gannon, Paul G
Mendota Heights
Dennis, Clarence
Minneapolis
Arom, Kit V
Bolman, R. Morton, III
Emery, Robert W
Foker, John E
Garamella, Joseph J
Helseth, Hovald K
Molina, J. Ernesto
Nicoloff, Demetre M
Shumway, Sara J
Rochester
Allen, Mark S
Bernatz, Philip E
Danielson, Gordon K
Deschamps, Claude
McGoon, Dwight C
McGregor, Christopher G A
Olsen, Arthur M
Orszulak, Thomas A
Pairolero, Peter C
Payne, W. Spencer
Puga, Francisco J
Schaff, Hartzell V
Trastek, Victor F
Shorewood
Kiser, Joseph C
St Paul
Lillehei, C. Walton
Stillwater
Kaye, Michael P
Waubun
DeNiord, Richard N
MISSISSIPPI
Carthage
Logan, William D, Jr
Jackson
Johnston, J. Harvey, Jr
Madison
Hardy, James D
MISSOURI
Bridgeton
Codd, John E
Chesterfield
Bergmann, Martin
Columbia
Bryant, Lester R
Curtis, Jack J
Silver, Donald
Walls, Joseph T
Kansas City
Ashcraft, Keith W
Borkon, A Michael
Killen, Duncan A
Piehler, Jeffrey M
Reed, William A
Van Way, Charles W, III
Mount Vernon
Campbell, Daniel C, Jr
St Louis
Earner, Hendrick B
Baue, Arthur E
Connors, John P
Cooper, Joel D
Ferguson, Thomas B
Fiore, Andrew C
Flye, M Wayne
Gay, William A, Jr
Huddleston, Charles B
Johnson, Frank E
Kaiser, George C
Kouchoukos, Nicholas T
Lewis, J Eugene, Jr
McBride, Lawrence R
Naunheim, Keith S
Pasque, Michael K
Patterson, G. Alexander
Roper, Charles L
Strevey, Tracy E, Jr
Willman, Vallee L
MONTANA
Missoula
Duran, Carlos Gomez
Oury, James H
NEBRASKA
Omaha
Fleming, William H
Schultz, Richard D

NEVADA
Las Vegas
Little, Alex G
NEW HAMPSHIRE
Center Harbor
Aaron, Benjamin L
Franconia
Taylor, Warren J
Jaffrey
Woods, Francis M
Lebanon
Nugent, William C
Sanders, John H, Jr
NEW JERSEY
Alpine
Holswade, George R
Belleville
Gerard, Franklyn P
Browns Mills
Fernandez, Javier
McGrath, Lynn B
Carnden
Camishion, Rudolph C
DelRossi, Anthony J
East Orange
Auerbach, Oscar
Hackensack
Hutchinson, John E, III
Jersey City
Demos, Nicholas J
Millburn
Parsonnet, Victor
Moorestown
Morse, Dryden P
Morristown
Parr, Grant V S
Neptune
Roberts, Arthur J
New Brunswick
Lewis, Ralph J
MacKenzie, James W
Scholz, Peter M
Newark
Bregman, David
Donahoo, James
Gielchinsky, Isaac
Swan, Kenneth G
Pittstown
Garzon, Antonio A
Short Hills
Hochberg, Mark S
Tenafly
Gerst, Paul H
Wyckoff
Adler, Richard H
NEW MEXICO
Albuquerque
Edwards, W. Sterling
Wernly, Jorge A
Buena Vista
Thai, Alan P
Santa Fe
Davila, Julio C
Santa Teresa
Glass, Bertram A
Silver City
Waddell, William R
NEW YORK
Albany
Foster, Eric D
Bay Shore
Ryan, Bernard J
Bronx
Altai, Lari A
Brodman, Richard F
Fell, Stanley C
Ford, Joseph M
Prater, Robert W M
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
Dietl, Charles A.
Guiraudon, Gerard M
Hoover, Eddie L
Lajos, Thomas Z
Salerno, Tomas A

East Amherst
Andersen, Murray N
East Quogue
McCormack, Patricia M
Fayetteville
Bugden, Walter F
Efiler, Donald B
Floral Park
Crastnopol, Philip
Honeoye Falls
Graver, William L
Larchmont
Steichen, Felicien M
Lido Beach
Mines, George L
Millerton
Green, George E
New Rochelle
Redo, S. Frank
Reemtsma, Keith
Rose, Eric A
Rusch, Valerie W
Skinner, David B
Smith, Craig R
Spencer, Frank C
Spotnitz, Henry M
Subramanian, Valavanur A
Tice, David A
Tyras, Denis H
Wichern, Walter, Jr
Wolff, William I
Patchogue
Finnerty, James
Plattsburgh
Potter, Robert T
Rochester

Rubin, Morris

New York

Adams, Peter X
Altorki, Nasser K
Anagnostopoulos, C E
Bains, Manjit S
Beattie, Edward, Jr
Bloomberg, Allan E
Boyd, Arthur D
Cahan, William G
Clauss, Roy H
Colvin, Stephen B
Conklin, Edward F
Culliford, Alfred T
Ergin, M Arisan
Friedlander, Ralph
Galloway, Aubrey C, Jr
Ginsberg, Robert J
Griepp, Randall B
Isom, O. Wayne
King, Thomas C
Kirschner, Paul A
Krieger, Karl H
Lansman, Steven L
Litwak, Robert S
Martini, Nael
McCord, Colin W
Michler, Robert E
Nealon, Thomas F, Jr
Oz, Mehmet C
Quaegebeur, Jan M

