

1993 ANNUAL MEETING PROGRAM



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FOR THORACIC SURGERY
1993 Annual Meeting**

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POSTGRADUATE COURSE

<p>AATS</p> <p>Postgraduate</p> <p>Course</p> <p>Congenital</p> <p>Heart</p> <p>Disease</p> <p>Sunday, April 25, 1993</p> <p>8:00 a.m. - 4:00 p.m.</p> <p>Hyatt Regency Hotel</p> <p>Chicago, Illinois</p>	<p>Objectives</p> <p>The 1993 Postgraduate Course on Congenital Heart Disease will address the following three topics: Systemic Outflow Tract Obstruction in the Neonate, Tetralogy of Fallot with Pulmonary Atresia and Aortopulmonary Systemic Collaterals, and Allografts and Autografts. This course will provide attendees with the opportunity to interact with recognized experts involved in the research and development of new techniques and procedures in congenital heart disease. The format of the course will include lectures on current issues within each of the three topic areas, with ample time provided during each session for discussion of specific questions from the audience.</p> <p>Registration</p> <p>Enrollment in this course will be by preregistration until March 26, 1993. Registration forms will be processed in the order in which they are received and must be accompanied by payment in full. After March 26, 1993, participants may register on site. The registration fee is \$35.00 and includes the PG course, coffee break, and lunch.</p> <p>Accreditation</p> <p>The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing education for physicians. The American Association for Thoracic Surgery designates this continuing medical education activity for 6 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.</p>
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SUNDAY, APRIL 25, 1993

Program - Grand Ballroom B

I. SYSTEMIC OUTFLOW TRACT OBSTRUCTION IN THE NEONATE

Moderators: Edward L. Bove, M.D., Ann Arbor, Michigan

Robert M. Sade, M.D., Charleston, South Carolina

8:00 a.m. PREOPERATIVE DETERMINATION OF LEFT VENTRICULAR SIZE: WHEN IS THE LEFT HEART TOO SMALL?

Robert H. Beekman HI, M.D., Ann Arbor, Michigan

8:15 a.m. SUBAORTIC STENOSIS WITH INTERRUPTED ARCH OR COARCTATION

Michel N. Ilbawi, M.D., Chicago, Illinois

8:30 a.m. TRANSPOSITION OF THE GREAT ARTERIES WITH OUTLET CHAMBER

Aldo R. Castaneda, M.D., Boston, Massachusetts

8:45 a.m. STAGED RECONSTRUCTION FOR HYPOPLASTIC LEFT HEART SYNDROME

Edward L. Bove, M.D., Ann Arbor, Michigan

9:00 a.m. CRITICAL AORTIC STENOSIS IN THE NEONATE

Lee Benson, M.D., Toronto, Ontario

9:15 a.m. DISCUSSION

10:00 a.m. REFRESHMENT BREAK

II. TETRALOGY OF FALLOT WITH PULMONARY ATRESIA AND AORTOPULMONARY SYSTEMIC COLLATERALS

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

Gary Lofland, M.D., Richmond, Virginia

10:30 a.m. ANATOMY OF SYSTEMIC COLLATERALS AND PULMONARY ARTERIES

Marlene Rabinovitch, M.D., Toronto, Ontario

10:45 a.m. INITIAL SURGICAL MANAGEMENT

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

11:00 a.m. ROLE OF INTERVENTIONAL CARDIOLOGY

Larry A. Latson, M.D., Cleveland, Ohio

11:15 a.m. RESULTS OF COMPLETE REPAIR

John W. Kirklin, M.D., Birmingham, Alabama

11:30 a.m. DISCUSSION

12:00 noon LUNCHEON

III. ALLOGRAFTS AND AUTOGRAFTS/MISCELLANEOUS TOPICS

Moderators: David R. Clarke, M.D., Denver, Colorado

Thomas Spray, M.D., St. Louis, Missouri

1:30 p.m. CELLULAR AND IMMUNOLOGIC CHARACTERISTICS OF ALLOGRAFTS

Flavian M. Lupinetti, M.D., Ann Arbor, Michigan

1:45 p.m. AORTIC VALVE REPLACEMENT WITH AUTOGRAFTS

Ronald C. Elkins, M.D., Oklahoma City, Oklahoma

2:00 p.m. FATE OF ALLOGRAFTS IN THE PULMONARY CIRCULATION

David R. Clarke, Denver, Colorado

2:15 p.m. DISCUSSION

2:45 p.m. STRESS RESPONSE IN INFANTS DURING AND AFTER CARDIAC SURGERY
Paul Hickey, M.D., Boston, Massachusetts

3:00 p.m. INTERRUPTED AORTIC ARCH AND COARCTATION: CHSS UPDATE

John W. Kirklin, M.D., Birmingham, Alabama

3:15 p.m. ETHICAL CONSIDERATIONS: WHOSE LIFE IS IT ANYWAY?

Robert M. Sade, M.D., Charleston, South Carolina

3:30 p.m. DISCUSSION

4:00 p.m. MEETING ADJOURNS

DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

<p>AATS</p> <p>Symposium</p> <p>Blood</p> <p>Transfusion</p> <p>Practices</p> <p>in</p> <p>Cardiac</p> <p>Surgery</p> <p>Sunday, April 25, 1993</p> <p>1:30 p.m. - 5:30 p.m.</p> <p>Hyatt Regency Hotel</p> <p>Chicago, Illinois</p>	<p>Objectives</p> <p>The symposium will present the coagulation and fibrinolytic changes associated with cardiopulmonary bypass, and the underlying physiologic mechanisms. Strategies to minimize these changes, and reduce the need for blood transfusions and blood products will be discussed. These include: On site monitoring of microvascular bleeding intraoperatively, the use of autologous transfusion, transfusion of shed blood preoperatively, and the use of Erythropoietin. Pharmacologic control of bleeding using Epsilon-Aminocaproic Acid, DD/AVP, Tranexamic Acid, and Aprotinin will be presented. Discussion will focus on areas of controversies and the sequential management of the bleeding cardiac surgery patient.</p> <p>Registration</p> <p>Enrollment in this course will be by preregistration until March 26, 1993. Registration forms will be processed in the order in which they are received and must be accompanied by payment in full. After March 26, 1993, participants may register on site. The registration fee is \$20.00.</p> <p>Accreditation</p> <p>The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing education for physicians. The American Association for Thoracic Surgery designates this continuing medical education activity for 3.5 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.</p>
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Program - Grand Ballroom F

SESSION I BALANCING BLOOD TRANSFUSION AND BLOOD CONSERVATION

Moderator: Safuh Attar, M.D., Baltimore, Maryland

1:30 p. m. Introduction:

THE SCOPE OF THE PROBLEM: BLOOD TRANSFUSIONS, SAFETY, RISKS, BENEFITS
AND COSTS

Safuh Attar, M.D., Baltimore, Maryland

1:40 p.m. CURRENT TRANSFUSION PRACTICE IN CARDIAC SURGERY

Lawrence Tim Goodnough, M.D., St. Louis, Missouri

2:00 p.m. SHOULD PREOPERATIVE AUTOLOGOUS BLOOD DONATIONS BE ROUTINE BEFORE CARDIAC SURGERY?

Robert L. Thurer, M.D., Boston, Massachusetts

2:20 p.m. THE ACQUIRED HEMOSTASIS DEFECTS RELATED TO EXTRACORPOREAL CIRCULATION AND THEIR EFFECT ON BLOOD LOSS

L. Henry Edmunds, Jr., M.D., Philadelphia, Pennsylvania

2:40 p.m. ON-SITE COAGULATION MONITORING: THE IMPACT ON INTRAOPERATIVE HEMOSTASIS AND TRANSFUSION NEEDS

George J. Despoils, M.D., Kirkwood, Missouri

3:00 p.m. COFFEE BREAK

SESSION II PHARMACOLOGIC APPROACHES TO REDUCE BLOOD TRANSFUSION REQUIREMENTS

Moderator: Lawrence Tim Goodnough, M.D., St. Louis, Missouri

3:30 p.m. RECOMBINANT HUMAN ERYTHROPOIETIN THERAPY

Michael D'Ambra, M.D., Boston, Massachusetts

3:50 p.m. APROTININ THERAPY IN AORTO-CORONARY BYPASS

Benjamin P. Bidstrup, FRACS, FRCSEd

London, England

4:10 p.m. APROTININ THERAPY: BLOOD LOSS, BLOOD TRANSFUSION, GRAFT PATENCY

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

4:30 p.m. DDAVP AND TRANEXAMIC ACID THERAPY

John W. Mammon, Jr., M.D.

Winston-Salem, North Carolina

4:50 p.m. PANEL DISCUSSION: IS BLOODLESS SURGERY A REALISTIC GOAL?

5:30 p.m. MEETING ADJOURNS

**American Association for
Thoracic Surgery
73RD ANNUAL MEETING
Hyatt Regency Hotel, Chicago, IL
APRIL 25-28, 1993**

MONDAY MORNING, APRIL 26, 1993

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

8:45 a.m. SCIENTIFIC SESSION - Grand Ballroom

Moderators: John L. Ochsner, M.D.

Martin F. McKneally, M.D.

1. Retransplantation in Heart-Lung Recipients With Obliterative Bronchiolitis

*DAVID H. ADAMS, M.D. *, ANDREW D. COCHRANE,*

FRACS, ASGHAR KHAGHANI, FRCS* and*

MAGDI H. YACoub, FRCS

Harefield, United Kingdom

Obliterative bronchiolitis remains the leading cause of morbidity and mortality in long term surviving heart-lung recipients. Despite enhanced immunosuppressive therapy, a significant number of patients progress to end stage respiratory failure leaving retransplantation as the only therapeutic option. Between October 1986 and September 1992, 33 heart-lung recipients with Obliterative bronchiolitis (80% ventilator dependent) have undergone retransplantation (range: 9-83 months; mean: 27 months after the first operation). Twenty-four patients underwent repeat heart-lung transplantation. Post-operative complications included bleeding, multi-system organ failure, and infection. Twelve patients (50%) died in <30 days, and 7 patients (29%) survived >1 year. Four patients are currently alive and well (survival range 33-67 months).

Recently, we have investigated the role of single lung retransplantation in 9 heart-lung recipients with Obliterative bronchiolitis. One patient died in <30 days, and thus far 4/7 patients (57%) have survived > 1 year. Five patients are currently alive (survival range 2-23 months) and 3 patients have returned to full-time employment.

Retransplantation in heart-lung recipients with Obliterative bronchiolitis is a high risk procedure, but it can result in rehabilitation of otherwise incapacitated patients. Single lung retransplantation appears to be the preferred option in carefully selected patients.

*By Invitation

2. Current Results and Indications of Single, Bilateral and Heart and Lung Transplantation for Pulmonary Hypertension

*KO BANDO, M.D. *, ROBERT J. KEENAN, M.D. *,*

*IRVIN L. PARADIS, M.D. *, JOHN M. ARMITAGE M.D. *,*

*KEITH L. STEIN, M.D. *, ROBERT L. HARDESTY, M.D.,*

HENRY T. BAHNSON, M.D. and

BARTLEY P. GRIFFITH, M.D.

Pittsburgh, Pennsylvania

The indications for single (SLT), bilateral (BLT) and heart and lung transplantation (HLT) in pulmonary hypertension (PH) remain controversial. We retrospectively analyzed the results from 10 SLT, 21 BLT and 21 HLT performed between January 1989 and August 1992 on 52 consecutive patients with PH due to primary pulmonary hypertension (n = 24), Eisenmenger's syndrome (ES) (n = 25), and CREST syndrome (n = 3). There were no differences among the 3 allograft groups (SLT, BLT and HLT) in age, gender, pre-operative (pre-op) pulmonary arterial pressure (PAP) or pre-op NYHA functional class. HLT was performed for PH with left ventricular (LV) dysfunction defined as an LV ejection fraction (EF) <35% determined by MUGA scan and for ES with complex congenital heart disease (CHD), ES with atrial septal defect (ASD) or patent ductus (PDA) was treated by isolated lung transplantation with cardiac repair (PDA closure: SLT(1), BLT(7); ASD closure: SLT(1), BLT(2). Early after transplantation (mean: post-op 4 weeks), all groups demonstrated significant hemodynamic improvement compared to before transplantation as follows (mean \pm SD, *p<.05 vs pre-op by paired T test).

	SLT (n = 10)		BLT (n = 21)		HLT (n = 21)	
	pre	post	pre	post	pre	post
Cardiac Index						
(L/min/m ²)	1.9 \pm 1.0	2.3 \pm 0.4	2.2 \pm 0.6	2.7 \pm 0.3*	2.1 \pm 0.6	2.6 \pm 0.3*
Systolic PAP						
(mmHg)	103.5 \pm 26.1	34.8 \pm 6.2	103.7 \pm 18.1	35.3 \pm 6.4*	99.4 \pm 28.0	25.7 \pm 5.5*
Right ventricle						
(RV EF (%))	31.4 \pm 9.8	49.0 \pm 7.2	28.3 \pm 16.1	46.1 \pm 16.1*	29.7 \pm 12.3	51.3 \pm 10.9*
LVEF (<%)	55.3 \pm 12.5	65.8 \pm 8.9	60.0 \pm 11.3	68.9 \pm 8.7*	35.1 \pm 13.2	61.3 \pm 9.6*

Post-op ventilation/perfusion (V/Q) scans (mean: 18 weeks, values expressed as % \pm SD) demonstrated significant V/Q mismatch in SLT allografts (V: 35.0 \pm 12.0/Q: 85.2 \pm 5.0 to allograft) whereas no V/Q mismatch was present in the BLT and HLT recipients (p<.05 SLT vs BLT & HLT by analysis of variance). Operative mortality was similar among the allograft groups (SLT:20%; BLT & HLT: 14%; p = NS). SLT pts experienced the lowest one year survival (SLT:40%; BLT:67%; HLT:70%; p<.05 by life table analysis) and symptomatic recovery (mean post-op NYHA class: SLT:2.2*; BLT:1.1; HLT:1.0; *p<.05 by Kruskal-Wallis statistic).

Conclusion: In spite of encouraging early hemodynamic improvement, SLT for PH is associated with significantly decreased late survival and poor functional outcome when compared to BLT and HLT. We conclude that BLT is a more satisfactory option for PH in pts with preserved

LV function. Pts with PH and severe LV dysfunction or ES combined with complex CHD still require HLT.

*By Invitation

3. Pediatric Lung Transplantation: Indications, Techniques and Early Results

THOMAS L. SPRAY, M.D., GEORGE B. MALLORY,

*M.D. *, CHARLES B. CANTER, M.D. * and*

*CHARLES B. HUDDLESTON, M.D. **

St. Louis, Missouri

Improvement in the results of adult lung transplantation (LTX) for end-stage pulmonary disease has led to application of these techniques to the pediatric population. From 7/90 to 9/92 30 LTX (21 bilateral sequential, 6 single) in 27 patients have been performed in our pediatric transplant program (1.5 - 23 years, mean age 10.6 years). Six children had been on continuous ventilator support for 18 days to 4.5 years prior to LTX and 2 were on extracorporeal membrane oxygenation. Indications for LTX in this pediatric population include: cystic fibrosis (N = 10), pulmonary hypertension and associated congenital heart disease (N = 7), pulmonary atresia, ventricular septal defect (VSD), and nonconfluent pulmonary arteries (N = 3), pulmonary fibrosis (N = 6), and acute respiratory distress syndrome (ARDS) (N = 1). Three children underwent retransplantation for acute graft failure (N = 2) or chronic rejection (N = 1). Pulmonary fibrosis was related to treatment of acute of myelogenous leukemia with bone marrow transplantation in two children and to bronchiolitis obliterans, bronchopulmonary dysplasia, interstitial pneumonitis, and Histiocytosis-X. Ten children underwent LTX and concomitant cardiac repair. Bilateral LTX, VSD closure and pulmonary homograft reconstruction of the right ventricular (RV) outflow tract to the transplanted lungs was performed in 3 children utilizing a new technique which avoids the need for combined heart/lung transplantation. Two patients had VSD closure and LTX for Eisenmenger's syndrome and 2 had ligation of a patent ductus arteriosus and LTX. Three additional children underwent atrial septal defect closure and LTX. There have been 7 early deaths (26%) and 3 late deaths (sepsis - 2, hemorrhage - 1, ARDS - 2, lymphoproliferative disease - 2, bronchiolitis obliterans (OB) - 2, and pseudoaneurysm of the RV - 1). Bronchial complications were seen in 7 of 53 anastomoses at risk (13%) (disruption - 1, stenosis - 6), and were treated with pneumonectomy (1) or stent implantation (6).

LTX in children has been associated with acceptable early results, although modification of the adult implantation technique has been necessary. LTX and repair of complex congenital heart defects is possible; heart/lung transplantation may only be required for patients with severe left heart dysfunction and associated pulmonary vascular disease. OB remains a major concern for longterm graft function in the pediatric LTX patient.

9:45 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

10:30 a.m. SCIENTIFIC SESSION - Grand Ballroom

Moderators: Aldo R. Castaneda, M.D.

Martin F. McKneally, M.D.

4. Aortic Dissection: Is Elective Reoperation Advisable?

JEAN E. BACHET, M.D., JEAN LUC TERMIGNON,

*M.D. *, BERTRAND GOUDOT, M.D. *,*

*GILLES DREYFUS, M.D. *, ALAIN PIQUOIS M.D. **

*and DANIEL GUILMET, M.D. **

Suresnes, France

From January 1977 to June 1992, 140 patients (pts) underwent emergency surgery for Type A acute Aortic dissection. Because of the location of the intimal tear, the replacement of the ascending aorta was extended to the transverse arch in 41 pts (30%). One hundred and nine pts (88%) survived surgery. During the same period, 30 pts had to be reoperated on, once (23), twice (3) or three times (4) for a total of 41 reoperations. Seventeen pts had had the initial repair in our Institution, 13 pts had been operated on elsewhere. Reoperation was indicated for: Aortic valve disease (7), recurring dissection (10), threatening aneurysmal evolution of a persisting dissection (22) or infective false aneurysm (2). The re-do procedure involved: the aortic root and/or ascending aorta in 13 cases including 8 Bentall (Group I); the transverse arch alone in 6 cases (Group II); the transverse arch and Descending aorta in 8 cases (Group III) and the descending or Thoraco-abdominal aorta in 14 cases (Group IV). The risk-factors for reoperation have been analyzed in the 109 survivors initially operated on in our Institution. Six out of 18 Marfan pts (33%) versus 11 out of 91 non-Marfan pts (12%) were reoperated upon ($p = 0.023$). None of 30 pts surviving arch replacement at initial repair, required a reoperation, versus 17 out of 79 (21.5%) pts surviving a replacement limited to the ascending aorta ($p = 0.013$). The overall mortality rate of reoperation was 16% (5/30 pts) with a risk of 12% (5/41) at each procedure. (Group I: 0%; Group II: 0%; Group III: 12.5%; Group IV: 29%). Hospital mortality was influenced by emergency (4/5) ($p = 0.005$) and Thoraco-abdominal replacement (5/22) ($p = 0.035$). The late survival rate after reoperation is $53.3 \pm 9.9\%$ and $45.6 \pm 11\%$ at 5 and 7 years respectively. The late survival rate, after the initial repair, of the reoperated pts is $79.3 \pm 7.4\%$ and $63.6 \pm 10.2\%$ at 5 and 10 years, respectively.

In conclusion, aortic dissection is an evolving process that may require one or several reoperations after the initial repair. At initial emergency operation, the resection of the entry site, when located on or extending to the transverse arch, has reduced the risk of reoperation, in our experience. Elective reoperation must be considered before the occurrence of complications, especially in Marfan pts. It entails a relatively low risk, except in case of Thoraco-abdominal replacement and allows a satisfactory long-term survival rate.

*By Invitation

5. The Influence of Arterial Coronary Bypass Grafts on the Mortality of Coronary Reoperations

*BRUCE W. LYTLE, MD, DERRICK McELROY, M.D. *,*

*PATRICK McCARTHY, M.D. *, FLOYD D. LOOP, M.D.,*

ROBERT W. STEWART, M.D.* and

DELOS M. COSGROVE, M.D.

Cleveland, Ohio

During the years 1988 through 1991, 1663 patients underwent a first reoperation for isolated coronary bypass grafting with 58 (3.5%) in-hospital deaths. At the primary operation 575 patients had received at least one internal mammary artery (IMA) graft and 489 patients had at least one patent IMA graft present at the time of reoperation. At reoperation 1014 patients received at least one IMA graft, 10 received an inferior epigastric graft and 37 a gastroepiploic graft. Subgroups according to numbers of IMA grafts at primary operation, reoperation and in-hospital mortality rates are shown below.

IMA Grafts at Primary Operation	0	0	0	1	1	2
IMA Grafts at Reoperation	0	1	2	0	1	0
Total Patients	289	673	126	345	215	15
In-Hospital Mortality	21	16	1	15	4	1
Mortality Percent	7.2%	2.4%	0.8%	4.3%	1.8%	6.7%

Of 489 patients with patent IMA grafts at reoperation the IMA was damaged in 17 (3.5%)

and of 428 with a patent IMA graft to the left anterior descending coronary artery (LAD) 14

(3.3%) were damaged necessitating regrafting. All patients with damaged IMA's survived.

Multivariate testing of variables for their association with in-hospital mortality identified no IMA graft at either primary surgery or reoperation ($p < 0.0001$), advancing age ($p = 0.0046$) and female gender ($p = 0.036$) as factors linked to increased risk. Left ventricular function, left main stenosis, extent of native coronary atherosclerosis and the interval between operations did not influence mortality. Furthermore, the presence of a atherosclerotic vein graft to the LAD, a factor shown to increase in-hospital risk in previous studies, did not increase risk during these years. The observation that patent IMA and atherosclerotic vein grafts do not appear to be factors specifically increasing the risk of reoperation we attribute to the use of retrograde car-dioplegia and increased surgical experience.

The use of IMA grafts at a primary operation does not increase the risk of a reoperation and the use of IMA grafts at reoperation does not increase in-hospital mortality.

11:15 a.m. PRESIDENTIAL ADDRESS

"Giants: How and Why They Grew"

John L. Ochsner, M.D., New Orleans, Louisiana

*By Invitation

MONDAY AFTERNOON, APRIL 26, 1993

12:00 noon ADJOURN FOR LUNCH - VISIT EXHIBITS

1:30 p.m. SCIENTIFIC SESSION - Grand Ballroom

Moderators: Bruce A. Reitz, M.D.

William A. Gay, Jr., M.D.

6. Surgical Management of Neonatal Coarctation: A Study of 221 Patients

*FRANCOIS LACOUR-GAYET, M.D. *, STEFANO CONTE, M.D. *,*

*ALAIN SERRAF, M.D. *, JACQUELINE BRUNIAUX, M.D. *,*

*MIGUEL SOUSA-UVA, M.D. *,*

*and CLAUDE PLANCHE, M.D. **

Plessis Robinson, France

Sponsored by: Aldo Castaneda, M.D., Boston, Massachusetts

Optimal surgical management of neonatal coarctation remains controversial. We report our entire experience using a single surgical technique: the extended end to end anastomosis (EEEE). From Aug 83 to Aug 92, 221 consecutive neonates underwent coarctation repair with EEEA. Mean age and weight at operation were respectively 13 days \pm 8 and 3.1 kg \pm 0.5. Pre-operative conditions required Prostaglandin E, infusion in 66% and ven-tilatory support in 50%. The coarctation was isolated in 68 patients, associated with isolated VSD in 72 and associated with complex intracardiac anomalies in 81. The EEEA was performed through a left thoracotomy in 196 patients and through a sternotomy associated with intracardiac repair in 25.

- *In the group of isolated coarctation:* all patients were operated through left thoracotomy; arch hypoplasia rate was 60%. Early mortality and 5 years actuarial survival rates were respectively 2.9% and 96%.

- *In the group of coarctation with VSD,* 96% were operated through left thoracotomy. Arch hypoplasia rate was 71%. A pulmonary artery banding was associated in 41% (30/72). A spontaneous closure of the VSD occurred in 38%. Early mortality and 5 years actuarial survival rates were respectively 1.4% and 93%.

- *In the group of complex coarctation:* 74% were operated through left thoracotomy and 26% through sternotomy in a single stage associated with either a biventricular repair or a palliative procedure. Arch hypoplasia rate was 96%. Early mortality was 20%; eighteen secondary deaths occurred, all in relation with the associated lesions. The 5 years actuarial survival rate was 58% ($p < 0.001$).

Mean follow up was 35 months \pm 27, ranging from 0.2 to 10 years. Overall residual or recurrent coarctation rate was 11% (25/221), leading to 18 reoperations and 2 angioplasties. Overall actuarial survival rate at 5 years was 80% \pm 3%.

	Arch	Left	One stage	Recurrent	Early	5 years
Pts Groups	Hypoplasia	Thoraco.	Sternotomy	Coa	Mortality	Survival
Isolated CoA	41	68	0	9	2	96%

68 pts	60%	700%	0%	13%	2.9%	
CoA + VSD	51	69	3	9	1	93%
72 pts	71%	96%	4%	12.5%	1.4%	
Complex CoA	78	59	21	7	16	58%
81 pts	96%	73%	27%	8.6%	20%	
Total	170	196	25	25	19	80%
221 pts	77%	89%	11%	11%	8.6%	

Conclusion: Neonatal coarctation is safely managed by EEEA; long term prognosis is mainly related to associated cardiac lesions.

*By Invitation

7. Staged Operation for Pulmonary Atresia and Ventricular Septal Defect With Major Aortopulmonary Collateral Arteries: Complete Unifocalization Based on New Concept of Peribronchial Surgery

KAZUO SAWATARI, M.D.*, YASU HARU IMAI, M.D.,
TAKAMASA TAKEUCHI, M.D.*, YUKIHISA ISOMATU, M.D.*,
KOJIRO KODERA, M.D.*, MAKOTO NAKAZA WA, M.D.* and
KAZUO MOMMA, M.D.*

Tokyo, Japan

Since 1982, we have followed the protocol of staged operation for pulmonary atresia and ventricular septal defect (VSD) with major aorto-pulmonary collateral arteries (MAPCAs). In first-stage repair (unifocalization), intrapulmonary arteries were unified and associated peripheral pulmonary stenosis was released. In case of absent or severely hypoplastic central pulmonary arteries (PA Index<50), new central pulmonary arteries were created. Finally, the unifocalization was completed by modified Blalock-Taussig shunt with the ligation of MAPCAs. In second-stage repair, right ventricular-pulmonary arterial (RV-PA) continuity was established with the closure of VSD. From 1982 to October 1992, 70 patients, whose ages ranged from 1 month to 24 years (mean 5.5 years), underwent unifocalization. There were one early and two late deaths (mortality rate 4%). Isolated area from 1 segment to 2 lobes, being solely supplied from MAPCAs due to arborization abnormalities, was present in 48 patients (69%). In the primary series of 18 patients, intrapulmonary arteries were unified at the hilum with equine pericardial conduits (intrapulmonary bridges). In the recent series, however, unification of intrapulmonary arteries was successfully achieved by direct anastomoses avoiding the use of prosthetic conduits in the vicinity of intrapulmonary bronchi locating in the middle of hilum where isolated intrapulmonary arteries continuing from MAPCAs became in close proximity to hilar intrapulmonary arteries connecting to central pulmonary arteries. New central pulmonary arteries were created with equine pericardial conduits in 11 out of 16 patients with severely hypoplastic central pulmonary arteries and in 11 patients with absent central pulmonary arteries. Second-stage repair has been completed in 45 patients. The VSD was closed with a perforated patch in 9 patients with severe pulmonary hypertension or severely hypoplastic hilar intrapulmonary arteries. There were 3 early and 2 late deaths (mortality rate 11%). Postoperative right ventricular/left ventricular systolic pressure ratios (RVP/LVP) ranged from 0.36 to 1.00 (mean 0.61). There was no relationship between the size of central pulmonary arteries (PA Index) and postoperative RVP/LVP. We conclude that

unifocalization can be best achieved by direct anastomoses between hilar intrapulmonary arteries around intrapulmonary bronchi in the middle of hilum where essential deformities of arborization abnormalities exist. The majority of patients with MAPCAs can have successful repair by our unifocalization technique when hilar intrapulmonary arteries are of adequate size without severe pulmonary hypertensive change.

*By Invitation

8. Early and Late Results of Mitral Valve Repair in Children

ALON S. AHARON, M.D. *, HILLEL LAKS, M.D.,

DAVIS C. DRINKWATER, M.D., REEMA CHUGH, M.D. *,

RICHARD N. GATES, M.D. *, LESTER C. PERMUT, M.D. * and

ABBAS ARDEHALI, M.D. *

Los Angeles, California

Mitral valve repair in children has the advantage of avoiding MV replacement with its attendant need for anticoagulation and reoperation. Forty-seven children with congenital mitral regurgitation underwent mitral valve repair between May, 1982 and February, 1992. The group ranged in age from 6 months to 17 years (mean 5.2 years + / - 4.2) and excluded patients undergoing primary repair of newly diagnosed complete atrioventricular canal (AVC). Two patients with prior repair of AVC, and 1 patient with congenital mitral insufficiency and bacterial endocarditis were included in the study. Five patients had grade III and 18 patients had grade IV MR by preoperative echocardiography. Associated cardiac anomalies were present in 37 of 47 patients and 81% of the patients required concomitant intracardiac procedures. Associated lesions included: Single ventricle (30%), ASD (27%), VSD (15%), pulmonary atresia/stenosis (15%), TV atresia (11%), TAPVR (6%), dextrocardial (6%), TV insufficiency (4%), AS (4%), and transposition of the great vessels (2%). The methods of mitral valve repair included annuloplasty in 44/47 (94%), repair of the anterior leaflet in 18/47 (38%), cleft closure of the anterior leaflet in 12/47 (26%), chordal shortening in 9/47 (19%) and repair of the posterior leaflet in 5/47 (11%). The technique of annuloplasty was modified to allow annular growth. Follow up data was available from 1 to 8 years, (mean 4.0 years + / - 2.5). All patients since 1988 had intraoperative transesophageal echocardiograms. There were 2 (4%) early (<30 days) deaths. One after Fontan procedure and MV repair, and 1 after combined aortic valve replacement and MV repair. Three late deaths (6%) occurred in 6, 9 and 20 months postoperatively. All 3 patients had persistent moderate to severe MR. The actuarial survival rate was 94% at 8 years. Mitral valve repair failed in 5/47 (11%) patients who then required MVR. Two of these patients had required annuloplasty only. One patient underwent emergent MVR for flail anterior leaflet on postoperative day 0, 1 patient underwent MVR on postoperative day 7 for severe MR and CHF, and 3 patients underwent MVR 6, 14 and 48 months postoperatively for progressive, severe MR. Actuarial freedom from reoperation was 91 % after 2 years and 89% after 4, 6, and 8 years. One thromboembolic event (2%) occurred resulting in transient right sided paralysis in a 17 year old patient after combined mitral valve repair and AVR 18 months postoperatively, despite adequate anticoagulation. Ninety-seven percent of long term survivors were asymptomatic. All patients received postoperative echocardiograms, and 94% had minimal to no mitral regurgitation. We conclude that mitral reconstruction can be performed with low early and late mortality. The need for reoperation is relatively low and valve growth has occurred with the use of a modified annuloplasty.

2:30 p.m. BASIC SCIENCE LECTURE

"Molecular Biology: New Common Ground for Cardiothoracic Surgery"

Andrew S. Wechsler, M.D., Richmond, Virginia

3:15 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

4:00 p.m. SCIENTIFIC SESSION - Grand Ballroom

Moderators: Tom R. DeMeester, M.D.

William A. Gay, Jr., M.D.

9. Survival Related to Nodal Status After Sleeve Resection for Primary Lung Cancer

REZA MEHRAN, M.D. *, JEAN DESLAURIERS, M.D.,

LIU GUOJIN, M.D. *, MICHEL PIRAUX, M.D. * and

MAURICE BEAULIEU, M.D. *

Ste-Foy, Quebec, Canada

Sleeve lobectomy is a lung saving procedure indicated for central tumors for which the alternative is a pneumonectomy. At present time, the relationship between survival and nodal status is unclear because in most series, the presence of N₁ disease significantly worsens the prognosis with few or no long term survivors.

During the period 1972-1992, 142 patients underwent sleeve resection for lung cancer at our institution. Mean age was 60.7 ± 9.1 years (11-78). Indications for surgery were a central tumor in 112 patients (79%), a peripheral tumor in 18 patients (13%) and compromised pulmonary function in 12 patients. One hundred and twenty patients had pre-operative mediastinoscopy which was negative in all but 6 patients. The general characteristics of the study population and the survival are shown in the table.

Pathological nodal	No	N ₁	PN ₀ -N ₁	N ₂	Total
Status					
Number of patients	73 (51.4%)	55 (38.7%)	-	14	142
Histology					
* Squamous	55 (75.3%)	43 (78.2%)	NS	5	103 (72.5%)
* Non squamous	14 (19.2%)	12 (21.8%)		9	35 (24.6%)
* Carcinoid	4	-		-	4
"T" status					
* T ₁	6	5		1	12
* T ₂	55 (75.3%)	43 (78.2%)	NS	9	107 (75.3%)
* T ₃₋₄	12	7		4	23
Extent of Surgery					
* Complete	67 (91.8%)	51 (92.7%)	NS	6	124 (87%)
* Incomplete	6	4		8	18

Survival

* Median	2931 days	1433 days	NS (0.12)	560 days	11 52 days
* 3 years	67%	58%		7%	57%
* 5 years	57%	46%	NS	0%	46%
* 10 years	44%	27%		0%	33%

The operative mortality was 2.1% (3/142). Follow-up was complete for the 139 remaining patients and there was no significant difference in survival between patients with N₀ or N₁ status. The incidence of local recurrence was also not significantly different between these two groups (N₀: 16.4%, N₁: 21.8%). Among the 14 patients with N₂ disease, none survived 5 years.

This data suggests that sleeve resection is a very adequate cancer operation for patients with No-N₁ status who can have complete resection of their tumor. The presence of N₂ disease significantly worsens the prognosis and does not justify the use of the procedure.

*By invitation

10. Segmentectomy vs. Lobectomy in Patients With Stage I Pulmonary Carcinoma: Five Year Survival and Patterns of Intrathoracic Recurrence

WILLIAM H. WARREN, M.D. and

L. PEN FIELD FABER, M.D.

Chicago, Illinois

From 1980-87, 73 patients had a segmentectomy and 112 patients had a lobectomy for Stage I (T1NO, T2NO) primary pulmonary carcinoma. Patients with a previous primary malignancy, incomplete staging and/or incomplete resections were excluded. Patients were followed for 5 years for survival and for pattern of recurrent tumor. Recurrent intrathoracic carcinoma was defined as recurrence of carcinoma in the ipsilateral or contralateral hemithorax without regard to time interval, precise location within the hemithorax, or histology. No attempt was made to distinguish local recurrence from solitary metastasis or second primary tumor.

The 5 year survival in the 2 groups was not statistically different ($p>0.05$). However, patients undergoing a segmentectomy had a 24.7% incidence of ipsilateral recurrence vs. 4.1% contralateral recurrence. In contrast, patients undergoing a lobectomy had an 8.9% incidence of ipsilateral recurrence vs. 3.5% contralateral recurrence. This higher incidence of ipsilateral recurrence among segmentectomies ($p>0.01$) occurred regardless of histology, tumor size and location within the lobe.

We conclude that segmental pulmonary resections may provide long term survival similar to lobectomies in Stage I carcinoma, but with a higher incidence of ipsilateral intrathoracic recurrence. Vigilant follow-up is therefore especially important for patients undergoing segmental pulmonary resections.

11. Temporary and Permanent Restoration of Airway Patency With the Tracheal T-Tube

*HERMES C. GRILLO, M.D., HENNING A. GAISSERT, M.D. *,*

DOUGLAS J. MATHISEN, M.D. and

*JOHN C. WAIN, M.D.**

Boston, Massachusetts

The advantages of the tracheal T-tube compared to regular tracheostomy tubes are a physiologic direction of airflow, preservation of laryngeal phonation, and superior patient acceptance. Between 1968 and 1991, 69 males and 69 females (age 7 months to 95 years, mean 43.8 years) underwent placement of T-, TY- (6 patients), or a modified extended T-tube (4 patients). On admission, 84 patients had tracheostomy tubes, 4 had T-tubes, and 2 patients arrived after emergent translaryngeal intubation. Of 34 patients without airway support, 28 (82.3%) had dyspnea, 24 (70.6%) had stridor, and 6 (17.8%) had persistent cough. In 13 patients tube insertion was performed in the immediate postoperative period.

Primary diagnosis was postintubation stenosis in 85 patients, burn injury in 13 patients, malignant airway tumors in 12 patients, and various disorders in 27 patients. Uses were as follows: (1) silastic tube stenting was temporary in 29 patients, of whom 14 underwent later operative reconstruction; (2) definitive permanent insertion was performed in 48 patients. A modified silastic tube was used in 4 patients with left main bronchial stenosis after right pneumonectomy and provided effective long-term palliation in 3. (3) Postoperative airway obstruction after reconstruction prompted placement in 32 patients. The T-tube was not tolerated in 28 patients (20.4%) due to obstruction of the upper limb (laryngeal edema, unreconstructible subglottic stenosis) and aspiration. Positioning of the T-tube above the vocal cords in 12 patients for subglottic stenosis was effective in 10. Five of 10 children under age 10 developed airway obstruction necessitating tube removal.

Successful long-term intubation in 109 patients exceeded 1 year in 48 patients and 5 years in 12 patients. Only 5 patients required tube removal for obstructive problems more than 2 months after placement. The tracheal T-tube restores airway patency reliably with excellent long-term results and represents the preferred management of airway obstruction not amenable to surgical reconstruction.

*By Invitation

TUESDAY MORNING, APRIL 27, 1993

7:30 a.m. FORUM SESSION I - Grand Ballroom

CARDIAC SURGERY

Moderators: Aldo R. Castaneda, M.D.

Bruce A. Reitz, M.D.

F1. Six-Year Experience With e-PTFE Chordal Replacement in Floppy Mitral Valve

*CLAUDIO ZUSSA, M.D. *, ELVIO POLESEL, M.D. *,*

UBERTO DA COL, M.D. and CARLO VALFRE, M.D.**

Treviso, Italy

Sponsored by: Robert W.M. Prater, M.D., Bronx, New York

Conventional reparative techniques yield better functional results when compared with valve replacement in mitral position, but they are not always completely satisfactory or not adequate at all in cases of diffuse chordal degeneration or rupture. After two years of animal experiments, in 1986 we started the clinical use of 5-0 expanded polytetrafluoroethylene (e-PTFE) sutures (W.L. Gore & Assoc., Flagstaff, AZ) for chordal replacement in selected cases of mitral repair (86 patients so far). A floppy mitral valve with severe insufficiency was present in 61 cases (39 males, 22 females) with a mean age of 52.6 years (range 15-70). The insertion of 2 to 14 e-PTFE chordae was associated with other procedures which alone were not adequate to achieve a satisfactory result: suture annuloplasty reinforced with autologous pericardium in 54 cases, Carpentier ring in 3, posterior leaflet quadrangular resection in 46, chordal shortening in one. Intraoperative transesophageal echocardiography (TEE) was utilized before cardiopulmonary bypass to evaluate the functional valve anatomy, and after the procedure to assess the result (two valves were replaced during the same operation). All patients were discharged from the hospital (in 56 cases with short term -3 months- oral anti-coagulation) and followed every six months with TEE. At the last follow-up (2-72 months, mean 19.3) the mitral gradient ranged from 2 to 4 mmHg, the valve area from 2.3 to 3.5 sq. cm, mitral insufficiency was absent in 46 cases, trivial in 10, mild in 2, and severe in the last (at the 18-month follow-up). At redo-operation this patient showed rupture of first order chordae shortened at operation, while artificial chordae were still in place, partially covered by host tissue. No deaths or other valve-related events have been reported so far. This innovative procedure has allowed us to expand the indications for mitral valve repair to most of the cases in which, because of diffuse degenerative pathology, the usual techniques are not adequate.

*By Invitation

F2. Prevention of Experimental Postoperative Pericardial Adhesions Using a Hyaluronic Acid Coating Solution

*JOHN D. MITCHELL, M.D. *, RAYMOND LEE, M.D. *,*

*KAZUO NEYA, M.D. *, GEORGE T. HODAKOWSKI, M.D. *,*

WOLFGANG HARRINGER, M.D. AND*

GUS J. VLAHAKES, M.D.

Boston, Massachusetts

Postoperative pericardial adhesions complicate reoperative cardiac procedures. Prior work on their prevention has focused primarily on pericardial patch substitutes, but difficulties with patch migration and infection, epicardial scarring, and late calcification have limited their general use. Recently, topical application of solutions containing hyaluronic acid (HA) have been shown to reduce adhesions following abdominal, pelvic and tendon surgery. The mechanism by which HA solutions prevent adhesion formation is unknown, but is thought to be due to a cytoprotective effect of mesothelial surfaces, thus limiting intraoperative injury. We hypothesized that the intra-pericardial use of a HA solution might reduce the severity of pericardial adhesion formation and epicardial reaction frequently seen at cardiac reoperations.