Chapel Hill

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

Charlotte

Robicsek, Francis
Selle, Jay G

Durham

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

Greensboro

Van Trigt, Peter, III

Greenville

Chitwood, W Randolph, Jr

High Point

Mills, Stephen A

Oriental

Deaton, W Ralph, Jr

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
DeWeese, James A
Hicks, George L
Schwartz, Seymour I
Stewart, Scott

Roslyn

Thomson, Norman B, Jr
Wisoff, George

Saranac Lake

Decker, Alfred M, Jr

Scarsdale

Robinson, George

Scottsville

Emerson, George L

Slingerlands

Kausel, Harvey W

Stony Brook

Soroff, Harry S

Syracuse

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

Valhalla

Moggio, Richard A
Reed, George E

NORTH CAROLINA

Asheville

Belts, Reeve H
Kroncke, George M
Scott, Stewart M
Takaro, Timothy

Cincinnati

Albers, John E
Callard, George M
Flege, John B, Jr
Gonzalez, Luis L
Helmsworth, James A
Hiratzka, Loren F
Ivey, Tom D
Wilson, James M
Wright, Creighton B
Yee, Edward S

Cleveland

Cosgrove, Delos M
Geha, Alexander S
Groves, Laurence K
Kay, Earle B
Kirby, Thomas J
Loop, Floyd D
Lytle, Bruce W
McCarthy, Patrick M
Rice, Thomas W
Snow, Norman J
Van Heeckeren, Daniel W

Columbus

Davis, J Terrance
Kakos, Gerard S
Meckstroth, Charles
Williams, Thomas E, Jr

Dayton

DeWall, Richard A

Delaware

Clatworthy, H. Williams, Jr

Grove City

Kilman, James W

OKLAHOMA

Jenks

LeBeck, Martin B

Meredith, Jesse H
Pennington, D. Glenn

OHIO

Blacklick
Myerowitz, P. David

Chagrin Falls
Ankeney, Jay L
Cross, Frederick S

Lawton
Barnhorst, Donald A

Oklahoma City
Elkins, Ronald C
Felton, Warren L, II
Fisher, R Darryl
Greer, Allen E
Munnell, Edward R
Zuhdi, M Nazih

OREGON

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

Camp Hill
Pennock, John L

Carlisle
DeMuth, William, Jr

Darby
McKeown, John J, Jr

Dresner
Jeevanandam, Valluvan

Hershey
Campbell, David B
Damiano, Ralph J, Jr
Pae, Walter E, Jr
Pierce, William S
Waldhausen, John A

Johnstown
Kolff, Jacob

Lancaster
Bonchek, Lawrence I
Rosemond, George P
Witmer, Robert H

Norristown
Dunn, Jeffrey M

Philadelphia
Addonizio, V. Paul
Bowles, L Thompson
Brockman, Stanley K
Diehl, James T.
DiSesa, Verdi J
Edie, Richard N
Edmunds, L. Henry, Jr
Fineberg, Charles
Gardner, Timothy J
Goldberg, Melvyn
Hargrove, W Clark, III
Kaiser, Larry R
MacVaugh, Horace
Mannion, John D
Nemir, Paul, Jr
Sink, James D
Spray, Thomas L
Wechsler, Andrew S.
Whitman, Glenn J R

Pittsburgh
Bahnson, Henry T
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
Myers, John L
Pontius, Robert G
Rams, James J
Siewers, Ralph D

Rosemont
Templeton, John Y, III

Rydel
Frobese, Alfred S

Wayne
Lemmon, William M

Wilkes-Barre
Cimochowski, George E

Wynnewood
Wallace, Herbert W

Yardley
Sommer, George N, Jr

RHODE ISLAND

Providence
Hopkins, Richard A
Moulton, Anthony L
Singh, Arun K

SOUTH CAROLINA

Charleston
Bradham, R Randolph
Crawford, Fred A, Jr
Kratz, John M
Parker, Edward F
Reed, Carolyn E
Sade, Robert M

Columbia
Almond, Carl H

Hilton Head Island
Humphrey, Edward W

Isle of Palms
Mullen, Donald C

Landrum
Stayman, Joseph W

Spartanburg
Utley, Joe R

TENNESSEE

Knoxville
Seybold, William D
Urschel, Harold, Jr

Dilley
Hood, Richard H, Jr

Galveston
Conti, Vincent R

Blake, Hu Al
Brott, Walter H
Domm, Sheldon E

Memphis
Cole, Francis H
Eastridge, Charles E
Hughes, Felix A, Jr
McBurney, Robert P
Pate, James W
Robbins, S Gwin, Sr
Rosensweig, Jacob
Shochat, Stephen J
Watson, Donald C

Nashville
Alford, William, Jr
Bender, Harvey W, Jr
Drinkwater, Davis C
Gobbel, Walter G, Jr
Merrill, Walter H
Randolph, Judson G
Rankin, J Scott
Sawyers, John L
Scott, H. William, Jr
Stoney, William S
Thomas, Clarence, Jr

Sparta
Labrosse, Claude C

TEXAS

Amarillo
Sutherland, R Duncan

Austin
Hood, R Maurice
Tyson, Kenneth R T

Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Lambert, Gary J
Mack, Michael J
Paulson, Donald L
Platt, Melvin R
Razzuk, Maruf A
Ring, W Steves
Derrick, John R
Zwischenberger, Joseph B