To test this hypothesis, eighteen mongrel dogs underwent median sternotomy and pericardiotomy followed by a standardized two hour protocol of forced warm air dessication and abrasion of the pericardial and epicardial surfaces with a dry gauze sponge. Group 1 (n = 6) served as untreated controls with subsequent pericardial and sternotomy closure. Group 2 (n = 6) received topical administration of 0.4% HA in phosphate-buffered saline at the time of pericardiotomy, at twenty minute intervals during the dessication/abrasion protocol, and at pericardial closure. Group 3 (n = 6) served as a vehicle control, receiving phosphate-buffered

saline as a topical agent in a fashion identical to Group 2. All animals underwent re sternotomy eight weeks after the initial surgery; the intrapericardial adhesions were graded on a 0 to 4 severity scale at seven different areas covering the ventricular, atrial, and great vessel surfaces, producing a mean adhesion score for each animal and for each experimental group. In both the untreated control (Group 1, mean score 3.2 ± 0.4) and vehicle control (Group 3, mean score 3.3 ± 0.2) animals, dense adhesions were encountered with frequent obliteration of anatomical dissection planes. In contrast, animals treated with the HA solution (Group 2, mean score 0.8 ± 0.3) characteristically had no adhesions or filmy, transparent adhesions graded significantly less severe than either the untreated control (Group 2 vs. Group 1, $p < 0.001$, t-test) or vehicle control (Group 2 vs. Group 3, $p < 0.001$, t-test) animals.

HA solutions are efficacious in the prevention of postoperative pericardial adhesions in this model. Further studies investigating the mechanism by which these solutions prevent adhesions, their optimal dose and method of application, and documentation of their safe use in the cardiopulmonary bypass environment are warranted.

*By invitation

F3. Oxygen Radical Mediated Vascular Injury Selectively Inhibits Receptor-Dependent Release of Nitric Oxide from the Canine Coronary Artery Endothelium

*JOHN F. SECCOMBE, M.D. *, PAUL J. PEARSON, M.D., Ph.D. **

and HARTZELL V. SCHAFF, M.D.

Rochester, Minnesota and Seattle, Washington

Reperfusion following global cardiac ischemia may injure coronary artery endothelium and lead to vasospasm and thrombosis. Superoxide anion (O_2^*) has been implicated as a mediator of this process, but the precise mechanism of injury is unknown. We hypothesized that (O_2^*) impairs coronary endothelial production of nitric oxide, a potent endogenous vasodilator and inhibitor of platelet adhesion. To test this theory, we developed an *in vitro* model of reperfusion injury in which segments of epicardial canine coronary artery were suspended in organ chambers (physiologic salt solution, 37 °C, 95% O_2 , 5% CO_2) and exposed to (O_2^*) (generated by adding xanthine [$10^{-4}M$] and xanthine oxidase [$100/\mu U/ml$] to the bathing solution for 70 min). Following (O_2^*) exposure, epicardial coronary artery smooth muscle exhibited normal contraction to potassium ions (20mM) and prostaglandin F_{2a} (4×10^{-6}); also, the rings relaxed normally upon exposure to isoproterenol and sodium nitroprusside (10^{-5} $10^{-4}M$) ($n = 6$). In contrast, endothelium-dependent vasodilation to *receptor-dependent* agonists acetylcholine and adenosine diphosphate (10^{-9} $10^{-4}M$) was impaired as compared to control vessels not exposed to (O_2^*) ($n = 9$, $P < 0.05$). Importantly, *receptor-independent*, endothelium-dependent relaxation to the calcium ionophore A23187 was normal ($n = 6$). Further, endothelium-dependent vasodilation to *receptor-dependent* agonist bradykinin (non-nitric oxide pathway), was normal following (O_2^*) exposure. This is the first study to demonstrate that (O_2^*) selectively impairs *receptor-dependent* nitric oxide production by the coronary endothelium. Diminished nitric oxide production is a likely mechanism of vasospasm and thrombosis following reperfusion of the ischemic heart.

*By Invitation

F4. Inhomogeneous and Complimentary Delivery of Antegrade and Retrograde Cardioplegia in the Absence of Coronary Artery Obstruction

GABRIEL S. ALDEA, M.D. *, DIHOIJ, M.D. *,

JAMES D. FONGER, M.D. * and

RICHARD J. SHEMIN, M.D.

Boston, Massachusetts

Optimal myocardial protection relies on adequate delivery of cardioplegia to all areas of the heart. Cardioplegia delivery may have an inhomogeneous distribution throughout the myocardium, placing some areas at risk for ischemic injury, even in the absence of coronary artery obstruction. This study compares flow and distribution of antegrade (AC) and retrograde (RC) cardioplegia delivery in hearts with unobstructed coronary vessels. Cardioplegia delivery (flow) to individual myocardial regions weighing on average 1 gram was accurately measured with radioactive microspheres. In-homogeneity of distribution to these areas was expressed as the coefficient of variation (CV = SD/meanx100).

Six pigs were placed on cardiopulmonary bypass and underwent warm blood cardioplegic arrest. AC and RC were delivered at 150 ml/min and flow to 1152 individual myocardial regions was determined twice for each route of delivery using four different radiolabeled microspheres. Patterns of flow to individual myocardial regions were matched and analyzed by linear regression. Using each region as its own control an R² value was determined to assess reproducibility of flow patterns. Reproducibility and comparison of AC an RC flows in a typical animal are represented in the figures below:

Data is reported as mean ± SD. Statistical comparisons were made by paired t-test analyses.

	AC	RC	
Flow (ml/gm/min)	1.37 ± 0.31	0.39 ± 0.09*	
CV	48% ± 17%	106% ± 16%*	
*p<0.001 vs. AC-rc			
	AC vs. AC	RC vs. RC	AC vs. RC
R ²	0.88 ± 0.12**	0.84 ± 0.10**	0.03 ± 0.04

**p<0.001 vs. AC-RC

In unobstructed coronary vessels AC delivered greater flow to each gram of myocardium than RC. Flow delivery to individual myocardial regions was significantly inhomogeneous for both AC and RC, but much more so for RC. Patterns of flow with AC and RC were different and therefore complementary. These findings support the routine combined use of AC and RC to enhance cardioplegia delivery to all regions of the heart and minimize the potential risk of post ischemic myocardial dysfunction.

*By Invitation

F5. Controlled Reoxygenation in Hypoxemic Immature Hearts: A Cardioprotective Strategy that Avoids Reoxygenation Injury and Maintains the Benefits of Cardioplegia

KIYOZO MORITA, M.D. *, KAIA. IHNKEN, M.D. *,
GERALD D. BUCKBERG, M.D., HELEN H. YOUNG, Ph.D. *

and MICHAEL P. SHERMAN, M.D. *

Los Angeles, California

We test the hypothesis that abrupt *uncontrolled reoxygenation* of cyanotic immature hearts when starting cardiopulmonary bypass (CPB) produces a reoxygenation injury that; a) nullifies the Cardioprotective effects of blood cardioplegia, and b) is avoidable by controlling pO₂ during induction of CPB and blood cardioplegia; *controlled reoxygenation* (reO₂).

METHODS: Eighteen immature piglets (< 3 weeks old) underwent 30 minutes of aortic clamping with hypocalcemic, blood cardioplegia. Six piglets remained normoxemic (control). Twelve piglets were made hypoxic (pO₂ 20-30mmHg) for up to 2 hours by decreasing ventilator FIO₂ to 6-7% before CPB. Six of these piglets underwent 5 minutes of abrupt *uncontrolled reoxygenation* by instituting CPB at pO₂ 400 mmHg, before receiving pO₂ 400 mmHg blood cardioplegia. Six others underwent *controlled reoxygenation* by starting CPB at ambient pO₂ (20-30mmHg), followed 5 minutes later by blood cardioplegia at pO₂ 150 mmHg. Post CPB myocardial function was evaluated from endsystolic elastance (Ees, conductance catheter), oxidant damage was assessed by measuring myocardial conjugated diene production (lipid peroxidation) and antioxidant reserve capacity determined by measuring malondialdehyde (MDA) produced from myocardium incubated in the oxidant, t-butyl hydroperoxide (t-BHP).

RESULTS: Blood cardioplegia preserved myocardial function, and produced no oxidant damage in normoxemic piglets. Uncontrolled reoxygenation offset these Cardioprotective effects and severe functional depression, lipid peroxidation, and reduced antioxidant reserve capacity occurred despite blood cardioplegia. Conversely, controlled reoxygenation allowed near complete recovery of function and avoided oxidant damage with the same cardioplegia protocol.

Group	% Ees (recovery)	Conjugated Dienes (A ₂₃₃ /min/100g)	Antioxidant Reserve (MDA @ 4.0mM t-BHP)
No Hypoxia	102 ± 7	3 ± 3	943 ± 88
Uncontrolled ReO ₂	21 ± 2*	42 ± 9*	1321 ± 79*
Controlled ReO ₂	83 ± 8**	4 ± 3**	982 ± 88**

*:p<0.05 vs No Hypoxia, **:p<0.05 vs Uncontrolled ReO₂ (ANOVA)

CONCLUSION: Abrupt *uncontrolled reoxygenation* causes cardiac damage and nullifies the cardioprotective effects of blood cardioplegia. *Controlled reoxygenation* avoids these detrimental effects. These findings imply that controlling pO₂ during induction of cardiopulmonary bypass and blood cardioplegia can be used to surgical advantage in cyanotic patients.

*By Invitation

F6. Prevention of Complement Induced Pulmonary Hypertension and Improvement of Right Ventricular Function by Selective Thromboxane Antagonism

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ROBERT F. APPELYARD, Ph.D. *,

ROBERT J. RIZZO, M.D. *, LISHAN AKLOG, M.D. *

and LAWRENCE H. COHN, M.D.

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Despite advances in myocardial preservation and post operative management, acute reactive pulmonary hypertension can occur following open heart operations and heart transplantation. Complement activation following car-diopulmonary bypass may lead to pulmonary hypertension that causes right ventricular dysfunction in hearts with poor contractile reserve. We hypothesize that reactive pulmonary vasoconstriction is caused by complement induced thromboxane (TXA₂) production.

Sheep (n = 12) were anesthetized, instrumented for baseline measurements and randomized to receive either placebo or SQ30741 (10 mg/kg/hr), a thromboxane receptor antagonist. All sheep then received 30 units/kg of Cobra Venom Factor -Naje Haje (CVF), a known activator of complement, to produce a model of acute pulmonary hypertension. Simultaneous pulmonary artery (PA) and left atrial (LA) thromboxane levels were measured by radioimmunoassay to determine pulmonary production.

	Baseline	15 min.	30 min.	1 hr.	2 hr.
Control PAP	8.9 ± 1.7	19.4 ± 1.6	17.8 ± 1.6	9.8 ± 1.6	9.4 ± 1.6
SQ PAP	10.1 ± 1.6	8.6 ± 1.6*	8.0 ± 1.6*	7.5 ± 1.6	9.2 ± 1.6
(mm Hg)					
Control PVR	427 ± 109	1030 ± 101	912 ± 101	593 ± 101	442 ± 101
SQ PVR	352 ± 101	299 ± 101*	268 ± 101*	290 ± 101	395 ± 101
(dyne sec/cm ⁵)					
Control RVSW	2.66 ± .75	4.67 ± .70#	4.17 ± .71#	2.31 ± .71	2.57 ± .71
SQ RVSW	3.48 ± .71	3.28 ± .71	3.02 ± .71	2.85 ± .71	3.02 ± .71
(ergs x 10 ⁵)					

values: Mean±SEM; PAP = pulmonary artery pressure; PVR = pulmonary vascular resistance; RVSW = right ventricular stroke work; *p<0.05 vs Control; #p<0.05 vs Baseline (ANOVA)

In all animals there was a marked increase in TXA₂ production in the pulmonary bed during the first 30 minutes of complement activation. In controls this was accompanied by significant increases in pulmonary pressure and resistance causing increased right ventricular stroke work. These changes in PAP, PVR and RVSW were completely prevented by thromboxane receptor blockade with SQ30741, suggesting that complement activation produces an increase in pulmonary vascular resistance that is specifically mediated by thromboxane production in the pulmonary bed. Systemic vascular resistance was not affected by complement activation nor thromboxane receptor blockade.

Select thromboxane receptor antagonism may effectively prevent postoperative complement mediated pulmonary vasospasm and thereby provide protection against RV dysfunction in patients with limited RV contractile reserve.

*By invitation

F7. Cyclosporine and Splenectomy Prolong Cardiac Xenograft Survival by Suppressing IL-6 Production and MHC Class II Expression

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Cyclosporine (CsA) and splenectomy (Spx) synergistically prolong xenograft survival in the hamster to rat model. Our objective was to identify actions of this therapy on the cellular and humoral response compared to untreated controls. Inbred male Lewis rats received 15 mg/kg/day CsA and were splenectomized 2 days after heterotopic heart transplantation from Golden Syrian hamsters (group 1). Control groups included isografts (group 2) and untreated xenografts (group 3). Plasma IL-6 activity was determined using proliferation of IL-6 dependent 7TD1 hybridoma cells. Deposition of IgM, IgG, and Complement factor 3 (C3) were demonstrated using FITC-conjugated antibodies. Graft infiltrating cells (GIC) expressing MHC Class II antigen were identified with immunoperoxidase staining and OX6 (antibody for Ia antigen). In a random fashion, OX6+ cells were counted in a minimum of 10hpf per specimen. Results of IL-6 plasma activity in U/ml (mean \pm sem):

	Day 1	Day 3	Day 10
Group 1		391 \pm 98	35 \pm 8
Group 2	113 \pm 52	279 \pm 88	152 \pm 43
Group 3	1470 \pm 223	1251 \pm 294	

Group 1 had a significant decrease in IL-6 activity at day 3 compared to untreated xenografts ($p < 0.01$). At day 3, light microscopy showed severe rejection in group 3. Group 1 hearts were beating strongly and only a mild cellular infiltrate was seen at day 10, similar to group 2 (isografts). Group 3 (control xenografts) displayed dense accumulation of IgM and C3 along the endothelium and interstitial capillaries, whereas groups 1 and 2 had minimal deposits. OX6 + GIC were markedly reduced in group 1 (2.8 ± 0.4) compared to group 3 (9.5 ± 0.6), $p < 0.0005$. The data indicate a direct correlation between IL-6 and xenograft rejection. The induction of B-cell maturation and

stimulation of Ig production is a property of IL-6, and may play a pivotal role in the initiation of the humoral response in concordant rejection. The significant decreased deposition of humoral components (IgM and C3), decreased IL-6 plasma levels, and decreased QIC's expressing MHC Class II antigen within the nonrejecting grafts suggests that the synergy of CsA and Spx depends on the attenuation of cellular and humoral mechanisms involved in xenograft rejection.

*By Invitation

F8. Complete Prevention of Post-Ischemic Spinal Cord Injury Using Regional Perfusion With Hypothermic Saline and Adenosine

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*LEE BUTTERFIELD, M.D. *, LORNE BLACKBOURNE, M.D. *,*

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Spinal cord injury following operations on the descending thoracic and thoracoabdominal aorta remains a persistent clinical problem. Previous attempts to decrease the risk of this devastating complication by lowering the rate of metabolism of the spinal cord have met with only varying success. We hypothesized that the tolerance of the spinal cord to an ischemic insult could be improved by using adenosine. Twenty New Zealand white rabbits underwent 40 minutes of isolated infrarenal aortic occlusion with heparin anti-coagulation. Clamps were placed both below the left renal vein and above the aortic bifurcation. In ten rabbits (Group A), a bolus of adenosine (100 mg) was infused into the isolated aortic segment immediately after cross-clamping and this bolus was followed by a flush of hypothermic saline (8 °C, 30 ml/kg) over the first 10 minutes of ischemia time. In one control group of five animals (Group B), cross-clamping of the descending infrarenal aorta was performed without infusion of adenosine or saline. In another control group of five animals (Group C), the aortic segment was flushed with normothermic saline (37 °C) in a fashion similar to the study group. The aortic clamps were removed after 40 minutes, the abdomen closed, and the animals allowed to recover from anesthesia. Spinal cord function was assessed 12, 24, 48, 72, and 96 hours after operation by the Tarlov scale (0-no movement, 1-slight movement, 2-sits with assistance, 3-sits alone, 4-weak hop, 5-normal hop). All animals were sacrificed at 96 hours after operation and spinal cords harvested for histologic analysis. The spinal cord function of all Group A animals was fully intact with Tarlov scores of 5, while Group B and Group C animals were all paraplegic with Tarlov scores of 0 ($p < 0.001$, ANOVA). Histologic examination of spinal cords from Group A revealed no evidence of cord injury, while Group B and Group C spinal cords had evidence of cord injury with central grey necrosis, axonal swelling, dissolution of Nissl substance, and astrocyte and macrophage infiltration. Regional infusion of the cross-clamped infrarenal rabbit aorta segment with hypothermic saline and adenosine *completely* prevented paraplegia in our model despite a 40 minute ischemic insult.

*By Invitation

F9. Effects of Pharmacologic Modification of Cerebroplegia Solution on Acute Recovery of Cerebral Blood Flow and Metabolism After Hypothermic Circulatory Arrest

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PA UL R. MICKEY, M.D. *, DA VID HOLTZMAN, M.D. *,
and RICHARD A. JONAS, M.D.*

Boston and Cambridge, Massachusetts

Previous studies have suggested that a simple crystalloid "cerebroplegic" solution may prolong the safe duration of hypothermic circulatory arrest (CA). We explored pharmacologic manipulation of cerebroplegia solutions including an organ preservation solution containing adenosine and glutathione (University of Wisconsin U.W.) and the excitatory neurotransmitter antagonist MK801 which has demonstrated efficacy in decreasing cerebral ischemic injury.

Forty-six 4-week old minipigs underwent core cooling to 15°C naso-pharyngeal temperature and 2 hours of CA, 45 minutes of rewarming and 3 hours of normothermic reperfusion. Group CPS (n = 12) had 50 ml/kg of saline infused into the carotid artery system at the onset of CA and repeat doses 10 ml/kg every 30 minutes. Group CPU (n = 11) received the same dose of UW. Group CPM (n = 10) received UW with 7.5 mg/l of MK-801. Group CNT (n = 13) served as control (2 hours CA at 15°C). All solutions were delivered at 4°C. Recovery of cerebral ATP and intracellular pH (pHi) was assessed by magnetic resonance spectroscopy in half the animals in each group, and cerebral blood flow (CBF) by microspheres, cerebral metabolic rate of oxygen (CMRO2), vascular resistance reactivity to acetylcholine (ACh) and nitroglycerin (TNG) in the carotid circulation, and brain temperature were measured in the rest. Brain water content was assessed at the end of the experiment in all animals.

Results are given as men±SEM. *: p<0.05 vs. group CNT by Student-Newman-Keuls test.

		ANOVA				
	time alter reperfusion	CNT	CPS	CPU	CPM	p values
ATP	45 min	34.1 ± 6.9	38.4 ± 4.0	62.7 ± 5.3*	63.2 ± 7.0*	0.004
(% recovery)	225 min	28.5 ± 9.3	57.3 ± 2.6*	72.2 ± 3.0*	72.9 ± 4.6*	0.000
pHi (Unit)	45 min	6.53 ± 0.06	6.43 ± 0.10	6.54 ± 0.08	6.71 ± 0.08	0.108
(baseline = 7.12 ± 0.001)	225 min	6.57 ± 0.11	6.90 ± 0.14	7.14 ± 0.02*	7.15 ± 0.01*	0.000
CBF	45 min	33.0 ± 6.1	52.1 ± 4.5'	75.5 ± 4.7*	42.5 ± 4.3	0.000
(% recovery)	225 min	78.3 ± 6.8	96.4 ± 5.6	128.3 ± 7.6*	93.5 ± 6.8	0.000
CMRO2	45 min	16.3 ± 9.1	27.6 ± 8.6	52.2 ± 4.7*	26.4 ± 4.9	0.000
(% recovery)	225 min	43.6 ± 5.0	59.2 ± 4.4	87.3 ± 13.1	0.002	
Brain water content (%)		82.10 ± 0.22	80.74 ± 0.25*	80.53 ± 0.43*	80.56 ± 0.31*	0.000

Brain temperature at the onset on CA was 15.0±0.1°C and dropped to 13.0±0.3°C after cerebroplegia infusion and stayed lower than group CNT throughout the CA. The groups CPS and CPU showed better vascular resistance response (vasodilation) to ACh (p = 0.008) and TNG (p = 0.048) at 60 minutes of reperfusion relative to groups CNT and CPM.

Cerebroplegia improves acute recovery after 2 hours circulatory arrest. Pharmacological modification with UW further improves the recovery of cerebral blood flow and metabolism. MK-

801 does not augment the protective effects of UW and reduces the recovery of cerebral blood flow, presumably by a direct vascular action. Modified cerebroplegia may provide a novel approach to improved cerebral protection when circulatory arrest is necessary.

*By invitation

TUESDAY MORNING, April 27, 1993

9:00 a.m. SCIENTIFIC SESSION - Grand Ballroom

Moderators: James L. Cox, M.D.

Martin F. McKneally, M.D.

12. Effects of Diltiazem on Perioperative Ischemia, Arrhythmias and Myocardial Function in Patients Undergoing Elective Coronary Bypass Grafting

*RAINALD SEITELBERGER, M.D. **

*WALTRAUD HANNES, M.D. *, MARK GLEICHAUF, M.D. *,*

*HORST ZAJONC, M.D., VOLKER SCHLOSSER, M.D. **

*and ROLAND FASOL, M.D. **

Freiburg, Germany

Sponsored by: Prof. Dr. Ernst Wolner, Vienna, Austria

Apart from the technical quality of the surgical procedure itself, the outcome of patients undergoing coronary bypass surgery highly depends on the quality of perioperative myocardial protection. Recent studies have shown that the continuous, perioperative infusion of calcium antagonists effectively reduces incidence and extent of myocardial ischemia. However, little is known about the influence of calcium antagonists in the prevention of perioperative arrhythmias and the preservation of myocardial function.

Consequently, a prospective, randomized study was performed on 120 patients undergoing elective coronary bypass grafting. The patients received a continuous, perioperative infusion of either the calcium antagonist diltiazem (0.1mg/kg/h, n = 60) or nitroglycerin (control group, 1µg/kg/min, n = 60) over a period of 24 hours. Perioperative monitoring included hemodynamic measurements and 3-channel Holter monitoring up to 24 hours post-operatively, repeated assessment of 12-lead ECG and analysis of ischemia-specific laboratory parameters (CK, CK-MB, CK-MB-mass, troponin-T and myoglobin) until the 6th postoperative day. In addition, regional and global myocardial function was assessed preoperatively and 1 and 4 hours after car-diopulmonary bypass by means of transesophageal echocardiography (monoplane 5MHz faced array transducer). In order to assess the diastolic compliance of the left ventricle, transmitral flow-velocity profiles were performed in the transesophageal long-axis view.

The two groups did not differ with respect to preoperative and operative (number of distal anastomoses, aortic cross clamp and extracorporeal circulation time, etc.) data. Except for a significant reduction in perioperative heart rate by an average of 9 beats/min, diltiazem had no influence on hemodynamic parameters. The antiischemic efficacy of diltiazem lead to a reduction of the number (17 ± 9 vs. 25 ± 5 , $p < 0.05$) and duration (69 ± 47 vs. 104 ± 87 min, $p < 0.05$) of transient ischemic events and a lower incidence of perioperative myocardial infarction (3.3 vs. 6.7%) as compared to the nitroglycerin group. In addition, peak values of all assessed laboratory parameters were significantly lower in the diltiazem group. With regard

to perioperative arrhythmias, patients treated with diltiazem had a lower incidence of perioperative atrial fibrillation (5 vs. 18%, $p<0.05$) and lower numbers of ventricular premature beats/hour (10 ± 8 vs. 19 ± 22 , $p<0.05$). Perioperative regional and global systolic myocardial function did not significantly differ between both groups. However, the incidence of diastolic compliance disturbances at 4 hours after cardiopulmonary bypass was 53% in the nitroglycerin-, but only 26% in the diltiazem group ($p<0.05$).

It is concluded that perioperative infusion of the calcium antagonist diltiazem does not adversely affect perioperative hemodynamics and systolic myocardial function and provides potent antiischemic and antiarrhythmic protection in patients undergoing aortocoronary bypass grafting. In addition, diltiazem markedly improves the postoperative diastolic compliance of the left ventricle.

*By Invitation

13. Chordal Preserving Mitral Valve Replacement: The Hemodynamic Study in the Early and Mid-Term Period

*YUTAKA OKITA, M.D. *, SHIGEHIITO MIKI, M.D. *,*

*YUICHI UEDA, M.D. *, TAKAFUMI TAHATA, M.D. *,*

*TETSURO SAKAI, M.D. * and*

*KATSUHIKO MATSUYAMA, M.D. **

Nara, Japan

Sponsored by: Tadaomi Miyamoto, M.D., Kitakyushu, Japan

The clinical significance of the chordae tendinae in regards to postoperative left ventricular (LV) performance was evaluated in patients with mitral regurgitation (MR) or mitral stenosis (MS) who underwent either mitral valve replacement (MVR) using St. Jude Medical valve with complete chordae preservation (P) or with conventional MVR (C), or valve repair (R). **[Patients and methods]** (*P group*): MVR preserving autologous chordae tendinae ($n = 37$) or replacing chordae with Gore-Tex sutures ($n = 13$) was performed in 50 patients, 30 with MR and 20 with MS. The valve pathology was rheumatic in 17 patients, degenerative in 10, and infective endocarditis in 3. (*C group*): conventional MVR involving 25 with MR and 28 with MS. (*R group*): valve repair in 24 with MR or commissurotomy in 27 patients with MS. The LV performance was analyzed by cineangiography (CAG) in the early (mean 1.2 months), and multiple gated blood scintigraphy (MUGA), or echocardiography (UCG) in the late postoperative periods (mean 5.4 years) in 3 groups of patients. Statistical analysis was performed using Student T-test. **[Results: Table]** C patients with MVR for MR had a significantly greater end-systolic volume index (ESVI), the LV ejection fraction (EF) was unchanged in the P group, but was decreased in C and R group. The LV contractility index [end-systolic circumferential left ventricular wall stress (ESS)-ESVI⁻¹] was better in P than C group. Both the LVEF by MUGA, and LV fractional shortening (FS) by UCG were significantly higher in P and R than in C group patients. P group exhibited superior LV performance than C group especially in those with MR and depressed preoperative left ventricular function ($EF<0.50$). There were no significant difference, except for better postoperative EF (CAG) in P group, between the 3 groups in patients with MS. **[Conclusion]** The results support the concept that maintenance of continuity between the mitral annulus and the papillary muscle has a beneficial effect on postoperative left ventricular performance, especially in patients with MR and depressed preoperative left ventricular function, but has no major effect in patients with MS.

MR				
Group		P(n = 30)	C(n = 25)	R(n = 24)
EDVI	<i>pre</i>	118 ± 46	126 ± 37	140 ± 42
(ml-m ⁻²)	<i>post</i>	96 ± 16*	94 ± 20*	113 ± 20
ESVI	<i>pre</i>	53 ± 20	58 ± 18	58 ± 25
(ml-m ⁻²)	<i>post</i>	42 ± 13	50 ± 15	52 ± 14
EF (CAG)	<i>pre</i>	0.53 ± 0.11	0.53 ± 0.08	0.59 ± 0.12
	<i>post</i>	0.56 ± 0.21*	0.49 ± 0.10	0.54 ± 0.09*
ESS/ESVI	<i>pre</i>	4375 ± 1572*	3622 ± 579	3809 ± 138
($\text{\$}$)	<i>post</i>	4063 ± 1027*	3224 ± 914	3837 ± 667*
EF (MUGA)	<i>pre</i>	n.a.	n.a.	n.a.
	<i>post</i>	0.57 ± 0.08*	0.51 ± 0.09	0.61 ± 0.07*
FS	<i>pre</i>	0.38 ± 0.08	0.34 ± 0.08	0.36 ± 0.06
	<i>post</i>	0.36 ± 0.07*	0.29 ± 0.08	0.33 ± 0.08*
MS				
Group		P (n = 20)	C(n = 28)	R(n = 27)
EDVI	<i>pre</i>	75 ± 22	85 ± 28	78 ± 18
(ml-m ⁻²)	<i>post</i>	91 ± 23	81 ± 22	72 ± 16
ESVI	<i>pre</i>	41 ± 15	42 ± 16	39 ± 13
(ml-m ⁻²)	<i>post</i>	40 ± 15	39 ± 14	33 ± 8
EF (CAG)	<i>pre</i>	0.47 ± 0.08	0.52 ± 0.08	0.50 ± 0.11
	<i>post</i>	0.56 ± 0.10*	0.48 ± 0.09	0.54 ± 0.08
ESS/ESVI	<i>pre</i>	4685 ± 1102	4412 ± 1369	4799 ± 1564
($\text{\$}$)	<i>post</i>	4787 ± 827	4526 ± 1587	4880 ± 528
EF (MUGA)	<i>pre</i>	n.a.	n.a.	n.a.
	<i>post</i>	0.57 ± 0.08	0.55 ± 0.11	0.56 ± 0.09
FS	<i>pre</i>	0.31 ± 0.09	0.30 ± 0.07	0.32 ± 0.08
	<i>post</i>	0.31 ± 0.08	0.34 ± 0.07	0.34 ± 0.09

Legends; All numerical shows Mean ± SD. n.a.: not available. ($\text{\$}$): kdyn-cm⁻¹ml⁻¹-m² Asterisk (*) shows statistically significant differences (p<0.05) compared with other groups.

*By Invitation

14. Management Needs Revision Following Valve Replacement by St. Jude Medical Prosthesis: Unexpected Results after 10 Years of Prospective Follow-Up of 600 Consecutive Patients

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WOLFGANG BIRCKS, M.D. and B.E. STRAUER, M.D. *
Dusseldorf, Germany*

Between 1978 and 1982, SJM-prostheses were implanted in 600 consecutive patients (P) averaging 50.7 ± 9.6 (14-81) years of age in the aortic (n = 298), mitral (n = 215) position or as multiple valve replacement (n = 87). P had in-hospital examinations every 3 to 6 months. Follow-up was complete and averaged 122.2 ± 1.1 months for late survivors. By random, three different anticoagulation regiments had been used with a target INR 3.0-4.5, INR 2.5-3.2 and INR 1.8-2.7. For these 3 regiments the incidences (events/100 patient-years) for thromboembolic (TE) and bleeding complications (BL) were calculated:

	INR 3.0-4.5	INR 2.5-3.2	INR 1.8-2.7
Aortic	n = 125	n = 86	n = 87
Severe TE	n = 1 (0.08)	n = 2 (0.24)	n = 1 (0.24)
Other TE	n = 21 (1.76)	n = 5 (0.61)	n = 6 (0.73)
Severe BL	n = 9 (0.75)	n = 4 (0.49)	-
Other BL	n = 59 (4.94)	n = 31 (3.80)	n = 16 (1.95)
All	n = 90 (7.54)	n = 42 (5.14)	n = 24 (2.93)
Mitral	n = 94	n = 62	n = 59
Severe TE	n = 2 (0.24)	n = 3 (0.52)	n = 3 (0.53)
Other TE	n = 28 (3.30)	n = 28 (4.81)	n = 33 (5.83)
Severe BL	n = 9 (1.06)	n = 3 (0.52)	-
Other BL	n = 80 (9.43)	n = 35 (6.02)	n = 16 (2.83)
All	n = 119 (14.02)	n = 69 (11.86)	n = 52 (9.19)
Multiple	n = 35	n = 29	n = 23
Severe TE	n = 1 (0.31)	n = 1 (0.37)	n = 1 (0.46)
Other TE	n = 11 (3.39)	n = 13 (4.86)	n = 15 (6.97)
Severe BL	n = 4 (1.23)	n = 2 (0.75)	n = 1 (0.46)
Other BL	n = 29 (8.94)	n = 19 (7.11)	n = 7 (3.25)
All	n = 45 (13.87)	n = 35 (13.09)	n = 24 (11.16)

The optimal efficacy risk relation for P with SJM aortic prostheses were calculated as a target INR 2.4-2.6; for SJM mitral prostheses P with atrial contraction, left atrial diameter < 26 mm/m², an INR 2.4-2.6; for SJM mitral prosthesis P without atrial contraction, left atrial diameter > 30 mm/m², an INR 2.7-3.1; and for SJM multiple valve replacement INR 2.8-3.2. These findings give further evidence of the low thrombogenicity of the SJM prostheses and supports arguments for a much lower intensity of anticoagulation than generally recommended.

10:00 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

10:45 a.m. SCIENTIFIC SESSION - Grand Ballroom

Moderators: John L. Ochsner, M.D.

Martin F. McKneally, M.D.

15. Surgical Treatment of Barrett's Carcinoma. Correlation Between Morphologic Findings and Prognosis

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*DIRK VAN RAEMDONCK, M.D. *, PAUL DELEYN, M.D.,*

*JEAN MARC MARNETTE, M.D. * and*

*KAREL GEBOES, M.D. **

Leuven, Belgium

Sponsored by: Tom R. DeMeester, M.D., Los Angeles, California

Barrett carcinoma occurred in 66 of 331 patients with adenocarcinomas of the oesophagus or GE junction. Thirty-two (46%) of these patients had a positive history for gastroesophageal reflux. A history of alcohol and tobacco was absent in 50% and 47.5%, respectively. The mean length of Barrett metaplasia was 7.37 cm. Operability was 98.5%, and resectability 95.5%. There was no postoperative or hospital mortality. Pathological staging was: stage I: 38.3%, stage II: 20.6%, stage III: 22.2% and stage IV: 19%. Overall survival is 80.5% at 1 year, 62.7% at 2 and 58.2% at 5 years. Five-year survival for stage I is 100%, for stage II: 87.5%, for stage III: 22.2% and for stage IV: 0. Thirty-four (51.5%) patients were under surveillance for a related or unrelated condition before diagnosis of their carcinoma, only 9 (26.5%) had positive lymph nodes. Thirty-two had their diagnosis made at their first medical contact, 78% had positive lymph nodes. Five-year survival in patients without nodal metastasis was 85.3% and significantly better than those with metastasis, 38.3% ($p = 0.0033$).

Out of 66 patients, 19 (28.7%) had a biopsy proven history of Barrett metaplasia before malignancy occurred. Mean time interval between diagnosis of metaplasia and malignant degeneration was 3.8 years ($89.5\% > 1$ yr). Over the surveillance period, the length of metaplastic Barrett remained unchanged in all patients. Barrett ulceration was present from the beginning in 14 patients, 3 patients had never had an ulcer. Intestinal metaplasia was seen in 18 patients. Combining pre- and postoperative pathology revealed severe dysplasia in 16 patients. 13.7% of the 19 patients had stage I disease.

Conclusions: 1) Half of the patients with Barrett carcinoma have no history of reflux or tobacco and alcohol use. Early diagnosis was possible in patients under surveillance with 73.7% diagnosed with stage I disease and 100% five-year survival. Barrett ulcer, intestinal type of metaplasia, were common findings in patients under surveillance with a biopsy-proven history of Barrett metaplasia before carcinoma.

*By Invitation

16. Risk of Replacement of Descending Aorta With a Standardized Left Heart Bypass Technique

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*BEA TE BUHNER * and JOACHIM LAAS, M.D. **

Hannover, Germany

From 4/1986 to 11/1992, 130 patients (pts) aged 48 (15 to 73) years underwent replacement of the descending aorta (DA) using centrifugal pump (Bio-Medicus) left heart bypass for the following conditions: Aneurysm = 66 (50.8%), dissection = 63 (48.5%), tumor = one. There were 55 aortic redos. The bypass was routed from the left atrium to peripheral vessels in 87, to the downstream aorta in 43 pts. Replacement extended to the 6th intercostal space (ICR) in 59 (45.4%), to the 9th ICR in 53 (40.8%), and was total/subtotal in 21 (16.2%) pts. Aortic clamp time was 45 (16 to 98) minutes. Intercostal arteries down to and including the 6th ICR routinely were sacrificed. Staged aortic clamping with or without intercostal artery reattachment was employed in 39 patients.

Thirty-day mortality was 3%. There were no bypass-related complications. Six patients (4.6%) showed spinal cord sequelae: paraplegia was seen in 4 pts which disappeared in one and improved in 2. Fully reversible paraparesis was noted in 2 pts. The rate of permanent neurological deficit therefore was 2.3%. These complications occurred only in aortic replacement to or beyond the 9th ICR and could not be totally prevented by staged aortic clamping and intercostal artery reconnection despite a significant reduction of spinal cord ischemia time thus pointing to yet uncontrolled factors.

We conclude that full exhaustion of the methodology of left heart bypass is associated with a low risk of early death and spinal complications. It therefore is considered the method of choice in replacement of the DA.

11:25 p.m. ADDRESS BY HONORED SPEAKER

"The Structure and Function of Tissue Valves; Some Lessons Learned From the Fate of Implanted Heart Valves"

Mark F. O'Brien, FRCS, Brisbane, Australia

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

12:10 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

Columbus Hall

*By Invitation

TUESDAY AFTERNOON, APRIL 27, 1993

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

Grand Ballroom B

Moderators: D. Glenn Pennington, M.D.

Irving L. Kron, M.D.

17. Results of Non-Guided Subtotal Endocardectomy Associated With Left Ventricular Reconstruction in 106 Patients With Ischemic Ventricular Arrhythmias

VINCENT DOR, M.D., MICHEL SABATIER, M.D. *,
FRANCOISE MONTIGLIO, M.D. *, PHILIPPE ROSSI, M.D. *,
MARISA DI DONA TO, M.D. * and
ANNA TOSO, M.D. *

Monte-Carlo, Monaco and Firenze, Italy

From June 1987 to September 1992, 284 pts underwent left ventricular (LV) reconstruction with endoventricular circular patch plasty and septal exclusion for postinfarction LV aneurysm or severe LV wall motion abnormalities. 106 out of these pts, presenting spontaneous and/or inducible ventricular tachycardia (VT), represent the study group (mean age 58 ± 8 years). There were 97 anterior akinetic or dyskinetic and 9 posterior aneurysms. 69 pts were in NYHA class III/IV; indication for surgery was angina in 35% of pts, intractable VA in 11% and a combination of angina, congestive failure and VA in the remaining pts. 18 pts were operated in emergency. In these 106 pts before LV patch reconstruction, subtotal endocardectomy of fibrous in-traventricular scar was performed; cryotherapy at the border of the lesion was associated in 67 pts and coronary revascularization, including infarcted area, was performed in 93% of pts. All pts underwent complete hemodynamic study including programmed ventricular stimulation (PVS), when not contraindicated, before and early after surgery (10-15 days). Clinical follow up is available in all pts (min 2 max 62 months, mean follow-up 19,8 m). At present 34 pts have complete hemodynamic control, including PVS after 1 year.

Preoperative data: 49 pts had documented episodes of spontaneous VT; 57 without spontaneous VTs had inducible VT at PVS and 20 had spontaneous and inducible VTs. PVS was contraindicated in 23 pts. Mean EF was $33 \pm 11\%$; contractile EF was $41 \pm 10\%$; EDVI was 124 ± 63 ml/m²; CI was 2.8 ± 6.5 l/min/m²; mean pulmonary artery pressure (PAP) was 20 ± 8 mmHg. **Postoperative data:** perioperative mortality rate was 7.5% (8 pts). EF improved significantly ($46 \pm 10\%$ $p < .001$) as well as PAP decreased (17 ± 7 mmHg $p < .001$); CI did not change significantly (2.7 ± 5.5 l/min/m²). Spontaneous VTs was recorded in 2 pts who had spontaneous VTs pre-operatively; VT was inducible in 8 pts (Wilcoxon test $p < .0001$). Four of these were contraindicated and 4 were inducible before surgery. **At late hemodynamic control** EF was still significantly increased (32 ± 10 basal; 47 ± 11 early control and 46 ± 11 % after 1 year $p < .01$, $n = 34$). In this group there were 11 spontaneous VTs and 23 inducible VTs in basal conditions. After 1 year VT was induced in 4/34 pts and no spontaneous VTs occurred. Three of the 4 pts with inducible VT were under amiodarone and they were inducible also at early control. Follow-up: two sudden deaths occurred among 7 late deaths; no spontaneous VTs were clinically recorded in the surviving pts, all controlled.

In conclusion, non guided subtotal endocardectomy +/- cryotherapy can be safely performed during surgery for LV aneurysm in pts with severely depressed LV function and it drastically and significantly reduces the occurrence of spontaneous and inducible VT early after surgery. In pts controlled after 1 year the beneficial effect on ventricular arrhythmias is maintained as well as the improvement of LV geometry and function.

*By invitation

18. The Effects of Myocardial Revascularization on the Incidence of Implanted Defibrillator Discharge in Patients With Cardiac Dysfunction

HOOSHANG BOLOOKI, M.D.,
MICHAEL D. HOROWITZ, M.D. *,

*GEORGE M. PALATIANOS, M.D. *,*

*ALBERTO INTERIAN, JR., M.D. *, MICHAEL BARRON, M.D.**

*and RICHARD A. FERRYMAN, M.D.**

Miami, Florida

We studied 115 patients (pts) who had survived sudden death and had received an automatic implantable cardioverter defibrillator (AICD) either alone (Group A, n = 70) or after coronary bypass operation (CABG + AICD = Group B, n=45). All patients had inducible ventricular tachycardia/ fibrillation after previous myocardial infarction associated with coronary disease and left ventricular (LV) dysfunction. Pts with LV aneurysm and ablative procedures were not included. Mean age (63 vs 64 yrs), N.Y.H.A. Class IV (95% vs 93%), LV ejection fraction (27% vs 27%), LV diastolic pressure (21 vs 20mmHg), operative mortality 2.9% vs 2%, mean follow-up 30 vs 42 months (range 18-90 months), and sudden death rate 3.5% vs 2.4%/pt year were similar in both groups. However, the incidence of AICD discharge was significantly higher in Group A than Group B pts (75% vs 59%, P = 0.04). Furthermore, all Group A pts who had defibrillator discharge experienced the first shock in the first 24 months while Group B pts experienced their first shock within the first 12 months after implantation of the device. The overall survival rate at 6.5 years was 58% and 85% (P = n.s.) and the defibrillator shock-free survival rate was 24% and 47% (p = 0.04) for Groups A and B pts respectively. Patients with malignant ventricular arrhythmia and LV dysfunction who receive myocardial revascularization and an AICD implantation experience fewer defibrillator shocks than pts who receive AICD alone.