Houston
Baldwin, John C
Beall, Arthur C, Jr
Burdette, Walter J,PhD,
Cooley, Denton A
Coselli, Joseph S
DeBakey, Michael E
Frazier, O. Howard
Hallman, Grady L
Henly, Walter S
Jones, James W
Lawrie, Gerald M
Mattox, Kenneth L
Ott, David A
Overstreet, John W
Putnam, Joe B, Jr
Reardon, Michael J
Reul, George J, Jr
Roth, Jack A
Safi, Hazim J
Walker, William E
Wukasch, Don C

Irving
Mills, Lawrence J

Kemp
Davis, Milton V

Lubbock
Bricker, Donald L
Feola, Mario
Wallsh, Eugene

San Antonio
Calhoon, John H
Cohen, David J
Dooley, Byron N
Heaney, John P
Treasure, Robert L
Trinkle, J. Kent

Temple
Brindley, G. Valter, Jr

Woodville
Harrison, Albert W

UTAH

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

VERMONT

Richford
Grondin, Claude M

West Dover
Humphreys, George H, II

VIRGINIA

Altavista
Pierucci, Louis, Jr

Annandale
Akl, Bechara F
Burton, Nelson A
Lefrak, Edward A

Arlington
Klepser, Roy G

Aylett
Gwathmey, Owen

Charlottesville

Mercer Island
Li, Wei-i

Poulsbo
Malette, William G

Seattle
Allen, Margaret D
Anderson, Richard P
Cochran, Richard P
Hill, Lucius D
Jones, Thomas W
Lupinetti, F. Mark
Manhas, Dev R
Mansfield, Peter B
Merendino, K. Alvin
Miller, Donald W, Jr
Rittenhouse, Edward
Sauvage, Lester R
Thomas, George I
Verrier, Edward D

Spokane
Berg, Ralph, Jr

WEST VIRGINIA

Charleston
Walker, James H

Huntington

Dammann, John F
Daniel, Thomas M
Kron, Irving L
Minor, George R
Muller, William H, Jr
Nolan, Stanton P
Spotnitz, William D
Tribble, Curtis G
Fredericksburg
Armitage, John M
McLean
Conrad, Peter W
Mills, Mitchell
Wallace, Robert B
Reston
Boyd, Thomas F
Richmond
Bosher, Lewis H, Jr
Brooks, James W
Cole, Dean B
Guerraty, Albert J
Lower, Richard R
WASHINGTON
Bellingham
Varco, Richard L
Friday Harbor
Lawrence, G Hugh
Issaquah
Jarvis, Fred J
Kirkland
Mills, Waldo O
Ferraris, Victor A
Morgantown
Graeber, Geoffrey M
Gustafson, Robert A
Hill, Ronald C
Murray, Gordon F
Warden, Herbert E
Parkersburg
Tarnay, Thomas J
WISCONSIN
Eau Claire
McEnany, M Terry
Madison
Chopra, Paramjeet S
Young, William P
Marshfield
Myers, William O
Ray, Jefferson F, III
Sautter, Richard D
Mequon
Narodick, Benjamin
Milwaukee
Johnson, W Dudley
Litwin, S Bert
Olinger Gordon N
Tector, Alfred J
West Bend
Gardner, Robert J
WYOMING
Teton Village
Kaunitz, Victor H

Geographical - Canada
1997-1998

CANADA
ALBERTA
Calgary
Bharadwaj, Baikunth
Miller, George E
Edmonton
Callaghan, John C
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
Cohen, Morley
NOVA SCOTIA
Halifax
Murphy, David A
Mabou
Thomas, Gordon W
ONTARIO
Collingwood
Heimbecker, Raymond
London
McKenzie, F Neil
Menkis, Alan H
Ottawa
Keon, Wilbert J
Sudbury
Walker, George R
Toronto
Baird, Ronald J
Bigelow, Wilfred G
Christakis, George T
Coles, John G
David, Tirone E
Feindel, Christopher M
Fremes, Stephen E
McKneally, Martin F
Mickleborough, Lynda L
Pearson, F. Griffith
Scully, Hugh E
Todd, Thomas R J
Trimble, Alan S
Trusler, George A
Weisel, Richard D
Williams, William G
Westbrook
Lynn, R Beverley
QUEBEC
Montreal
Blundell, Peter E
Carrier, Michel
Chartrand, Claude C. C
Chiu, Chu-Jeng (Ray)
Cossette, Robert
Dobell, Anthony R C
Duranceau, Andre C H
MacLean, Lloyd D
Morin, Jean E

Novick, Richard J
North York
Goldman, Bernard S
Nottawa
Key, James A
Oakville
Allen, Peter
Mulder, David S
Pelletier, L. Conrad
Scott, Henry J
Shennib, Hani
Sainte-Foy
DesLauriers, Jean