*By Invitation

19. Nonthoracotomy Lead System for Implantable Defibrillator

*BRADFORD P. BLAKEMAN, M.D. *,*

*HENRY J. SULLIVAN, M.D., ALVARO MONTOYA, M.D. *,*

*DAVID WILBER, M.D. *, BRIAN OLSHANSKY, M.D. *,*

JEFFREY BAERMAN, M.D, JOHN KALL, M.D. **

and ROQUE PIFARRE, M.D.

Maywood, Illinois

Automatic implantable defibrillators have become a standard therapy for ventricular arrhythmias. A new lead system consisting of one (CPI) or two (Medtronic) endocardial leads and a subcutaneous patch not requiring a thoracotomy are currently under investigation at our institution. Eighty-five insertions for the nonthoracotomy lead system (NTL) have been attempted and sixty-four were successful (75 percent). Sixty-five of the total patients were male and mean age was 57 years. Forty-nine patients (56 percent) had previous open heart surgery. Left ventricular ejection fraction for the entire group demonstrated a mean of 28.4 percent, a range of 12 to 74 percent. Operative data noted defibrillation thresholds (DFT) for the sixty-four successful patients to be a mean of 18.9 joules (range 3-25). The number of defibrillations necessary for successful NTL implants was a mean of 10.3. The reasons for unsuccessful implants were insufficient DFT's - 19 patients and inability to position endocardial lead - 2 patients. Conventional lead systems were implanted in NTL failure patients by the following: lateral thoracotomy 11, sternotomy 9 and subxyphoid 1. Ten of the twenty-one NTL failure patients required three or more conventional patches to attain adequate DFT's. Length of procedure for a successful NTL system was a mean of 123.7 minutes (range

30-270 minutes). Success of implant could not be linked to previous heart surgery, size of chest wall or ejection fraction. No inappropriate or unsuccessful defibrillations have occurred with implanted systems to date. Complications directly related to the device requiring further surgery included lead migration - 5 patients, hematoma - 3 patients and infection - 1 patient. The nonthoracotomy lead system demonstrates reasonable promise in this population.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION A CARDIAC SURGERY

Grand Ballroom B

20. Aprotinin for Coronary Bypass Surgery: Efficacy, Safety and Influence on Early Vein Graft Patency. Results of a Multi-center, Randomized, Double-Blind, Placebo-Controlled Study

*JOHN H. LEMMER, JR., M.D. *, WILLIAM STANFORD, M.D.,*

*SHARON L. BONNEY, M.D. *, JEROME F. BREEN, M.D. *,*

*EVA V. CHOMKA, M.D. *, W. JAY ELDREDGE, M.D. *,*

*WILLIAM W. HOLT, M.D. *, ROBERT B. KARP, M.D.,*

*GLENN W. LAUB, M.D. *, MARTIN J. LIPTON, M.D. *,*

HARTZELL V. SCHAFF, M.D., CONSTANTINE J. TATOOLE, M.D.

*and JOHN A. RUMBERGER, Ph.D., M.D. **

Iowa City, Iowa

Two hundred sixteen patients (pts) undergoing primary (151) and repeat (65) coronary bypass surgery (CABG) procedures at 5 hospitals were randomized to receive high-dose aprotinin or placebo during surgery. Saphenous vein graft (SVG) patency (330 total grafts) was evaluated by cine-computed tomography 7 to 45 days after operation in 164 (76%) pts. Assessment of patency was determined by group consensus without knowledge of the patients' randomized status. Patency was analyzed on both a per-patient and per-graft basis using Chi Square and Fishers Exact Test methods. Significance was at a $p \leq 0.05$ level.

Results:

	Aprotinin	Placebo	p-Value
Primary CABG pts requiring RBCs	28/74 (38%)	35/67 (52%)	0.052
Repeat CABG pts requiring RBCs	7/23 (30%)	23/32 (72%)	0.001
Primary pts-RBC vol transfused (n = 141)	362ml	606ml	0.023
Repeat pts-RBC vol transfused (n = 54)	164ml	931 ml	0.005

Pts with %1 closed SVG	13/83 (16%)	7/81 (9%)	0.170
Closed SVGs	14/176 (8%)	8/163 (5%)	0.248
Myocardial infarcts	10/108 (9%)	7/108 (6%)	0.448
Deaths	6/108 (6%)	4/108 (4%)	0.517

Conclusions: Prophylactic aprotinin decreases transfusion requirements in CABG pts, particularly in repeat procedures. In this study, there was a trend toward a higher rate of early SVG closure in patients who received aprotinin as compared to those who received placebo, although this result did not reach statistical significance. While this trend did not translate into a difference in perioperative myocardial infarctions or patient deaths, further investigations regarding the safety of routine aprotinin use appear indicated.

*By Invitation

21. Arterial Revascularization in 300 Patients With the Right Gastroepiploic Artery and Internal Mammary Arteries

*JAN G. GRANDJEAN, M.D. *, PIET W. BOONSTRA, M.D., Ph.D. *,*

*PETER DEN HEIJER, M.D. * and TJARK EBELS, M.D., Ph.D. **

Groningen, Ho/land

Sponsored by: John W. Kirklin, M.D., Birmingham, Alabama

From September 1989 to September 1992, the right gastroepiploic artery (GEA) in combination with the internal mammary artery (IMA) was used in 300 patients who underwent coronary artery bypass grafting. The GEA was the primary choice in preference to a saphenous vein. There were 263 men and 37 women, ranging in age from 31 to 77 years (mean age 58.2 years). Thirty-nine patients (13%) underwent previous bypass procedures with vein grafts. In 150 patients (50%) we used the left IMA in conjunction with the GEA (in two patients combined with a vein graft), and in 133 patients (44.3%) both IMA's were used with the GEA in one operation. In 17 patients (5.7%) we used the GEA as a single graft. Revascularization in 9 patients (3%) was combined with another cardiac procedure; three times an aortic valve replacement, two mitral valve repairs, and four resections of an aneurysm of the left ventricle. Ten patients died in hospital (3.3%; 70% CL 2.3-4.8%); two cases were directly related to the GEA. There was no late mortality. Four patients had to be operated again; one had a new stenosis in a previously not stenosed coronary vessel, one patient due to mitral and tricuspid valve endocarditis, one patient with an open GEA had an occlusion of the left IMA, and one with a closure of a single GEA was reoperated with a right IMA. Eighty patients were re-catheterized 1 to 18 months postoperatively (mean 9 months). Graft patency in GEA increased from 77% in the first semester of the program to 94% in the fourth semester. Patency of the internal mammary grafts was 96%. We conclude that graft failure of the GEA was related to a "learning curve". Furthermore, the GEA may well be the graft of choice in conjunction with the internal mammary arteries.

*By Invitation

22. Donor Shortage in Heart Transplantation: Is Extension of Donor Age Limits Justified?

UGOLINO LIVI, M.D. *, UBERTO B. BORTOLOTTI, M.D. *,

GIOVANBATTISTA LUCIANI, M.D. *,

GIOVANNI BOFFA, M.D. *, GAETANO THIENE, M.D.*

and DINO CASAROTTO, M.D.*

Padova, Italy

Sponsored by: Norman E. Shumway, M.D., Stanford, California

Chronic shortage of donor organs for HTx led us to extend donor age limits. To verify the effectiveness of such policy we have compared the results of HTx in 40 patients (pts) using donors >40 years (yrs) (Group 1) with 69 pts >50 yrs of age who had HTx using donors <40 yrs (Group 2) from November 1985 to September 1992. The 2 groups were comparable in terms of mean recipient age, recipient and donor sex and indication for HTx. Mean donor age was 46 ± 4 yrs in Group 1 (range, 40 to 59) and 23 ± 7 yrs in Group 2 (range, 8 to 38) ($p < 0.01$). In Group 1 cerebrovascular accidents were more common as cause of donor death (60% vs 16%, $p < 0.01$), while no difference was found in ischemic time (139 ± 41 vs 151 ± 52 min, $p = \text{ns}$). There were 4 early (<30 days) deaths in Group 1 (10%) and 10 in Group 2 (14%) ($p = \text{ns}$); 2 pts (5%) died late post-HTx in Group 1 and 3 (4%) in Group 2 ($p = \text{ns}$). Acute graft failure leading to death or re-HTx was more frequent in Group 1 (10% vs 6%, $p < 0.01$). Mean follow-up is 29 ± 20 months (range, 1 to 72) in Group 1 and 30 ± 20 months (range, 2 to 74) in Group 2 ($p = \text{ns}$). Actuarial survival is $86 \pm 6\%$ vs $83 \pm 7\%$ and $84 \pm 7\%$ vs $80 \pm 8\%$ ($p = \text{ns}$) at 1 and 4 yrs in Group 1 vs Group 2, respectively. Angiographic control has shown a similar left ventricular ejection fraction at 1 ($59 \pm 14\%$ vs $63 \pm 10\%$) and 4 yrs ($66 \pm 14\%$ vs $62 \pm 10\%$) ($p = \text{ns}$). However, Group 2 pts had a higher freedom from coronary artery disease (CAD) of any degree at 4 yrs ($84 \pm 7\%$ vs $75 \pm 8\%$, $p < 0.01$).

Donors >40 yrs of age can be used for HTx with mid-term results comparable to that of younger donors. A higher incidence of CAD and acute graft failure seems not to affect survival after HTx with donors >40 years, but the impact of CAD on the performance of older grafts must be assessed at longer follow-up.

*By Invitation

23. Seven Years Experience With Bridging to Cardiac Transplantation

D. GLENN PENNINGTON, M.D.,

LAWRENCE R. McBRIDE, M.D. *, PAMELA S. PEIGH, M.D. *,

LESLIE MILLER, M.D. * and

MARC T. SWARTZ, B.A. *

St. Louis, Missouri

Although bridging to cardiac transplantation has become a therapeutic option for transplant candidates who deteriorate while awaiting a donor heart, short term efficacy has not

been proven and long term survival has not been reported. We retrospectively reviewed 42 patients (pts) who had circulatory assist devices placed as a bridge to transplant between May 1985 and July 1992. The 33 men and 9 women ranged in age from 12-65 years (mean 43 years). Thirty pts were supported with Thoratec (17 left ventricular, 13 biventricular), 10 with Novacor and 2 with Jarvik J-7-70 devices. The duration of device support was from 4 hours - 440 days (mean 47 days). Fourteen pts were not transplanted because of infection (10 pts), renal failure (5 pts), bleeding (9 pts), cerebrovascular accident (3 pts) and died. Two pts were weaned from support and survived without transplantation. Twenty-six pts were transplanted, with 25 survivors (96%). Overall survival is 64% (27/42). Duration of survival has ranged from 3-90 months (mean 35.3 months). Of the 27 survivors, there were 3 late deaths (all transplants) at 4, 6 and 14 months. Post-transplant actuarial survival at 1, 5 and 7 years is 86%, 81% and 81%. Twenty-three of the 24 pts presently alive are NYHA functional status I.

These data demonstrate the short and long term efficacy of bridging to transplantation with circulatory support devices. The excellent survival rates and full functional recovery of transplanted patients ensures that donor organs are not being "wasted" on the sickest patients.

4:40 p.m. EXECUTIVE SESSION (Members Only)

7:00 p.m. ANNUAL DINNER/DANCE Black Tie (Optional)

Regency Ballroom

*By Invitation

TUESDAY AFTERNOON, April 27, 1993

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

Grand Ballroom D

Moderators: L. Penfield Faber, M.D.

J. Kent Trinkle, M.D.

24. Determinants of Outcome for Lung Transplantation in Cystic Fibrosis

*HANI SHENNIB, M.D. *, GILBERT MASSARD, M.D. *,*

*MICHAEL P. KAYE, M.D., JOHN WALLWORK, M.D. *,*

*MICHEL NOIRCLERC, M.D. *, DA VID MULDER, M.D.*

and BRUCE REITZ, M.D. Montreal, Canada

Cystic Fibrosis (CF) is currently the commonest indication for bilateral lung replacement. The choice of patient, procedure and post-operative management is controversial. We analyzed data on 227 CF transplant recipients reported to the International Heart and Lung Transplant Registry as of December 1991. There were 156 heart/lung (HLT) and 71 bilateral lung transplants (BLT) [29 En bloc (EB) and 42 sequential single (SL) transplants]. Recipient's age, sex, CMV and functional status, type of procedure and site of transplantation (North America/United Kingdom), donor age, CMV match, ischemia time and whether induction steroids and cytolytic therapy were used or not, were examined as univariate and multivariate determinants of outcome. Overall, one year survival was 68.5% for HLT and 60% for BLT. One year survival of HLT in U.K. (74%) was significantly better than in North America (NA) (45%) ($p < 0.01$) where less HLT were done. Patient characteristics were similar, however ischemia time was longer in U.K. (204 ± 60 min) than in NA (173 ± 72 min) ($p < 0.025$). Bilateral lung transplants, on the other hand, did much better at 1 year in NA (63%) than in U.K. (33%). However, much smaller numbers were done in the U.K. to allow valid statistical testing. On further analysis of North American data, 1 year survival of SL (71%) were noted

to be superior to EB transplants (51%) and patients with NYH-III or less functional status (72%) did better than NYH-IV patients (29%) ($p < 0.01$). Steroid administration was associated with significant ($p < 0.05$) improvement in survival (steroid 64%, no steroids 37%) while cytolytic therapy had no effect. Results also appeared to improve with time ($p < 0.03$). As most mortality occurred in the early post-operative period [EB (41%), HLT (22%), SL (17%)], we conclude that first year survival in CF transplant recipients is determined primarily by 1) The type of surgical procedure and where it is performed i.e. center's experience, 2) Pre-operative functional status of recipient and, 3) Early post-operative administration of steroids. In North America, the best results were achieved with bilateral sequential lung transplantation of patients with NYH-III or less functional status and with early post-operative use of steroids.

*By Invitation

25. Anastomotic Pitfalls in Lung Transplantation

*BARTLEY P. GRIFFITH, M.D., MITCHELL J. MAGEE, M.D. *,*

*REMI HOUEL, M.D. *, IVAN F. GONZALES, M.D. *,*

*JOHN M. ARMITAGE, M.D. * and
ROBERT J. KEENAN, M.D. **

Pittsburgh, Pennsylvania

While airway, arterial, and venous connections required for lung transplantation appear simple, in practice we have encountered morbid early stenosis and obstructions which are now avoided by technical modifications gradually made since 1985 in 184 cases (60 SL and 74 DL).

Our eight initial DL procedures were performed with tracheal anastomoses and omental wraps, but ischemic disruption, a 75% (6/8) rate of complications, resulted in our subsequent use of bi-bronchial connections (Table). 192 bronchial anastomoses (BA) have been reviewed (60 SL, 66 DL). While all have been constructed between the donor trimmed to 1-2 rings above the upper lobe origin and host divided at its emergence from the mediastinum, the suture technique has evolved. 9/28 (32%) early BA with end-to-end suture and intercostal muscle wrap developed ischemic or stenotic complications, but the telescoping technique without wrap in 164 BA has reduced the problem to 12% (17/164). Twelve anastomoses required temporary intraluminal stenting. Because the telescopic method using horizontal mattress suture has rarely been associated with an obstructing flange of invaginated cartilage, it has been replaced by a telescoping suture figure-of-eight which holds the invaginated rings closely to the outer wall.

AIRWAY ANASTOMOSES

	N	Ischemia	Dehiscence	Stenosis	TOTAL
TRACHEAL					
End-to-End	8	47% (3)	47% (3)	-0-	75% (6)
BRONCHIAL					
End-to-End	28	13% (4)	13% (4)	4% (1)	32% (9)

Telescoping	164	7% (11)	-0-	5% (8)	12% (19)
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Vascular anastomotic obstructions have occurred in 6 arterial (excessive length 4, restrictive suture/clot 2) and 3 venous (excessive length 1, restrictive/clot 2) connections. Suspicion of arterial obstruction has been prompted by persisting pulmonary hypertension and reduced flow to the allograft measured by postoperative nuclear scan and widened A-a gradient. Venous obstructions are suggested by persisting radiographic and clinical pulmonary; edema. Currently, donor and recipient arteries are shortened to within 2-3 cm of the hilum and mediastinum respectively, and the donor venous atrial cuffs are trimmed to a muscle border of only 5 mm. The arteries are divided to maximize circumference of the suture line which is now interrupted.

Modifications of earlier techniques have improved our early success in lung transplantation and might be considered by others entering this demanding field.

*By Invitation

26. Results of Single and Bilateral Lung Transplantation in 130 Consecutive Recipients

JOEL D. COOPER, M.D.,

G. ALEXANDER PATTERSON, M.D.

*and ELBERT P. TRULOCK, M.D.**

St. Louis, Missouri

From its inception in July, 1988 until July 31, 1992, 142 lung transplants were performed in 138 patients by our lung transplant group. Eight en bloc double lung transplants were performed in the initial year with 75% mortality, after which this procedure was abandoned in favor of the bilateral sequential technique. Experience with the remaining 134 single (SLT) or bilateral (BLT) transplants performed in 130 recipients forms the basis for this report. Seventy-three patients underwent SLT and 57 patients underwent BLT for the following indications: emphysema-68; cystic fibrosis (C.F.)-20; primary pulmonary hypertension (P.P.H.)/Eisenmenger's-20; idiopathic pulmonary fibrosis (I.P.F.)-14 and other diagnoses-8. Hospital mortality occurred in 11 patients (8%) and late mortality in an additional 12 (9%). One hundred and seven patients (82%) are currently alive with a mean survival time of 17 months. By the actuarial method one-year survival is 81% and two-year survival is 80%. Survival, and results by diagnostic groups are as follows:

SURVIVAL

Diagnosis	n	% alive	mean followup (months)	1 year actuarial survival
emphysema	68	82%	16	84%
C.F.	20	85%	12	81%
P.P.H./Eisenmenger's	20	85%	16	81%
I.P.F.	14	71%	15	68%

RESULTS

Diagnosis	6 minute walk (meters)		Room air PO2 (mmHg)	
	preop	1 year	preop	1 year

emphysema	266	595	56	89
C.F.	348	711	48	98
P.P.H./Eisenmenger's	194	596	60	82
I.P.F	261	550	53	76

The 11 hospital deaths resulted from cardiac failure (2); aspergillus infection (2); sepsis (3); ARDS (2), arrhythmia (1), and airway dehiscence (1). Bronchiolitis obliterans currently affects approximately 25% of survivors and was the direct or indirect cause of 6 out of the 12 late deaths.

Lung transplantation can now achieve early results similar to those obtained with the more established types of organ transplants. The major unsolved problems relate to morbidity and mortality associated with chronic immunosuppression and chronic rejection, and the acute shortage of donor organs.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

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

Grand Ballroom D

27. Surgical Management of Non Small Cell Carcinoma With N2 Disease

GOPI C. MANNAM, FRCS, PETER COLOSTRA W, FRCS*,*

DA VID K. KAPLAN, FRCS and*

*PANOS MECHAIL, M.D.**

London, England and Rhodes, Greece

Sponsored by: Thomas W. Shields, M.D., Chicago, Illinois

Between 1979 and 1989, 876 patients with Non Small Cell Carcinoma (NSCLC) were referred to our unit. One hundred and forty-six patients were judged not suitable for surgical treatment on clinical, radiological or bronchoscopic findings. Cervical mediastinoscopy and/or anterior mediastinotomy showed that 151 patients had metastases into the superior mediastinal lymph nodes (N2 disease) and were therefore deemed inoperable. Except for one patient who had single nodal station positive at mediastinoscopy, all other patients proceeding to thoracotomy, 578, were thought on the basis of Ct scan (89) and/or mediastinal exploration (59), not to have N2 disease. Despite our efforts to avoid surgery in patients with N2 disease, routine mediastinal node dissection showed that 149 patients had previously unsuspected N2 disease. Resection was possible in 130 (87.3%) by pneumonectomy (72), bilobectomy (7), lobectomy (49), or lesser resection (2). In three patients the resection was incomplete (2.3%), but in 127 cases a complete resection was performed (85%). The histology of tumors in these 149 patients showed, 72 were squamous cell carcinoma, 54 adenocarcinoma, 14 large cell carcinoma and 9 of mixed type.

Five patients died in hospital following thoracotomy. Complete follow-up was obtained in 109 patients and the mean follow-up period was 27.25 months (1-116). The actuarial 5 year survival for those having complete resection was 19.4%. Neither cell type nor the nodal station of the metastatic node influenced long-term survival. There was however, a statistically

significant difference favoring long-term survival in those patients with only one nodal station involved compared to those with more than one ($p < 0.033$).

Despite rigorous preoperative investigations it is possible to encounter mediastinal node metastasis first time at thoracotomy by routine mediastinal node dissection. We consider resection is justified in these patients who have already necessarily incurred the morbidity and mortality of thoracotomy, as long as complete resection is possible.

*By Invitation

28. Comparison of Survival and Lung Function Following Sleeve Lobectomy and Pneumonectomy for Lung Cancer

*HENNING A. GAISSERT, M.D. **

DOUGLAS J. MATHISEN, M.D., HERMES C. GRILLO, M.D.,

*ASHBY C. MONCURE, M.D., JOHN C. WAIN, M.D. **

and ALAN D. HILGENBERG, M.D.

Boston, Massachusetts

Sleeve lobectomy is a widely accepted bronchoplastic procedure for patients with normal and compromised lung function. Scarce information is available regarding postoperative lung function and comparative survival after pneumonectomy. We have performed 71 sleeve lobectomies for lung cancer. Pulmonary or cardiac function was compromised in 38 patients and normal in 33. Histology was squamous cell in 49, adenocarcinoma in 18, large cell in 3, and adenosquamous in 1. Resection involved the upper lobe in 47 patients (right 37, left 10), lower and middle lobe in 10, and bilobectomies in 14. Postsurgical stage was I in 28 patients (39 percent), II in 33 patients (47 percent), and III in 10 patients (14 percent).

Bronchoplastic procedures were compared to 53 patients undergoing pneumonectomy for lung cancer. Operative mortality for sleeve lobectomy was 2.8 percent compared to 7.5 percent for pneumonectomy. Cumulative 5-year survival following pneumonectomy was 42 percent vs. 40 percent for all sleeve lobectomies. In the bronchoplastic group, 5-year survival in patients with normal lung function was 57 percent vs. 32 percent for compromised function. Survival in NO disease was 52 percent vs. 36 percent in N1 disease. Survival following upper sleeve lobectomy was 44 percent and 40 percent for lower, middle, and bilobectomies.

Mean postoperative reduction in FEV1 was 8.6 percent in patients with compromised function and 10 percent in patients with normal function. Ventilation perfusion scans confirmed preservation of function in remaining pulmonary parenchyma.

Sleeve lobectomy is the procedure of choice for anatomically suitable lesions for patients with both normal and compromised lung function. Survival is comparable to pneumonectomy and superior for patients with normal lung function. Preservation of lung function in the remaining lobes is confirmed by our studies.

*By Invitation

29. Prospective Evaluation of Unilateral Adrenal Masses in Patients With Operable Non-Small Cell Lung Cancer: Impact of Magnetic Resonance Imaging

MICHAEL E. BURT, M.D., Ph.D., ROBERT HEELAN, M.D.*

DANIEL COIT, M.D. *,

PATRICIA M. McCORMACK, M.D.

and ROBERT J. GINSBERG, M.D.

New York, New York

With computed tomography (CT) in the staging of NSCLC, 4 percent of otherwise operable patients have been found to have a unilateral adrenal mass. Previous studies have suggested that MRI has the ability to differentiate between benign (adenoma or hyperplasia) and malignant adrenal masses. Since this differentiation is critical to treatment planning, we designed a prospective study to evaluate the efficacy of MRI in differentiating a benign from a malignant adrenal mass in patients with otherwise operable NSCLC.

Methods: All patients with potentially operable NSCLC were prospectively staged by history, physical examination, pulmonary function testing, cardiac evaluation, CT scan of the chest and upper abdomen (including the adrenals), CT head scan, and bone scan. All operable patients with a unilateral adrenal mass underwent respiratory and cardiac gated thin section MRI of the adrenals (1.5 Tesla GE signa system). One radiologist interpreted the MRI blinded and based on the T1 and T2 weighted images judged whether the adrenal mass was benign or malignant. The patients then underwent a percutaneous needle biopsy of the adrenal mass, if technically feasible. If the result of the needle biopsy was non-diagnostic, or if the biopsy was not feasible, an adrenalectomy through a posterior approach was performed. Data expressed as mean \pm SD. Differences were determined by Fisher exact test or Student's unpaired t-test. Significance defined as $p < 0.05$.

Results: Twenty-seven patients with a unilateral adrenal mass entered the study; there were 11 men and 16 women whose ages ranged from 42-75 yrs (median 58). Four patients had epidermoid and 23 adenocarcinoma. The locoregional stage was I in 8, II in 4, IIIA in 12, and IIIB in 3. Twenty-five completed the MRI (2 did not, due to claustrophobia). Five adrenal masses (19%) were metastatic NSCLC (adeno CA = 4, epidermoid = 1); 22 masses (81%) were benign (adenoma = 20, hyperplasia = 2). There were no significant differences in age, sex, histology, or locoregional stage between those with a benign versus a malignant mass. However, the malignant masses were significantly larger (3.8 ± 1.9 cm; range 2.5 - 7.1; median 3.1) than the benign (2.0 ± 0.4 cm, range 1.2 - 2.8; median 2.0) ($p = 0.002$). Of those having an MRI ($n = 25$), MRI correctly predicted a malignant mass in the four patients with a histologically confirmed metastasis from NSCLC. However, of the 21 histologically benign masses, the MRI was interpreted as benign in only 4 and malignant in 17. Therefore, although the false negative rate was 0%, the false positive rate was 81%.

Conclusion: Most adrenal masses in otherwise operable patients with NSCLC are benign. If during staging an adrenal mass is found, histologic diagnosis must be obtained. MRI is not useful in the differentiation of benign and malignant adrenal masses in patients with NSCLC.

*By Invitation

30. Treatment and Prognosis in Bronchial Carcinoids Involving Regional Lymph Nodes

*NAEL MARTINI, M.D., MUHAMMAD ZAMAN, M.D. *,*

MANJITS. BAINS, M.D., MICHAEL E. BURT, M.D., Ph.D.,

PATRICIA M. McCORMACK, M.D.,

VALERIE W. RUSCH, M.D. and

ROBERT J. GINSBERG, M.D.

New York, New York

Bronchial carcinoids constitute less than 5% of all lung tumors and 10-15% of these have regional lymph node metastases at diagnosis. Over a 40 year period (1953-92), 25 patients were surgically treated by us for bronchial carcinoids with metastases to regional lymph nodes (N1 or N2). The tumors were located centrally, involving main or lobar bronchi in 12 patients and were peripheral in 13. None had a carcinoid syndrome of MI disease. Histologically, the carcinoids were classified as typical in 12 and atypical (neuroendocrine carcinoma) in 13. The median age of patients with typical carcinoids was 42 years and for atypical carcinoids 62.

Pneumonectomy was performed in 11 patients, sleeve lobectomy in 1, lobectomy in 7 and bilobectomy in 6. All had a mediastinal lymph node dissection. At final staging, 10 had N1 disease and 15 had N2. The number of N1 or N2 patients was equally divided between the 2 types of carcinoids.

No adjuvant treatment was given to the 10 patients with N1 disease. External radiation therapy was given postoperatively to 9 of 15 N2 patients, and oral cyclophosphamide to 1. There was only 1 local recurrence (in a patient with N1 disease) and 7 distant metastases in liver, bone or brain (6 in patients with N2 and 1 in a patient with N1).

The overall 5-year survival (Kaplan-Meier) was 83% (median follow-up: 62 months). There was no difference in disease free survival between patients with N1 or N2 disease. However survival and recurrence rates differed between typical and atypical carcinoids. In typical carcinoids, the 5-year survival was 92% and the 5-year disease free survival 100% (the one recurrence occurred at 8 1/2 years). In atypical carcinoids, the 5-year survival was 73% ($p = .06$) and the 5-year disease free survival 57% ($p = .025$).

We conclude that complete resection is effective for bronchial carcinoids, despite the presence of regional lymph node metastases, and results in long-term survival. In this group of patients, recurrence appears more dependent on histologic subset than nodal status. There is no evidence that postoperative radiation therapy is beneficial and we are unable to assess the merit of systemic adjuvants since none had effective systemic treatment.

4:40 p.m. EXECUTIVE SESSION (Members Only)

7:00 p.m. ANNUAL DINNER/DANCE Black Tie (Optional)

Regency Ballroom

*By Invitation

TUESDAY AFTERNOON, April 27, 1993

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

Grand Ballroom F

Moderators: Edward L. Bove, M.D.

Thomas L. Spray, M.D.

31. Cardiopulmonary Bypass Significantly Impairs Surfactant Activity in Children

FRANCIS X. MCGOWAN, M.D. *, PEDRO J. DEL NIDO, M.D. *,

GEOFFREY KURLAND, M.D. *, MACHIKO Ikegami, Ph.D. *,

ETSURO K. MOTOYAMA, M.D. * and RALPH D. SIEWERS, M.D. *

Pittsburgh, Pennsylvania and Los Angeles, California

Sponsored by: Bartley P. Griffith, M.D., Pittsburgh, Pennsylvania

Pulmonary dysfunction following cardiopulmonary bypass (CPB) in children remains a primary source of morbidity and mortality, particularly in infants. The effects of cardiopulmonary bypass on surfactant activity and lung mechanics in children has not been directly evaluated. In experimental animal models of lung injury (endotoxin), surfactant activity has been shown to significantly decrease with loss of the surface active, high density large surfactant aggregates (LA), and an associated rise in the less active, low density small aggregates (SA), producing a decrease in the LA/SA ratio. To determine the effects of CPB on lung surfactant and lung mechanics we studied 12 children, ages 0.6 to 12 years, undergoing elective cardiac surgery for congenital heart disease. Pulmonary function testing, with deflation flow volume curves, was done to measure forced vital capacity (FVC) and maximum air flow at 25% lung volume ($V_{\max 25\%}$). Bronchoalveolar lavage was then performed to assay surfactant aggregates and lavage cell counts pre-CPB, 1 hr and 6 hrs post-CPB. CPB duration was 112 ± 28 min. The results are shown below:

	LA/SA ratio	FVC (l/sec)	$V_{\max 25\%}(\%)$	Cell Count (PMN's/ml)
PRE-CPB	6.8 \pm 1.0	1.4 \pm .3	70 \pm 9	7 \pm 5
1 hr POST	3.6 \pm 0.5*	1.0 \pm .2*	47 \pm 7*	35 \pm 11*
6 hrs POST#	1.5 \pm 0.3*	0.9 \pm .1*	52 \pm 7*	55 \pm 4*

(all values are mean \pm SE, * - $p < 0.05$ vs pre-CPB, n = 12 except for # where n = 3)

Along with the increase in PMN's there was a significant decline in lung monocytes in the lavage fluid post-CPB at both time points. Lung compliance was also significantly decreased by 6 hrs post-CPB.

We conclude that in children, cardiopulmonary bypass of even moderate duration exerts a deleterious effect on surfactant activity with an associated decline in lung mechanics. The effects of CPB on surfactant activity may be of even more importance in neonates undergoing open heart surgery due to their limited ability to produce surfactant.

*By Invitation

32. Evaluation of Cerebral Metabolism and Quantitative EEG Following Hypothermic Circulatory Arrest and Low Flow Cardiopulmonary Bypass

*CRAIG K. MEZROW, M.S. *, PETER K. MIDULLA, M.D. *,*

*ALIM. SADEGHI, M.D. *, ALEJANDRO GANDSAS, M.D. *,*

*ROSARIO ZAPPULLA, M.D. **

and RANDALL B. GRIEPP, M.D.

New York, New York

Although widely used for repair of complex cardiovascular pathology, long intervals of hypothermic circulatory arrest (HCA) and low flow cardiopulmonary bypass (LFCPB) may both result in cerebral injury (CI). This study examines cerebral hemodynamics, metabolism, and electrical activity in order to evaluate the relative risks of CI after 60 min of HCA at 8 C, 13 C and 18 C, compared with 60 min LFCPB at 18 C.

Twenty-four puppies were randomly assigned to one of 4 experimental groups, and centrally cooled to the appropriate temperature. Serial evaluations of quantitative EEG (QEEG), radioactive microsphere determinations of cerebral blood flow (CBF), calculations of cerebral oxygen consumption (CMRO₂), cerebral glucose consumption (CMR_{glu}), cerebral vascular resistance (CVR), cerebral oxygen extraction, systemic oxygen metabolism and systemic vascular resistance (SVR) were carried out. Measurements were obtained at baseline (37 C), at the end of cooling, at 30 C after rewarming, and at 2, 4, and 8 hrs after HCA or LFCPB. A $p < 0.05$ as determined by ANOVA was accepted as statistically significant.

At the end of cooling, CVR remained at baseline levels in all groups, but SVR was almost triple at 18 C, almost twice normal at 13 C, and 1 $\frac{1}{2}$ baseline at 8 C. CMRO₂ became progressively lower as temperature was reduced: it was only 5% of baseline at 8 C; 20% at 13 C; and 34% and 39% at 18 C. QEEG was silent in the 8 and 13 C groups, but significant slow wave activity was present at 18 C.

SVR and CMRO₂ returned to baseline values in all groups by 2 hrs after HCA or LFCPB, but CVR remained elevated at 2 and 4 hrs, not returning to baseline until 8 hrs after HCA or LFCPB.

All of the long-term survivors (20/24) appeared neurologically normal, with one exception: after HCA at 8 C, one animal had an unsteady gait. Comparison of QEEG preoperatively and 6 days postoperatively showed a significant increase in slow wave activity and decrease in fast wave activity in all animal groups. These changes were more pronounced after HCA and LFCPB at 18 C.

Although undetected postoperatively by simple behavioral and neurological assessment, significant differences in cerebral metabolism, vasomotor responses and QEEG do exist during and following HCA and LFCPB at various temperatures, and may be implicated in the occurrence of CI. The data from this study suggest that for an interval of 60 minutes, HCA at 8 or 13 C may provide cerebral protection superior to HCA or LFCPB at 18 C.

*By Invitation

33. Energy Expenditure in Children With Congenital Heart Disease Before and After Cardiac Surgery

*IAN M. MITCHELL, M.D. *, PETER S. W. DAVIES, Ph.D. *,*

JANICE M.E. DAY, JAMES C.S. POLLOCK, FRCS**

*and MORGAN P.O. JAMIESON, FRCS**

Glasgow, Scotland and Cambridge, England

Sponsored by: Professor D.J. Wheatley, Glasgow, Scotland

Poor growth and failure to thrive are common features of children with congenital heart disease; some degree of nutritional impairment being evident, even in seemingly asymptomatic patients. Whether this is the result of a poor intake, or whether it is due to an abnormally high basal metabolic rate, is unknown, yet the state of nutrition has a profound effect on the metabolic response to injury and strongly influences the outcome from surgery. In nutritionally compromised children it is clearly important to recommence feeding at an early stage, but the exact energy requirements in the postoperative period are also unknown. The aim of this study was therefore to measure the pre- and postoperative energy requirements in children with congenital heart disease, to determine not only why growth should be poor, but also the caloric cost of cardiac surgery.

Seventeen children undergoing cardiac surgery were studied aged (mean age 15.8 months, range 4 - 33 months), cardiopulmonary bypass being required in 14. Each child was given two oral doses of doubly labelled water (H_2^{18}O and $^2\text{H}_2\text{O}$), the first, one week prior to operation and the second, 6 hours after the end of surgery. By measuring the relative concentrations of each isotope in daily urine samples, carbon dioxide production and hence energy expenditure could be calculated. Preoperative results demonstrate that energy expenditure was essentially normal in 5 children, elevated in 8 and low in 4, suggesting that an elevated basal metabolic rate is an important factor in the observed failure to thrive. In the week following surgery however, total body water fell by approximately 5% and energy requirements by 36% (range 4% to 73%), reaching values below normal for healthy (non-operated) children, irrespective of whether or not cardiopulmonary bypass had been required. These results suggest that the stress of surgery leads to smaller energy requirements than have previously been thought.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

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

Grand Ballroom F

34. The Effect of the Hypoplastic Left Heart Class on Outcomes in Interrupted Aortic Arch

*RICHARD A. JONAS, M.D., JAN M. QUAEGBEUR, M.D. *,*

GEORGE R. DAICOFF, M.D.,

EUGENE H. BLACKSTONE, M.D.

and JOHN W. KIRKLIN, M.D.

Boston, Massachusetts, New York, New York, St. Petersburg,

Among 232 neonates with interrupted aortic arch (IAA) entering 30 institutions for treatment (1987-1992), 167 had coexisting VSD. Considering aortic atresia to be hypoplastic left heart class IV (HLH IV), 10 of the 167 pre-repair had HLH III (two additional important left heart obstructions, such as supravalvar, valvar, or subvalvar mitral narrowing, left ventricular hypoplasia, subvalvar, valvar, or annular aortic valve narrowing, or ascending aortic hypoplasia) and 37 had HLH II (one additional important left heart obstruction). The HLH assignment strongly (negatively) correlated with the pre-repair Z-values (echocardiography) of the diameter of the subvalvar area, the anulus, and the ascending aorta (for the 50th percentile, -6.3, -4.4, and -4.1, respectively). In some patients the subvalvar and annular Z-values decreased soon after repair.

Twenty-two different initial procedures were performed! One, 12, 24, and 36 month survival after the initial procedure was 75%, 66%, 65% and 64%. Risk factors (hazard function domain) were identified (Table); the strength of some variables is illustrated in the nomograms. If Z-value of the aortic anulus replaces "HLH class" in the equation, its strength is shown.

Incremental Risk Factors for Death		
After Arch Repair		P-value
<hr/>		
Patient		
Demographic		
(Lower)	Birthweight	.0001
(Younger)	Age at repair	.0003
	Female gender	.08
Morphologic		
(Higher)	HLH class (I-III) ¹	.0006
(Smaller)	Size of VSD	.0002
	IAA type B	.09
Institutional		
	H	< .0001
	B	.0004
Procedural		
	DKS ²	.001
<hr/>		

Thus, when HLH II or III coexists with IAA and VSD, or when the Z-value of the anulus is %□ about -4, an initial classical repair is reasonable, but important LV-aortic gradients may require later Konno type repair. But, when instead a DKS repair is performed initially, early survival is lessened even though the procedure may not be necessary (HLH I). The resolution of this dilemma requires continuation of the study and longterm follow-up, and re-analysis in 3-5 years.

*By Invitation

35. Unifocalization in Pulmonary Atresia With Ventricular Septal Defect and Major Aorto-Pulmonary Collateral Arteries

*TOSHIKATSU YAGIHARA, M.D. *, FUMIO YAMANO, M.D. *, KYOICHI NISHIGAKI, M.D. *, OSAMUMATSUKI, M.D. *, HIDEKI UEMURA, M.D. * and YASUNARU KAWASHIMA, M.D.*

Osaka, Japan

For the purpose of extending the indication of corrective surgery for patients with pulmonary atresia, ventricular septal defect and major aorto-pulmonary collateral arteries (MAPCA), we have applied the surgical procedures to unifocalize the pulmonary blood supply. Since December 1985, 48 patients underwent unifocalization at the age from 3 months to 26 years with an average of 5.6 years.

Eighty staged unifocalizations were performed in 47 patients, while one patient received one stage repair. Procedures included anastomosis between central pulmonary artery (CPA) and intrapulmonary artery (IPA) supplied by MAPCA, directly or with interposition of graft (n = 32), pulmonary angioplasty (n = 7), creation of CPA with heterogenous pericardial roll (n = 33), and others (n = 8). In the patients group without CPA or with vestigial small CPA and needed heterogenous pericardial roll, one end of the pericardial roll was anastomosed to IPA and another end was fixed at mediastinal pleura anteriorly with a shunt from the subclavian artery. Anastomosis was made mainly inside the lung dividing in-terlobular fissure. In addition to these procedures, central palliation which aids a small CPA to grow was performed in 3 patients. There were 5 operative and 5 late deaths out of these 83 procedures.

Twenty-five patients underwent intracardiac repair after the staged unifocalization. In 15 patients, confluency of surgically created CPA's were achieved as well as the reconstruction of right ventricular outflow tract using external conduit. Six patients are ready for correction. Right to left ventricular systolic pressure ratio immediately after intracardiac repair in 26 patients including one who underwent one stage repair ranged 0.24 to 0.91 with an average of 0.56 ± 0.18 . There were 1 operative and 4 late deaths 2 months to 2 years after surgery, which related to bronchial hemorrhage or respiratory infection.

Surgical unifocalization is possibly a beneficial procedure as a part of correction for the patients with pulmonary atresia, ventricular septal defect and MAPCA, even for patients without CPA.