Geographical - OTHER COUNTRIES
1997-1998

ARGENTINA
Buenos Aires
Favaloro, Rene G
AUSTRALIA
Brisbane
O'Brien, Mark F
Beaumont
Sutherland, H D'Arcy
Melbourne
Karl, Tom R
AUSTRIA
Leonding
Bruecke, Peter E
Salzburg
Unger, Felix H
Vienna
Wolner, Ernst
BELGIUM
Leuven
Lerut, Antoon E. M. R
Sergeant, Paul T
BRAZIL
Rio de Janeiro
Meier, Milton A
Sao Paulo
Jatene, Adib D
CENTRAL AMERICA
Guatemala
Castaneda, Aldo R
DENMARK
Copenhagen
Pettersson, Gosta B
ENGLAND
Bath, Avon
Belsey, Ronald
Cambridge
Kennedy, John H
Wallwork, John
Herts
Lennox, Stuart C
Liverpool
Donnelly, Raymund J
London
Braimbridge, Mark V
de Leval, Marc R
Lincoln, Christopher R
Ross, Donald N
Stark, Jaroslav F
Taylor, Kenneth M
Yacoub, Magdi
Oxford
Clark, Richard E.
Westaby, Stephen
Somerset
Abbey-Smith, R
FINLAND
Kauniainen
Mattila, Severi P
FRANCE
Bordeaux
Baudet, Eugene M
Fontan, Francis M
Le Plessis Robinson
Binet, Jean-Paul
Dartevelle, Philippe G
Lacour-Gayet, Francois
Lyon
Champsaur, Gerard L
Marseille
Metras, Dominique R
Montpellier
Thevenet, Andre A
Paris
Blondeau, Philip
Cabrol, Christian E A
Carpentier, Alain F
Loisance, Daniel
Menasche, Philippe
Piwnica, Armand H
Planche, Claude
Weldon, Clarence S
Pessac
Couraud, Louis
Suresnes
Bachet, Jean E
GERMANY
Aachen
Messmer, Bruno J
Berlin
Hetzer, Roland
Freiburg
Beyersdorf, Friedhelm
Hannover
Haverich, Axel
Munich
Borst, Hans G
Neuss
KOREA
Seoul
Cho, Bum-Koo
MONACO
Dor, Vincent
NETHERLANDS
Wassenaar
Brom, A Gerard
NEW ZEALAND
Barratt-Boyes, Brian G
P.R. OF CHINA
Beijing
Ying-Kai, Wu
PORTUGAL

Bircks, Wolfgang H
StraBlach-Dinghartin
Sebening, Fritz
GREECE
Athens
Palatianos, George M
GUATEMALA
Guatemala City
Herrera-Llerandi, Rodolfo
IRELAND
Dublin
O'Malley, Eoin
ITALY
Bergamo
Parenzan, Lucio
Milan
Peracchia, Alberto
Naples
Cotrufo, Maurizio
Pisa
Bortolotti, Uberto
Rome
Marcelletti, Carlo
JAPAN
Kamakura
Suma, Hisayoshi
Kanazawa-city
Iwa, Takashi
Kitakyushushi
Miyamoto, Alfonso T
Osaka
Kawashima, Yasunaru
Matsuda, Hikaru
Sendai
Mohri, Hitoshi
Shinjuku-ku
Imai, Yasuharu
Tokyo
Koyanagi, Hitoshi
Wada, Juro J
Coimbra
Antunes, Manuel J
Lisbon
Machado Macedo, Manuel E M
ROMANIA
Targu-Mures
Deac, Radu C
RUSSIA
Moscow
Bockeria, Leo A
SAUDI ARABIA
Riyadh
Landymore, Roderick W
SCOTLAND
Edinburgh
Logan, Andrew
Glasgow
Wheatley, David J
SPAIN
Barcelona
Aris, Alejandro
Madrid
Rivera, Ramiro
Santander
Revuelta, Jose Manuel
SWEDEN
Sollentuna
Bjork, Viking
SWITZERLAND
Arzier
Hahn, Charles J
Pully
Naef, Andreas P
Zurich
Senning, Ake
Turina, Marko I
VENEZUELA
Caracas
Tricerri, Fernando E

THE 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
J. Frank Corbett
Armistead C. Crump
Charles N. Dowd
Kennon Dunham
Edmond Melchior Eberts
Max Einhorn
Herman Fischer
Albert H. Garvin
Arthur A. Law
William Lerche
Howard Lilienthatl
William H. Luckett
Morris Manges
Walton Martin
Rudolph Matas
E.S. McSweeny
Samuel J. Metzler
Willy Meyer (Founder)
James Alexander Miller
Robert T. Miller
Fred J. Murphy
Leo S. Peterson
Eugene H. Pool
Walter I. Rathbun
Martin Rehling
B. Merrill Ricketts
Samuel Robinson

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

BY-LAWS OF

THE AMERICAN ASSOCIATION

FOR THORACIC SURGERY

ARTICLE I. NAME

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

ARTICLE II. PURPOSE

The purposes of the Association shall be:

To associate persons interested in, and carry on activities related to, the science and practice of thoracic surgery, the cure of thoracic disease and the related sciences.

To encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology and therapy, and to correlate and disseminate such knowledge.

To hold scientific meetings featuring free discussion of problems and developments relating to thoracic surgery, and to sponsor a journal for the publication of scientific papers presented at such meetings and other suitable articles.

To succeed to, and continue to carry on the activities formerly conducted by The American Association for Thoracic Surgery, an unincorporated association.

ARTICLE III. MEMBERSHIP

Section 1. There shall be three classes of members: Honorary, Senior, and Active. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws. Honorary members shall have the privilege of voting but shall not be eligible to hold office.

Section 2. Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with concurrence of the Association.

Section 3. The number of Senior Members shall be unlimited. Active Members automatically advance to Senior Membership at the age of sixty-five years. In addition, a younger Active Member may be eligible for Senior Membership if incapacitated by disability, but for no other reason.

Section 4. Active Membership shall be limited to six hundred. A candidate to be eligible must be a citizen of the United States of America or Canada, unless in unusual cases this citizenship requirement shall have been waived by the Council. The candidate shall have achieved distinction in the thoracic field or shall have made a meritorious contribution to knowledge pertaining to thoracic disease or its surgical treatment.

Section 5. Election to Honorary, Senior or Active Membership shall be for life, subject to the provisions of Section 8 following. All new members shall be elected directly to Honorary or Active status.

Section 6. Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active, Senior or Honorary Members. Such nomination must have been in the hands of the Membership Committee for not less than four months, and the name of the candidate must have been distributed to all members of the Association before final action may be taken on any new candidate for election to Active Membership. Provided the foregoing requirements have been met and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a future regularly convened annual meeting for final action. A three-fourths vote of those present and voting shall be required to elect. Any candidate for membership in the Association who has failed of election three times shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

Section 7. The report of the Membership Committee shall be rendered at the second executive session of each annual meeting of the Association. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; Candidates for Active Membership; members dropped from the rolls of the Association.

Section 8. Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving such member ample opportunity to appear in his own behalf.

Section 9. The Council shall recommend that any Active Member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from three consecutive annual meetings, shall have his membership terminated.

Section 10. Notwithstanding Section 9, any member of the Association over 65 years of age is excused from the attendance requirement and upon his specific request may likewise be excused from the payment of dues.

ARTICLE IV. Board of Directors ("Council")

Section 1. The Board of Directors of the Association shall be called the Council and shall be composed of the President, Vice-President, Secretary, Treasurer, five Councilors and the Editor of the Association shall be a member ex-officio without vote. All members of the Council must be Active or Senior Members of the Association, except that the Editor may be an Honorary Member.

Section 2. The Council shall be the governing body of the Association, and shall have full power to manage and act on all affairs of the Association, except as follows:

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

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

c. It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article III, Section 8.

Section 3. At the conclusion of the annual meeting, the retiring President shall automatically become a Councilor for a one-year term of office. One of the other four Councilors shall be elected at each annual meeting of the Association to serve for a four-year term of office in the place of the elected Councilor whose term expires at such meeting, but no Councilor may be re-elected to succeed himself. Any Councilor so elected shall take office upon the conclusion of the annual meeting at which he is elected.

Section 4. Vacancies in the office of Councilor shall be temporarily filled by the Council subject to approval of the Association at the next annual meeting of the Association.

ARTICLE V. Officers

Section 1. The officers of the Association shall be President, a Vice-President, a Secretary, and a Treasurer. All officers must be Active or Senior Members of the Association. Said officers shall be *ex officio* members of the Council of the Association.

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

Section 3. The President, Vice-President, Secretary and Treasurer shall be elected at the annual meeting of the Association and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for a one-year term of office and neither may be re-elected to succeed himself in the same office, unless such officer is filling the unexpired term of an officer previously elected to such office. The Secretary and the Treasurer shall be elected for a one-year term of office and may be re-elected for not more than four additional terms.

Section 4. The President of the Association shall perform all duties customarily pertaining to the office of President. He shall preside at all meetings of the Association and at all meetings of the Council.

Section 5. The Vice-President of the Association shall perform all duties customarily pertaining to the office of the Vice-President, both as to the Association and the Council. In the event of a vacancy occurring in the office of President, the Council shall advance the Vice-President to the Presidency and appoint an interim Vice-President.

Section 6. The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve as Secretary of the Association and as Secretary of the Council. When deemed appropriate an Active or Senior Member may be elected to serve as an understudy to the Secretary in anticipation of the latter's retirement from office.

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve as Treasurer of the Association.

Section 8. The Editor of the Association is not an officer of the Association. He shall be appointed by the Council at its annual meeting; provided, however, that such appointment shall not become effective until approved by the Association at the annual meeting of the Association. The Editor shall be appointed for a five-year term and may not be appointed to more than two successive

terms; provided, however, that an Editor completing two years or less of the unexpired term of a previous Editor may be appointed for two successive five-year terms. The Editor shall serve as the Editor of the Official Journal and shall be *ex officio* the Chairman of the Editorial Board and a member of the Council of the Association without vote.

Section 9. Vacancies occurring among the officers named in Section 1 or a vacancy in the position of Editor shall be temporarily filled by the Council, subject to approval of the Association at the next meeting of the Association.

ARTICLE VI. Committees

Section 1. The Council is empowered to appoint a Membership Committee, a Program Committee, a Necrology Committee and such other committees as may in its opinion be necessary or desirable. All such committees shall render their reports at an executive session of the Association, except that no *ad hoc* committee need report unless so directed by the Council.

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

Section 3. The Program Committee shall consist of at least nine members: the President, the Vice President, the Secretary and at least six members-at-large, three representing each of the areas of adult cardiac, pediatric cardiac and general thoracic surgery. Three of these members-at-large shall be appointed each year by the President for a three-year term. Additional committee members shall be appointed for one-year terms. The Editor shall serve as an ex-officio member of the Committee without vote. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

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

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

Section 6. The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such

Committees shall not exceed such annual or special appropriations as may be specifically voted for such purposes by the Association as a whole. Members are urged to cooperate with all Scientific or Research Committees of the Association.