*By Invitation

36. Extended Aortic Valvuloplasty for Recurrent Aortic Valvar Stenosis and Regurgitation in Children

*MICHEL N. ILBAWI, M.D., JOSEPH CASPI, M.D. *,
DAVID A. ROBERSON, M.D. *, R. ABDULLAH, M.D. *,
WILLIAM PICCIONE, M.D. * and
HASSAN NAJAFI, M.D.*

Oak Lawn, Illinois

Recurrent significant aortic valvar stenosis and/or regurgitation (AVSR) following balloon or open valvotomy in pediatric patients, often requires aortic valve replacement (AVR). In an attempt to preserve the aortic valve, extended aortic valvuloplasty repair (EAV) was performed in 13 children with recurrent AVSR from 1/89 to 5/92. Previous related procedures were one or more open aortic valvotomy (n = 9), balloon valvotomy (n = 3), and repair of iatrogenic valve tear (n = 1). Mean age at the time of the EAV was 4 ± 1.4 yrs. Mean pressure gradient (MPG) across the aortic valve was 53 ± 12 torr. Regurgitation was moderate (Grade 2 to 3) in 7, and severe (Grade 4) in 6 pts. EAV techniques consisted of: thinning of valve leaflets (n = 7), augmentation of scarred retracted or torn leaflets using autologous pericardium (n = 5), release of rudimentary commissure from aortic wall (n = 2), resuspension of valve commissure (n = 8), extension of the valvotomy incision into the aortic wall on both sides of the commissure (n = 12), and patch repair of sinus of valsalva perforation (n = 1). There was no operative death or morbidity. Postoperative MPG assessed by most recent Doppler echocardiography or cardiac catheterization at a follow-up of 24 ± 9 mos was 16 ± 5 mmHg ($p < 0.01$ vs preoperative). Aortic regurgitation was absent in 9, mild in 2, and moderate to severe requiring subsequent AVR in 2. This short term experience indicates that EAV is a safe and effective surgical approach that minimizes the need for AVR in children with significant recurrent AVSR.

*By Invitation

37. Late Outcome Following Repair of Supravalvar Aortic Stenosis

*JACQUES A. VAN SON, M.D. **

GORDON K. DANIELSON, M.D., DWIGHT C. McGOON, M.D.,

*HARTZELL V. SCHAFF, M.D., AMITA RASTOGI, M.D. **

and FRANCISCO J. PUGA, M.D.

Rochester, Minnesota

To determine the long-term outcome we reviewed 79 patients who had repair of localized (group A) (n = 67) or diffuse (group B) (n = 12) supravalvar aortic stenosis (SAS) from 1956 to 1992, including 30 patients with the Williams-Beuren syndrome. In group A the aortic root was enlarged with a diamond-shaped (DS) patch (n = 61) or a pantaloon-shaped (PS) patch (n = 6). In group B patch enlargement of the aorta was confined to the root (n = 4) or extended into the ascending aorta or aortic arch (n = 7); 1 patient had a left ventricular-aortic conduit. Two patients in group B in whom the patch enlargement was confined to the aortic root died intraoperatively (2.5%). During follow-up extending to 33 years there were 5 late deaths (2 related to coronary pathology) in group A and 1 in group B. There was no significant difference between patients with a DS or PS patch in terms of late gradient (mean \pm SEM: 18 ± 2 mm Hg vs 9 ± 4 mm Hg, respectively) and aortic insufficiency (AI). By Cox multivariate model independent predictors of late death were concomitant aortic valvotomy ($p = 0.04$) and presence of preoperative AI ($p = 0.006$). In group B, survival was better in patients who received an extended patch vs aortic root patch ($p = 0.02$). Risk factors for the development of late AI by univariate analysis were: bicuspid aortic valve ($p = 0.02$), valvar aortic stenosis ($p = 0.01$), absence of Williams-Beuren syndrome ($p = 0.007$), and concomitant aortic valvotomy ($p = 0.006$). On multivariate analysis the latter 2 factors were independent predictors of late AI (both $p < 0.01$). Conclusions: 1. Both the DS and PS patch techniques provide excellent long-term relief of localized SAS; 2. In diffuse SAS aortic enlargement should be extended into the ascending aorta or beyond if necessary; 3. Concomitant aortic valvotomy may be associated with development of late AI as well as increased late death; 4. Early surgical intervention prior to development of significant AI or coronary artery disease may improve the long-term survival.

4:40 p.m. EXECUTIVE SESSION (Members Only)
7:00 p.m. ANNUAL DINNER/DANCE Black Tie (Optional)

Regency Ballroom

*By Invitation

WEDNESDAY MORNING, APRIL 28, 1993

7:30 a.m. FORUM SESSION II - Grand Ballroom

GENERAL THORACIC SURGERY

Moderators: Douglas J. Mathisen, M.D.

Martin F. McKneally, M.D.

F10. Cold Ischemia and Reperfusion Each Produce Pulmonary Vasomotor Dysfunction in the Transplanted Lung

DAVID A. FULLERTON, M.D. *, MAX B. MITCHELL, M.D. *,

ROBERT C. McINTYRE, M.D. *,

ANIBAN BANERJEE, Ph.D. *, DAVID N. CAMPBELL, M.D. *,

ALDEN H. HARKEN, M.D. and

FREDERICK L. GROVER, M.D.

Denver, Colorado

BACKGROUND Pulmonary vascular resistance (PVR) is significantly increased in the transplanted lung. Ischemia and/or reperfusion incurred by the transplanted lung may produce pulmonary vasomotor dysfunction, which may in turn contribute to increased PVR. Therefore, the following pulmonary vasomotor control mechanisms were studied in a canine model of autologous lung transplantation and each related to **Cold Ischemia and Reperfusion**: (1) Endothelial-dependent cGMP-mediated dilation (response to Acetylcholine, ACh) (2) Endothelial-independent cGMP-mediated dilation (response to Nitroprusside, NP) and (3) Vascular smooth muscle Beta adrenergic cAMP-mediated dilation (response to Isoproterenol, ISO).

METHODS Autologous lung transplantation was performed in 5 dogs. After infusing PGE₁ (10µ/kg), modified Euro-Collins Solution (4°C, 30cc/kg) was infused into the right pulmonary artery. The right lung was removed, stored in saline (4°C) for 3 hrs, then reimplanted. 2 third order pulmonary arteries were dissected from each lung at each of 3 times: **Control** (immediately post harvest), **Cold Ischemia** (3 hrs in saline 4°C), **Cold Ischemia plus Reperfusion** (1 hr after lung reimplantation). The vasorelaxing effects of ACh 10⁻⁶M, NP 10⁻⁶M, and ISO 10⁻⁶M were studied in isolated pulmonary arterial rings, suspended on fine wire tensiometers in individual organ chambers. Statistical analysis was by ANOVA (Scheffe's F-test).

RESULTS Reperfusion produced endothelial dysfunction as the response to ACh, but not NP, was impaired. However, vascular smooth muscle Beta adrenergic relaxation (response to ISO) was impaired by cold ischemia alone, and worsened by reperfusion.

CONCLUSION Cold ischemia and reperfusion each produce different patterns of pulmonary vasomotor dysfunction. Cumulatively, such dysfunction may contribute to increased PVR in the transplanted lung.

*By Invitation

F11 Successful Canine Bilateral Single Lung Transplantation After 21-Hour Lung Preservation

*HIROSHI DA TE, M.D. *, SADANOBU IZUMI, M.D. *,*

*YOSHIO MIYADE, M.D. *, AKIO ANDO, M.D. *,*

*NOBUYOSHISHIMIZU, M.D. * and*

*SHIGERU TERAMOTO, M.D. *,*

Okayama, Japan

Sponsored by: Joel D. Cooper, M.D., St. Louis, Missouri

We utilized a bilateral single lung transplantation (BSLT) model to confirm the results of lung preservation studies previously obtained in a canine single lung transplant model. The donor lungs were flushed with low potassium dextran glucose (LPDG) solution, inflated with 100% oxygen and preserved at 8°C. After the preservation period, BSLT was performed without using a cardiopulmonary bypass. In group I (n = 5), the mean ischemic time of the right lung was 3 hrs 5 min ± 27 min and for the left lung 6 hrs 2 min ± 33 min. In group II (n = 5), they were 18 hrs 8 min ± 27 min and 21 hrs 42 min ± 42 min, respectively. Following BSLT, animals were maintained on a ventilator for 12 hours and pulmonary function was monitored. All five cases in group I and four of five cases in group II completed BSLT and survived for 12 hours with excellent lung function as shown below:

PaO₂	1hr	2hrs	4hrs	6hrs	8hrs	10hrs	12hrs
Group I	538 ± 44	563 ± 49	559 ± 15	534 ± 14	528 ± 36	569 ± 22	590 ± 18 mmHg
Group II	565 ± 67	534 ± 96	510 ± 99	498 ± 99	507 ± 99	623 ± 19	615 ± 18 mmHg
mPAP							
Group I	27 ± 4	27 ± 3	27 ± 3	27 ± 4	27 ± 3	27 ± 3	27 ± 3 mmHg
Group II	23 ± 3	23 ± 2	24 ± 3	22 ± 3	22 ± 3	20 ± 4	20 ± 4 mmHg

(FiO₂ = 1.0, mPAP: mean pulmonary artery pressure)

Following the 12 hour period of post-transplant assessment, the animals were extubated. All animals in each group showed satisfactory spontaneous ventilation and were returned to the cage. FK506 (0.1 mg/kg) was given intramuscularly every day. Despite the excellent immediate graft function, survival of the animal was modest. The longest survival time was 4 days in group I and 8 days in group II. In no case did postmortem examination of the lungs reveal any overt signs of lung injury. The animal in group II which died of bronchial dehiscence at 8 days showed excellent arterial blood gas on room air for 6 days as shown below:

	1 day	3 day	6 day
PaO₂	79.3	75.0	70.5 mmHg

PaCO₂ 36.1 37.9 36.8 mmHg

We conclude that lungs flushed with LPDG solution, inflated with 100% oxygen and preserved at 8°C for 21 hours provides excellent immediate and very satisfactory early graft function in a canine BSLT model in which the animal is totally dependent on the function of transplanted lung tissue.

*By Invitation

F12. Detection of Canine Allograft Lung Rejection by Pulmonary Lymphoscintigraphy

*RENATO RUGGIERO, M.D. *, ROBERT FIETSAM, M.D. *,*

*GREGORY A. THOMAS, M.D. *,*

*JAROSLAW MUZ, M.D. *, THOMAS A. KOWAL, P.A. *,*

*JONA THAN L. MYLES, M.D. *,*

LARRY W. STEPHENSON, M.D. AND

*FRANK A BACIEWICZ, M.D. **

Detroit, Michigan

We previously demonstrated that lymphoscintigraphy could be used to study pulmonary lymphatic flow. Radiocolloids, high molecular weight protein tagged with radioactive markers, are injected percutaneously in the periphery of the lung, these molecules enter the lymph, are transported via lymphatic channels and concentrated in the tributary hilar and mediastinal lymph nodes where they can be detected by nuclear scan.

The goal of this study was to determine whether pulmonary lymphoscintigraphy could be used to detect allograft rejection after lung transplantation.

Thirteen mongrel dogs underwent left lung allotransplantation using standard surgical techniques. Cyclosporine 15 mg/kg/day and azathioprine 1 mg/kg/day were given orally for postoperative immunosuppression. Lymphoscintigraphic studies were obtained one week after the surgery and then at weekly intervals. Animals were divided into groups A and B. In group A (n = 5), immunosuppression was continued until the animals death (n = 1) or until they were euthanized at six weeks. Lymphoscintigraphy demonstrated re-establishment of lymphatic drainage between the lung graft and the mediastinum in all (n = 5) the animals two to four weeks after the transplant. In Group B (n = 8), immunosuppressive medications were discontinued after re-establishment of graft lymphatic drainage was documented by two consecutive lymphoscintigraphic studies. The dogs continued to be studied with weekly scans. In this group, lymphatic drainage from the lung graft to the mediastinum disappeared between one and three weeks following discontinuation of the immunosuppressive medications. Rejection was diagnosed clinically and confirmed histologically with open lung biopsies in group B. The difference in disappearance of lymphatic drainage between groups A and B was statistically significant (p<0.001).

This study shows that canine allograft lung rejection is associated with disappearance of lymphatic drainage from the lung graft to the mediastinum. The significance of this finding is not yet clear but was documented by pulmonary lymphoscintigraphy, a minimally invasive technique that can be easily repeated. Pulmonary lymphoscintigraphy may be useful for early clinical detection of lung allograft rejection.

*By Invitation

F13. Isolated Single Lung Perfusion With Doxorubicin is Effective in the Treatment of Metastatic Sarcoma in the Rat

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MICHAEL E. BURT, M.D., Ph.D.

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Currently the only effective therapy for patients with soft-tissue sarcoma metastatic to the lung is resection, with five year survival after resection approximating 25%. Systemic chemotherapy has not impacted on survival. We have previously shown that isolated single lung perfusion (ILP) with dox-orubicin (DOX), in the rat, results in significantly higher tissue concentration than systemic injection. We evaluated the efficacy and toxicity of ILP with DOX and the treatment of rat sarcoma lung metastases.

Methods: Experiment 1. Three groups of F344 (n = 5) rats were randomized to left ILP for 10 min. at 0.5 ml/min, with perfusate concentrations of 320, 480 or 640 ug/ml of DOX in saline. Right pneumonectomy was performed 21 days post-perfusion. Experiment 2. Twenty F344 rats had intravenous injection of 10⁷ methylcholanthrene-induced sarcoma cells. Six days post-injection, 10 animals were randomized to ILP with 320 ug/ml of DOX and 10 animals to ILP with saline. On day 20 all animals were sacrificed and lungs were examined for number of metastases. Statistical analysis by Fisher exact test. Significance defined as p<0.05.

Results: Experiment 1. Survival after left ILP followed by right pneumonectomy was 80%, 0%, 0% with 320, 480, 640 ug/ml of DOX, respectively (p<0.05). Experiment 2. Three animals died post-operatively, 2 from the saline group and one from the DOX group; one animal from the DOX group was excluded due to the presence of mediastinal tumor noticed during ILP. Seven out of 8 animals perfused with DOX had total clearing of the left lung and all animals perfused with saline had the left lung fully replaced with tumor (p<0.001). All right unperfused lungs were completely replaced.

Conclusion: Isolated single left lung perfusion with 320 ug/ml of DOX does not cause morbid lung injury since 80% of animals perfused survived contralateral pneumonectomy. ILP with DOX is effective in eradicating metastatic sarcoma in this model.

*By Invitation

F14. Tumor Necrosis Factor Induces Doxorubicin Resistance in Lung Cancer

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*HELEN W. POGREBNIAK, M.D. **

and HARVEY I. PASS, M.D.

Bethesda, Maryland

Cytokines can alter the cell cycle (CC) of tumor cells and aid hemopoietic stem cell recovery from chemotherapy. We theorized that cytokines might alter the chemosensitivity of cancer cells by CC modulation. METHODS: A549 human lung cancer cells were exposed for 24 hours to TNF, IL-1, or IL-6. CC kinetics were then measured by flow cytometry (n = 4).

Six day growth of TNF exposed A549 was measured via the MTT assay (n = 3). A549 cells were treated for 24 hours with 1 µg/ml TNF or control media (CM), then exposed to 1 hour of DOX (n = 4), cis-platinum (CDDP, n = 3), or mitomycin C (MITO, n = 3), and 8 day survival fractions measured by clonal assay. Intracellular DOX levels were determined by fluorescence spec-trophotometry to rule out any effects on [DOX] by TNF preexposure.

RESULTS: TnF shifted A549 from S phase to (G₀/G₁) as seen below:

CELL CYCLE EFFECTS OF TNF ON A549 CELLS				
TNF □g/ml	0	0.001	0.01	1.0
% cells in G ₀ G ₁	53 ± 1	54 ± 3	62 ± 4*	65 ± 1*
% cells in S phase	40 ± 3	42 ± 5	34 ± 3	31 ± 1*

*p<0.05 from 0 /□g/ml TNF

This shift to the resting phase of the CC was verified by inhibition of A549 growth in TNF over 6 days (1.9 ±.2OD vs 1.4 ± .1 OD, 0 vs 1 µg/ml TNF, p₂<.05). TNF pre-treatment rendered A549 resistant to DOX, a CC- specific drug, as seen below.

SURVIVAL OF DOX-EXPOSED A549 ± TNF PRETREATMENT					
[DOX]					
□M	0.50	1.00	2.50	5.0	10.0
-TNF	0.80 ± .13	0.53 ± .09	0.09 ± .04	0.033 ± .02	0.011 ±.004
+TNF	0.90 ± .11	0.57 ±.09	0.34 ± .08*	0.170 ± .04*	0.034 ± .007*

*p<0.05, CM vs TNF

TNF did not affect intracellular DOX levels. Moreover, TNF did not affect chemosensitivity to CDDP or MITO, which are not CC-specific.

CONCLUSIONS: (1) TNF induces chemoresistance in lung cancer cells. (2) This chemoresistance may involve shift of cells into the CC resting phase. (3) Local elaboration of TNF by tumor associated macrophages may induce chemoresistance and could in part explain treatment failure in lung cancer.

*By Invitation

F15. A High Frequency of p53 Mutations Occurs in Patients With Squamous Cell Carcinoma of the Esophagus

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*CAROL YN E. REED, M.D. *, JONA THAN S.*

*BROMBERG, M.D., Ph.D. *, ERIC T. EVERETT, M.S. *,*

ROBERTA L.D. DIKEMAN, B.S. and*

*PAUL L. BARON, M.D.**

Charleston, South Carolina

Sponsored by: Fred A. Crawford, Jr., M.D., Charleston,

Coastal South Carolina has one of the highest rates of esophageal cancer in the world. Although environmental exposures have been implicated in its development, their precise role in generating and promoting tumorigenesis has not been established. Mutations in the p53 tumor suppressor gene have been described in 35% of esophageal cancers from other high incidence areas, namely, China and Southern France. Our study was designed to determine if patients with squamous cell carcinoma of the esophagus from the South Carolina Lowcountry have p53 mutations and whether the pattern of such mutations correlates with their environmental exposures. Specimens obtained by esophagoscopy were either grown in tissue culture or cryopreserved in liquid nitrogen. The total cellular RNA was extracted and reverse transcribed to cDNA. A 606 base pair segment of the expressed p53 gene spanning exons four through nine (the region with 98% of the known mutations) was selectively amplified by the polymerase chain reaction, cloned into pBluescript II KS + plasmids, grown in competent *E. coli*, and sequenced by a modification of the dideoxy random chain termination method. All eight patients has a significant history of heavy tobacco use:

Case	Race	Path	Base Change	Codon Number	Amino Acid Change
RA	W	Squamous	None	-	-
HL	B	Squamous	None	-	-
BC	W	Squamous	None	-	-
JB	B	Squamous	T to G	270	Phe to Cys
VW	B	Squamous	C to T	241	Ser to Phe
EH	B	Squamous	C to G	282	Arg to Cys
			A to G	292	Lys to Arg
HD	W	Squamous	G to A	181	Arg to His
AA	W	Squamous	G to A	175	Arg to His
			T to G	270	Phe to Cys

Overall five of eight squamous cell tumors had at least one missense mutation (63%) with all five being found in freshly frozen specimens. (Cases RA and HL were prepared from cultured samples.) Mutation at a CpG di-nucleotide or a transition from G to A occurred at four of the six different mutations. These are the expected sites for mutations caused by deaminating and alkylating agents. Such mutagens are present in significant quantities in tobacco. In conclusion, squamous cell carcinomas of the esophagus in patients from the South Carolina Lowcountry have a high frequency of p53 mutations, and it may be speculated that the mutational spectrum is consistent with their common exposure to tobacco use.

*By Invitation

F16. Molecular Markers of Poor Prognosis in Adenocarcinoma of the Lung Define Unique Groups of Patients

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MARK B. ORRINGER, M.D. and

*DAVID G. BEER, Ph.D.**

Ann Arbor, Michigan

The development of human adenocarcinoma of the lung involves multiple genetic changes including activation of oncogenes and loss of tumor suppressor genes. Patients whose lung tumors contain either mutation or nuclear protein accumulation of the tumor suppressor gene p53, K-*ras* oncogene mutation or erbB-2/neu protein overexpression have been shown to have a worse prognosis. We examined these three genetic indicators in 29 lung adenocarcinomas to determine if these markers are present in the same tumors or if they represent molecular changes which define different subsets of patients. P53 nuclear protein accumulation and erbB-2/neu protein overexpression were determined using immunohistochemical analysis of cryostat sections of tumor specimens and the corresponding normal lung tissue. ErbB-2/neu gene amplification was examined by Southern blot analysis. K-*ras* mutations were detected by radiolabeled oligo probes, specific for the various 12th codon mutations, hybridized to tumor DNA amplified by polymerase chain reaction. Increased nuclear accumulation of p53 protein was found in 10 adenocarcinomas (34%). All of the p53 positive tumors were found to show high level staining and homogenous expression of erbB-2/neu protein. K-*ras* mutations were detected in 6 tumors (21%), all of which overexpressed erbB-2/neu. The presence of a K-*ras* mutation did not correlate with p53 expression. In total, 86% of the tumors were found to over-express erbB-2/neu with the highest in tumors with erbB2/neu gene amplification. In conclusion, erbB-2/neu overexpression is a common event in lung adenocarcinomas. Furthermore, the presence of K-*ras* mutation and p53 protein accumulation define separate groups of patients. The mechanisms by which these genetic alterations adversely affect prognosis or interact are unknown.