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

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

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

Section 10. The Thoracic Surgical Workforce Committee shall be a Joint Committee of this Association and The Society of Thoracic Surgeons. The Committee shall consist of two members of this Association, two members of The Society of Thoracic Surgeons, and a Chairman who shall be a member of this Association and The Society of Thoracic Surgeons. The duties of this Committee, and the manner of appointment and term of its members and chairman, shall be determined jointly by the Council of this Association and the Council of The Society of Thoracic Surgeons.

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

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

ARTICLE VII. Finances

Section 1. The fiscal year of the Association shall begin on the first day of January and end on the last day of December each year.

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

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

Section 4. Funds derived from the payment of initiation fees shall not be available to current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact.

ARTICLE VIII. Meetings

Section 1. The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council and the provisions of the By-Laws.

Section 2. Notice of any meeting of the Association shall be given to each member of the Association not less than five nor more than forty days prior to any annual meeting and not less than thirty nor more than forty days prior to any special meeting by written or printed notice delivered personally or by mail, by or at the direction of the Council, the President or the Secretary. Such notice shall state the place, day and hour of the meeting and in the case of a special meeting shall also state the purpose or purposes for which the meeting is called.

Section 3. A special meeting of the Association may be called by the Council or on the written request of fifteen members delivered to the Council, the President or the Secretary. The specific purposes of the meeting must be stated in the request.

Section 4. Attendance at annual meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members, but it shall be expected from all Active Members.

Section 5. Each annual meeting shall have at least two executive sessions.

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

1. Appointment of necessary committees.
2. Miscelleneous business of an urgent nature.

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

1. Reading or waiver of reading of the minutes of the proceeding meetings of the Association and the Council.
2. Report of the Treasurer of the last fiscal year.
3. Audit Report.
4. Report of the Necrology Committee.
5. Report of the Program Committee.
6. Action on amendments to the Articles of Incorporation and By-Laws, if any.
7. Action on recommendations emanating from the Council.
8. Unfinished Business.
9. New Business.
10. Report of the Membership Committee.
11. Election of new members.
12. Report of the Nominating Committee.

13. Election of officers.

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

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

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

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

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

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

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

ARTICLE IX. Indemnification and Directors and Officers

Section 1. The Association shall indemnify any and all of its Councilors (hereinafter in this Article referred to as "directors") or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against expenses actually and necessarily incurred by them in connection with the defense or settlement of any action, suit or proceeding in which they, or any of them, are made parties, or a party, by reason of being or having been directors or officers or a director or officer of the Association, or of such other corporation or association, provided, however, that the foregoing shall not apply to matters as to which any such director or officer or former director or officer or person shall be adjudged in such action, suit or proceeding to be liable for willful misconduct in the performance of duty or to such matters as shall be settled by agreement predicated on the existence of such liability.

Section 2. Upon specific authorization by the Council, the Association may purchase and maintain insurance on behalf of any and all of its directors or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against any liability or settlement based on asserted liability, incurred by them by reason of being or having been directors or officers as a director or officer of the Association or of such other corporation or association, whether or not the Association would have the power to indemnify them against such liability or settlement under the provisions of Section 1.

ARTICLE X. Papers

Section 1. All papers read before the Association shall become the property of the Association. Authors shall leave original copies of their manuscripts with the Editor or reporter, at the time of presentation, for consideration for publication in the official Journal.

Section 2. When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions.

ARTICLE XI. Initiation Fees, Dues and Assessments

Section 1. Honorary Members of the Association are exempt from all initiation fees, dues, and assessments.

Section 2. Annual dues for Active Members shall be $200.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Senior Members are exempt from dues.

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

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

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

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

ARTICLE XII. Parliamentary Procedure

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

ARTICLE XIII. Amendments

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

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

As amended, April, 1995

Meetings of the American Association for Thoracic Surgery

1918-Chicago..... President, Samuel J. Meltzer
1919-Atlantic City..... President, Willy Meyer
1920-New Orleans..... President, Willy Meyer
1921-Boston..... President, Rudolph Matas
1922-Washington..... President, Samuel Robinson
1923-Chicago..... President, Howard Lilienthal
1924-Rochester, Minn..... President, Carl A. Hedblom
1925-Washington..... President, Nathan W. Green
1926-Montreal..... President, Edward W. Archibald
1927-New York..... President, Franz Torek
1928-Washington..... President, Evarts A. Graham
1929-St. Louis..... President, John L. Yates
1930-Philadelphia..... President, Wyman Whittemore
1931-San Francisco..... President, Ethan Flagg Butler
1932-Ann Arbor..... President, Frederick T. Lord
1933-Washington..... President, George P. Muller
1934-Boston..... President, George J. Heuer
1935-New York..... President, John Alexander
1936-Rochester, Minn..... President, Carl Eggers
1937-Saranac Lake..... President, Leo Eloesser
1938-Atlanta..... President, Stuart W. Harrington
1939-Los Angeles..... President, Harold Brunn
1940-Cleveland..... President, Adrian V. S. Lambert
1941-Toronto..... President, Fraser B. Gurd
1944-Chicago..... President, Frank S. Dolley
1946-Detroit..... President, Claude S. Beck
1947-St. Louis..... President, I. A. Bigger
1948-Quebec..... President, Alton Ochsner
1949-New Orleans..... President, Edward D. Churchill
1950-Denver..... President, Edward J. O'Brien
1951-Atlantic City..... President, Alfred Blalock
1952-Dallas..... President, Frank B. Berry
1953-San Francisco..... President, Robert M. Janes
1954-Montreal..... President, Emile Holman
1955-Atlantic City..... President, Edward S. Welles
1956-Miami Beach..... President, Richard H. Meade
1957-Chicago..... President, Cameron Haight
1958-Boston..... President, Brian Blades
1959-Los Angeles..... President, Michael E. De Bakey
1960-Miami Beach..... President, William E. Adams
1961-Philadelphia..... President, John H. Gibbon, Jr.
1962-St. Louis..... President, Richard H. Sweet (Deceased 1-11-62)
..... President, O. Theron Clagett
1963-Houston..... President, Julian Johnson
1964-Montreal..... President, Robert E. Gross
1965-New Orleans..... President, John C. Jones
1966-Vancouver, BC..... President, Herbert C. Maier
1967-New York..... President, Frederick G. Kergin
1968-Pittsburgh..... President, Paul C. Samson
1969-San Francisco..... President, Edward M. Kent
1970-Washington, DC..... President, Hiram T. Langston

1971-Atlanta..... President, Thomas H. Burford
1974-Las Vegas..... President, Lyman A. Brewer, III
1975-New York..... President, Wilfred G. Bigelow
1976-Los Angeles..... President, David J. Dugan
1977-Toronto..... President, Henry T. Bahnson
1978-New Orleans..... President, J. Gordon Scannell
1979-Boston..... President, John W. Kirklin
1980-San Francisco..... President, Herbert Sloan
1981-Washington, DC..... President, Donald L. Paulson
1982-Phoenix, Arizona..... President, Thomas B. Ferguson
1983-Atlanta..... President, Frank C. Spencer
1984-New York..... President, Dwight C. McGoon
1985-New Orleans..... President, David C. Sabiston
1986-New York..... President, James, R. Malm
1987-Chicago..... President, Norman E. Shumway
1988-Los Angeles..... President, Paul A. Ebert
1989-Boston..... President, W. Gerald Austen
1990-Toronto..... President, F. Griffith Pearson
1991-Washington, DC..... President, Keith Reemtsma
1992-Los Angeles..... President, John A. Waldhausen
1993-Chicago..... President, John L. Ochsner
1994-New York..... President, Aldo R. Castaneda
1995-Boston..... President, Robert B. Wallace
1996-San Diego..... President, Mortimer J. Buckley
1997-Washington, DC..... President, David B. Skinner

GRAHAM EDUCATION AND RESEARCH FOUNDATION

13 Elm Street, Manchester, Massachusetts 01944, (978) 526-8330
President James L. Cox, M.D., Washington, DC
Vice President Andrew S. Wechsler, M.D., Philadelphia, Pennsylvania
Secretary-Treasurer William T. Maloney, Manchester, Massachusetts
Director Hillel Laks, M.D., Los Angeles, California

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, 43 young surgeons from 23 countries have completed their training at thoracic surgical centers.

1st	1951-52	L.L. Whytehead Winnepeg, Manitoba, CANADA
2nd	1953-54	W.B. Ferguson Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley London, ENGLAND
4th	1955-56	Raymond L. Hurt Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth London, ENGLAND

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

33rd	1983-84	Georg S. Kobinia Klagenrurt, AUSTRIA
34th	1984-85	Aram Smolinsky Tel Hashomer, ISRAEL
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, PEOPLE'S REPUBLIC OF CHINA
39th	1989-90	Christopher John Knott-Craig Cape Town, SOUTH AFRICA
40th	1991-92	Ko Bando Okayama, JAPAN
41st	1992-93	Timothy E. Oaks Hershey, PA, USA
42nd	1993-94	Alain E. Serraf Le Plessis Robinson, FRANCE
43rd	1995-96	Cornelius McKown Dyke Richmond, VA, USA
44th	1996-97	Monica Robotin-Johnson Sydney, AUSTRALIA
45th	1997-98	Jun Wang Beijing, PEOPLE'S REPUBLIC OF CHINA
46th	1998-99	Christian Kreutzer Buenos Aires, ARGENTINA

THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION ORGANIZATION

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

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

The mission of The Foundation is to: support research and education initiatives to increase knowledge and enhance treatment of patients with cardiothoracic diseases; develop the skills of cardiothoracic surgeons as surgeon-scientists and health policy leaders; and, strengthen society's understanding and trust in the profession.

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Frank Kurtz, PhD Amy Hedmark

Executive Director Development Associate

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. Magovern, MD

Development Committee
All fund raising activities of The Foundation are monitored and coordinated by this committee.

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

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

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

Martin F. McKneally, MD

Education Committee
This committee organizes health care policy education for cardiothoracic 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 Robert B. Wallace, MD, *Chairman*

James L. Cox, MD Delos M. Cosgrove, III, MD

Martin F. McKneally, MD Robert W. Jamplis, 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 Winfieid J. Wells, 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*

Joint Committee on Allocations for Research and Education Programs
This committee determines 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

Major Gifts Subcommittee
This committee will be responsible for developing, publicizing and soliciting planned gifts to The Foundation.

Leonard L. Bailey, MD F. 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*

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

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 Robert W. Jamplis, MD

Arthur C. Beall, Jr., MD Sidney Levitsky, MD

John H. Bell, MD Alex G. Little, MD

Lawrence I. Bonchek, MD Christopher T. Maloney, MD

Lawrence H. Cohn, MD James B.D. Mark, MD

Robbin Gerald Cohen, MD Robert M. Mentzer, MD

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

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

George L. Hicks, Jr., 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.

Bartley P. Griffith, MD, *Chairman* Valerie W. Rusch, MD

Larry R. Kaiser, MD David H. Sachs, MD

Lynda L. Mickleborough, MD Julie A. Swain, MD

G. Alexander Patterson, MD Ross M. Ungerleider, MD

D. Glenn Pennington, MD

THE THORACIC SURGERY FOUNDATION AWARDS

Note: Recipients of the AATS Graham Foundation are listed on page 318

1998 Research 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.

Abbas El-Sayad Abbas, MD, University of Pennsylvania
Daniel Kreisel, MD, University of Pennsylvania
Paul C. Lee, MD, University of Pittsburgh
Michael A. Smith, MD, Washington University
Vinod H. Thourani, MD, Emory University School of Medicine
Tomasz A. Timek, MD, Stanford University

Nina S. Braunwald Research Fellowship

Provides salary support to women who wish to acquire investigation skills.

Melina R. Kibbe, MD, University of Pittsburgh
Elizabeth N. Morgan, MD, University of Washington

1998 Education Award Recipients

* Alley-Sheridan Executive Course Scholarships
* Alley-Sheridan Scholar-in-Residence

1998-99 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. Two individuals were awarded scholarships in 1998.
Edward J. Dunn, MD, Milwaukee, WI
Edgar L. Feinberg, III, MD, Lafayette, LA

Previous Research Award Recipients

The Thoracic Surgery Foundation Research Fellowship

Edward M. Boyle, Jr., MD, The University of Washington
Seth Force, MD, The University of Pennsylvania
Julie R. Glasson, MD, Stanford University School of Medicine
Joseph H. Gorman, III, MD, Hospital of the University of Pennsylvania
Robert S. Poston, Jr., MD, Stanford University Medical Center
Andrew J. Sherman, MD, Northwestern University Medical School

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.
Richard P. Embrey, MD, The Medical College of Virginia
Joren C. Madsen, MD, Massachusetts General Hospital
John D. Mannion, MD, Thomas Jefferson University
Si M. Pham, MD, The University of Pittsburgh
Todd K. Rosengart, MD, The New York Hospital - Cornell Medical Center
David S. **Schrump, MD,** National Cancer Institute

Nina S. Braunwald Career Development Award provides salary support to women in academic cardiothoracic surgery at early stages of their faculty careers.
Margaret D. Allen, MD, University of Washington School of Medicine
Mary C. Mancini, MD, Louisiana State University Medical Center
Patricia A. Thistlethwaite, MD, University of California - San Diego

Nina S. Braunwald Research Fellowship
Elaine E. Tseng, MD, Johns Hopkins Hospital
Jennifer Dale Walker, MD, Medical University of South Carolina

Previous Education Award Recipients

Alley-Sheridan Scholar-in-Residence at Harvard
Peter P. McKeown, MD, Tampa, Florida
Joseph J. McNamara, MD, Honolulu, Hawaii
Paul N. Uhlig, MD, Wichita, Kansas

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 cardio-thoracic 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: An 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, MD, Washington, DC
Richard P. Embrey, MD, Richmond, VA
Timothy J. Gardner, MD, Philadelphia, PA
Keith S. Naunheim, MD, St. Louis, MO
Anthony Louis Picone, MD, Syracuse, NY
Keith Eric Sommers, MD, Pittsburgh, PA
Clifford H. Van Meter, MD, New Orleans, LA

April. 1997 Alley-Sheridan Executive Course Scholars
Aurelio Chaux, MD, Los Angeles, CA
Stanley W. Dziuban, Jr, MD, Albany, NY
Steven R. Hazelrigg, MD, Springfield, IL
Bruce M. Toporoff, MD, New Orleans, LA

November. 1997 Alley-Sheridan Executive Course Scholars
Daniel P. Harley, MD, Baltimore, MD
Robert S.D. Higgins, MD, Detroit, MI
Mitchell J. Magee, MD, Springfield, IL
Peter C. Pairolero, MD, Rochester, MN
Joe B. Putnam, MD, Houston, TX
Thomas R.J. Todd, MD, Toronto, Ontario, Canada
Daniel W. van Heeckeren, MD, Cleveland, OH
Henry L. Walters, III, MD, Detroit, MI

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY RESEARCH SCHOLARSHIP

The American Association for Thoracic Surgery Research Scholarship was established by the Association in 1985. Funded by the Association and individual contributions, the Research

Scholarship provides opportunity for research, training and experience for North American surgeons committed to pursuing an academic career in cardiothoracic surgery. Administered by the Graham Education and Research Foundation, the program is undertaken within the first three years after completion of an approved cardiothoracic residency and is about two years in duration.

EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

1986-1988 Mark K. Ferguson

University of Chicago, Department of Surgery

ALFRED BLALOCK RESEARCH SCHOLARSHIP

"Efficacy and Toxiciry 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"

1996-1998 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

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

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 Lishan Aklog

West Roxbury, MASSACHUSETTS

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 honoree's name and biography is printed in the *Journal of Thoracic and Cardiovascular Surgery.*

SCIENTIFIC ACHIVEMENT AWARD RECIPIENTS

1995 - John W. Kirklin, Birmingham, Alabama

1998 -Norman E. Shumway, Stanford, California