*By Invitation

F17. Carcinogenic Specificity of P53 Tumor Suppressor Gene Mutations in Lung Cancer

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*MANUELA FODINGER, M.D. *,*

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and PROF. ERNST WOLNER

Vienna, Austria

Mutations in the p53 tumor suppressor gene, whose encoded protein is one of the chief regulators of the cell cycle, are proving to be the most common genetic alteration in human cancers. Point mutations have been detected in numerous types of human solid tumors.

Interestingly, the mutations are clustered in four regions of the gene which coincide with regions highly conserved through evolution. However, in lung cancer there is reason to believe that an additional hot spot region for mutations exists.

To investigate the role of p53 mutations in the development of lung cancer we have so far examined tumor specimens from 28 patients with different types of lung cancer (13 squamous cell, 6 large cell and 9 adenoid carcinomas). We extracted total RNA from tumor specimens and from corresponding normal material and peripheral blood as a control from each patient. Total RNA was reverse transcribed into cDNA, and cDNA sequences corresponding to the 11 exons of the p53 gene were amplified using polymerase chain reaction (PCR). Using single strand conformation polymorphism analysis we could detect the exact exon which contained the mutation and subsequently sequenced this exon.

17/28 patients showed p53 point mutations in tumor tissue. No mutations could be found in the corresponding normal tissue or peripheral blood, indicating that the mutations we found are somatic, acquired events. 50% of mutations were located in the additional hot spot region mentioned above. This region is not affected by mutations in other tumors. In 9 of 17 point mutations, the nucleotide guanine was replaced by thymine (G-T transversion). This mutation has occasionally been described for hepatocarcinomas but never for colon carcinomas although the overall incidence for p53 mutations is about the same.

Lung cancer is one of the most common cancers characterized by a well defined risk factor as there is exposure to carcinogen. Environmental carcinogens like benzpyrene have been shown to cause G-T transversions in vitro. In our patients we found that age and a history of smoking were associated with the occurrence of G-T transversions. Our results indicate that the nature and position of p53 mutations are influenced by tissue type which may be due to differences in exposure to mutagens of these cells.

*By Invitation

F18. Pain Management for Thoracotomy: Thoracic Epidural Versus Paravertebral Block

*GILBERT J. GRANT, M.D. *, KRISTIEN VERMEULEN, M.D. *,*

*HERMAN TURNDORF, M.D. **

and ARTHUR D. BOYD, M.D.

New York, New York

Background/Objective: After thoracotomy, patients experience intense pain and a significant decrease in pulmonary function. Continuous thoracic epidural (EPI) analgesia has been considered the optimal method for post thoracotomy pain control, but recently continuous extrapleural paravertebral (PARA) nerve block for post thoracotomy analgesia has been described. This study was designed to compare the effectiveness of these two methods for post thoracotomy analgesia.

Methods: Twenty-five patients undergoing pulmonary resection were randomized to 2 groups (EPI = 12, PARA= 13). EPI catheters were inserted at T5-6 or T6-7 levels before surgery. PARA catheters were placed during surgery into a paravertebral parietal pleural pocket extending two interspaces above and below the incised interspace. Prior to rib approximation, the EPI group received a 0.2 mg/kg bolus of 0.125% bupivacaine + fentanyl 2 µg/ml; the PARA group received a 0.3 ml/kg bolus of 0.25% bupivacaine. An infusion of bupivacaine 0.125% + fentanyl 2 µg/ml was then started in both groups at 0.1 ml/kg/hr. Pain was managed by giving a 0.15 ml/kg bolus and increasing the infusion in 0.01 ml/kg/hr increments as needed. Pain scores and blood gases

were obtained. FEV₁, VC, and FVC were assessed preoperatively and at 6 and 24 hours postoperatively.

Results: Age, weight, sex and surgery were similar in both groups. PARA patients required more bupivacaine (342 ± 56 mg) and fentanyl (632 ± 90 µg) during the first postoperative day than EPI patients (bupiv 237 ± 61 ; fent 379 ± 98 µg). Pain was well controlled in both groups, with no significant differences in pain scores or blood gases. Arterial pressure was lower in EPI than PARA patients between 14-16 hours postoperatively. FEV₁, VC, and FVC were all significantly lower than preoperative values, but there were no intergroup differences (expressed below as % pre-op):

	FEV ₁ 6h	FEV ₁ , 24h	VC 6h	VC 24h	FVC 6h	FVC 24h
PARA		60 ± 19	60 ± 16	52 ± 13	56 ± 17	56 ± 15
EPI		57 ± 17	57 ± 18	50 ± 14	53 ± 15	56 ± 15

Conclusions: Continuous PARA block is as effective as continuous EPI block. Thus, PARA block should be considered as an excellent alternative to EPI block for post thoracotomy pain relief.

*By Invitation

WEDNESDAY MORNING, April 28, 1993

9:00 a.m. SIMULTANEOUS SCIENTIFIC SESSION D CARDIAC SURGERY

Grand Ballroom B

Moderators: Delos M. Cosgrove, M.D.

Robert A. Guyton, M.D.

38. Long Term Results of Mitral Valve Reconstruction for Regurgitation of the Myxomatous ("Floppy") Mitral Valve

*LAWRENCE H. COHN, M.D., GREGORY S. COUPER, M.D. *,*

*ROBERT J. RIZZO, M.D. *, SARYF. ARANKI, M.D. *,*

*NANCY M. KINCHLA, B.S. **

and JOHN J. COLLINS, JR., M.D.

Boston, Massachusetts

The myxomatous, prolapsed or "floppy" mitral valve is the most common etiology of mitral regurgitation (MR) in North America. Mitral valve reconstruction for MR was carried out on 201 consecutive patients with a myxomatous mitral valve, from 1984 - 1992. There were 127M/74F, aged 23-84 years, 63 years; 35% of patients ≤ 70 years, 78% were in Functional Class III or IV, and 30% had coronary artery disease requiring coronary bypass.

The posterior leaflet was resected in 146 patients (73%), the anterior leaflet in 14, anterior and posterior leaflet in 12, chordoplasty plus annuloplasty ring in 10 patients, and a commissuroplasty plus annuloplasty ring in 19 patients. A flexible Duran ring was used in 95 patients (47%), Carpentier ring in 45 patients (22%), and no ring was used in 61 patients (30%). There were 5 operative deaths (2.5%); 4/5 occurred in patients ≤ 70 (5.7%); only 1 death occurred in the 131 patients < 70 years of age (0.8%).

In the late postoperative period (mean follow-up 3 years), 90% of patients are asymptomatic, 2 developed endocarditis (IVD), 6 patients had throm-boemboli, (4 transient, 2 permanent). Structural valve degeneration (SVD) requiring reoperation occurred late in 12 patients; 8 were in

posterior leaflet resection, 2 in anterior or anterior and posterior; 6/12 had no annuloplasty ring. There was a zero incidence of systolic anterior motion of the mitral valve noted on postoperative echo prior to discharge. Actuarial analysis at 5 years is indicated below:

5 Years

Overall Survival	86 ± 5 %	
Freedom from IVD	98 ± 2%	
Freedom from TE	93 ± 2%	
Freedom from SVD		
Overall	83 ± 4%	
Flexible Ring	91 ± 5%	
Rigid Ring	85 ± 6%	p = .05
No Ring	68 ± 12%	
Freedom from Reoperation	84 ± 4%	

Mitral valve reconstruction for complicated myxomatous disease of the mitral valve, regardless of leaflet involvement, is feasible and offers excellent early and late results.

*By Invitation

39. The Use of Homograft Valves for Reoperations on the Aortic Valve

*MARIO ALBERTUCCI, M.D. *, KIT WONG, FRCS*,*

*STERGIOS THEODOROPOULOS, M.D. **

*MARIO PETROV, M.D. * and MAGDI YACOUB, FRCS*

London, England

Homograft Valves offer several theoretical advantages when used for patients who have had previous operations on the Aortic Valve. Between 1972 and 1992, 110 patients received Aortic Homografts after previous Aortic Valve operation, 84 patients had previous Aortic Valve Replacement (Homograft in 69, Prosthetic Valves in 11 and Stented Xenografts in 4) while 26 had previous Valve Repair. The indication for reoperation was Valvular Deterioration or Malfunction in 82 (75%) and Endocarditis in 28 patients. The valves were obtained under sterile conditions in 20 and sterilized in Antibiotics in 90.

The Homograft was used to replace the Aortic Valve and root in 32 patients and inserted in the subcoronary position in the remaining 78 patients. The hospital mortality was 5% and was not influenced by the type or number of previous operations or the presence or absence of Aortic Endocarditis ($P>0.1$). Homografts were particularly useful in patients with active Endocarditis on the native or Prosthetic Valve who had multiple root abscesses. None of the patients developed recurrence of infection within the first 6 months after the operation. The actuarial survival free of Valve related complications was 78% at 10 years.

It is concluded that reoperations using Unstented Aortic Homografts give good early and medium term results and offer considerable advantages for patients with Endocarditis on previously replaced Valves.

*By Invitation

40. Reoperation After Mitral Valve Reconstruction: Analysis of 33 Cases

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*MARTIN LeBOUTILLIER, M.D. *,*

*F. GREGORY BAUMANN, Ph.D. *,*

FRANK C. SPENCER, M.D. and

*STEPHEN B. COLVIN, M.D. **

New York, New York

From 1/80 through 6/92, 664 patients (pts) underwent mitral valve repair using Carpentier reconstructive techniques. Of those pts, 33 (5.0%) have subsequently required reoperations from one day to 8.3 yrs postoperatively (mean interval = 2.3 yrs; mean age = 46 yrs, range 13-82 yrs). The etiology of the original mitral disease was rheumatic in 11 pts (33%), degenerative in 8 pts (24%), ischemic in 7 pts (21%), congenital in 6 pts (18%) and endocarditis in 1 pt (3%). Reoperative mortality was 6.1%.

Early reoperation (<6 months, n = 10) was attributed to technical problems in 5 pts (50%) (ring dehiscence in 3 pts, non-quadrangular leaflet resection in 1 pt, and inappropriate repair of a congenital valve in 1 pt), endocarditis in 2 pts (20%), ischemia with ruptured chordae in 2 pts (20%), and rheumatic disease in 1 pt (10%). Technical or judgement errors accounted for 6 of 10 (60%) early failures. Re-repair was accomplished successfully in 2 pts (20%).

Late operation (>6 months, n = 23) was due to rheumatic disease in 9 pts (39%), progression of non-rheumatic disease (chordal rupture and leaflet retraction) in 5 pts (22%), endocarditis in 6 pts (26%) and technical problems (ring dehiscence or ring malorientation) in 3 pts (13%).

Potentially preventable errors in judgment and technique are responsible for 60% of the early and 13% of the late failures after mitral valve reconstruction. Rheumatic disease remains the primary cause of late reoperation.

*By Invitation

41. Determinants of Reoperation After 963 Valve Replacements With Carpentier-Edwards Prostheses

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*ANGELA C. HATTON, B.A. *,*

W. GLENN YOUNG, M.D., WALTER G. WOLFE, M.D.

and JAMES E. LOWE, M.D.

Durham, North Carolina

During the period of 1977-1990, 963 Carpentier-Edwards Standard valve prostheses were placed in 879 operations (368 aortic, 374 mitral, 59 tricuspid, 1 pulmonic, 58 aortic and mitral, 1 aortic and tricuspid, 11 mitral and tricuspid, 7 aortic and mitral and tricuspid). Over 4795 patient-years of follow-up, 206 Carpentier-Edwards valves required reoperation with freedom from

reoperation at 8/10/12 years being $72 \pm 3/54 \pm 4/39 \pm 5\%$ for mitral valve replacement and $85 \pm 3/71 \pm 4/61 \pm 5\%$ for aortic valve replacement. By Cox univariate proportional hazards model, preoperative comorbidity, gender, ejection fraction $<40\%$, left main or 3 vessel coronary disease, concurrent coronary bypass, and concurrent valve operation did not affect the likelihood of reoperation. The only independent determinant of reoperation for aortic and mitral valves was age. Likelihood of reoperation decreased with age ($p < 0.05$) with freedom from reoperation after 10 years in patients aged <60 yrs vs ≥ 60 yr being 60 ± 5 vs $84 \pm 5\%$ after aortic valve replacement and 45 ± 5 vs $72 \pm 6\%$ after mitral valve replacement. For mitral valve replacement, larger valve size made reoperation more likely ($p = 0.01$, 80/101 for prosthetic dysfunction) with freedom from reoperation at 10 years being $63 \pm 6\%$ for sizes <31 mm and $47 \pm 6\%$ for sizes ≥ 31 mm. For aortic valve replacement, any prior valve procedure increased the likelihood of reoperation ($p < 0.01$), with freedom from reoperation at 10 years falling from $75 \pm 4\%$ to $32 \pm 12\%$ when any prior valve procedure was present.

Thus, the low incidence of reoperation affirms the suitability of the Carpentier-Edwards prosthesis for selected patients over the age of 60 years. Reoperation is more likely for large mitral sizes primarily due to prosthetic dysfunction and for aortic valve replacement after a prior valve procedure. Comorbidity and concurrent valvular operation have relatively little effect on the likelihood of reoperation and should have less influence on the choice of valvular prosthesis.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

11:05 a.m. SIMULTANEOUS SCIENTIFIC SESSION D CARDIAC SURGERY

Grand Ballroom B

42. Early and Late Phase Events Following Valve Replacement With the St. Jude Medical Prosthesis in 1,200 Patients

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*MARK S. ADKINS, M.D. *, WILLIAM A. ANDERSON, M.D. **

and CHAO CHEN, Ph.D, BRIDGET M. BAILEY, B.S.N. *,*

*LINDA M. NEALON, B.S.N. * and*

LYNN B. MCGRATH, M.D.

Browns Mills, New Jersey

The object of this investigation was to determine early and late phase events following valve replacement with the St. Jude Medical (SJM) valve. From May, 1982 to August, 1991, 1200 patients (pts) underwent valve replacement with the SJM valve. There were 615 males (51%) and 585 females. Ages ranged from 2 to 89 yrs, mean 58 yrs. Preoperatively, 830 pts (69%) were in functional class III and IV. Six-hundred eleven pts (51%) had aortic valve replacement (AYR), 490 (41%) had mitral valve replacement (MVR), 2 (0.2%) had tricuspid valve replacement (TVR), and 97 (8%) had multiple valves replaced. There were 81 hospital deaths (6.8%). Risk factors for hospital death included older age ($p = 0.0001$), higher preoperative left ventricular end diastolic pressure ($p = 0.05$), longer aortic cross-clamp (AXC) time ($p = 0.0001$), longer cardiopulmonary bypass (CPB) time ($p = 0.0001$), female gender ($p = 0.02$) and previous cardiac surgery ($p = 0.0005$). Follow-up was 98% complete (3153 pt-yrs) at a mean of 2.9 yrs, range 0.1-9 yrs. There were 149 late deaths; 25 (17%) were considered valve-related: 8 prosthetic valve endocarditis

(PVE), 4 valve thrombosis (VT), 6 thromboembolism (TE), 5 anticoagulant related hemorrhage (ACRH), 1 paravalvular leak (PVL), 1 hemolytic anemia. Overall actuarial survival was 74% (CL: 70%) at 5 yrs postoperatively; actuarial survival for AYR vs. MVR vs. multiple valve replacement did not differ significantly ($p = 0.06$). Risk factors for late death included older age ($p = 0.03$), lower preoperative ejection fraction ($p = 0.005$), longer AXC ($p = 0.0001$), longer CPB ($p = 0.0001$), higher preoperative functional class ($p = 0.0001$) and previous cardiac surgery ($p = 0.0003$). Late valve-related events included: TE (2.1% pt-yr); ACRH (1.0% pt-yr), PVE (0.5% pt-yr), VT (0.2% pt-yr), PVL (0.9% pt-yr). Actuarial freedom at 5 yrs from TE was 92%; ACRH (94%), PVE (98%), reoperation (98%), PVL (96%), VT (99%). Actuarial freedom from all valve-related events was 75% and actuarial freedom from valve-related deaths was 95% at 5 years. At follow-up, 97% of the survivors were in functional class I and II. We conclude that the low incidence of valve-related events and low mortality supports the continued use of the SJM valve.

*By Invitation

43. Outcome of Mitral Valve Repair in Patients With Preoperative Atrial Fibrillation: Should the Maze Procedure be Combined With Mitral Valvuloplasty?

YEOW L. CHUA, M.D.; HARTZELL V. SCHAFF, M.D.,*

THOMAS A. ORSZULAK, M.D. and

*JAMES J. MORRIS, M.D.**

Singapore and Rochester, Minnesota

The low risk of mitral valvuloplasty and encouraging preliminary results of operation to prevent atrial re-entry tachycardia have stimulated interest in combining the procedures for patients (pt) with mitral valve regurgitation (MR). Little is known, however, about the extent to which atrial fibrillation (AF) compromises early and late results of valve repair. Therefore, we reviewed 323 consecutive pts who underwent mitral valve (MV) repair for MR from 1980-91; average age of the 215 men and 108 women was 64 yr (range 14-88 yr), and 224 pt (70%) were in NYHA class III or IV preop. The main indications for operation were severe MR (76%), coronary artery disease (15%), and aortic valve disease (6%). At the time of MV repair, coronary artery bypass grafting was performed in 35% of pt, aortic valve replacement was performed in 7%, and multiple other procedures were performed in 10%. For all pt, 30-day mortality was 2.5% (70% C.L. 1.6%-3.4%), and survivorships at 3 and 5 yr were 81% and 76% respectively. Preoperatively, 215 pt were in sinus rhythm (SR), and 97 pt had AF; in the latter group, 11 pt had onset of AF within 3 mo. preceding MV repair. Comparing pt with preop AF to those with SR, we found no significant difference in operative mortality (3% vs 2%) or 5-yr survival (75% vs 77%). At late follow-up, AF was present in 5% of pt with preop SR, 80% of pt with preop chronic AF, and 0% of pt with preop recent onset AF ($p < 0.01$). The left atrial (LA) size by echocardiography was larger in pt with preop AF compared to those with SR ($59 \pm 1.4 \text{ cm}^2$ vs $50.9 \pm 0.7 \text{ cm}^2$, $p < 0.05$). There was, however, no correlation between preop LA size and late AF. Further, age, gender, and associated coronary artery disease did not correlate with presence of AF at late follow-up. Risk of stroke during follow-up was similar in pt with preop SR compared to those with AF.

In conclusion, MV repair should be performed before or soon after the onset of AF in order to maximize the chance of postop SR and avoid long-term anticoagulation with Coumadin. However, the decision for concomitant procedures to control AF should be made with the realization that early and intermediate-term outcome of MV repair in such pt is good, and concomitant operation for supraventricular arrhythmia must have negligible morbidity and no adverse effect on operative mortality.

*By Invitation

44. Glutamate Excitotoxicity - A Mechanism of Neurologic Injury Associated With Hypothermic Circulatory Arrest

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WILLIAM A. BAUMGARTNER, M.D.

Baltimore, Maryland

Glutamate, the major CNS neurotransmitter, may have potent neurotoxic activity under conditions of metabolic stress. By receptor autoradiography (RA), we have demonstrated that brain regions most vulnerable to injury during prolonged HCA have the highest density of glutamate receptors. To test the hypothesis that such injury could be mediated by GE, we used MK-801, a selective NMDA-glutamate receptor antagonist in a canine model of HCA. Twelve male dogs (20-25kg) were placed on closed-chest car-diopulmonary bypass (CPB), subjected to 2 hours of HCA at 18 °C, and rewarmed to 36°-37°C on CPB. All were mechanically ventilated and monitored for 20 hours before extubation and survived for 3 days. Grp 1 dogs (n = 6) received a pre-HCA IV bolus of MK-801 (0.75 mg/kg) followed by continuous infusion (75ug/kg/hr), resulting in EEC silence. MK-801 was weaned before extubation. Grp 2 dogs (n = 6) received vehicle only. Using a species-specific behavior scale which yielded a neurodeficit score (NDS) ranging from 0 (normal) to 500 (brain dead), all animals were neurologically assessed every 12 hours. Following sacrifice at 72 hrs, brains were examined by RA and histologically for patterns of selective neuronal necrosis and scored blindly from 0 (normal) to 100 (severe injury). Grp 1 dogs had better neurologic function compared to Grp 2, (NDS 21 ± 15 Vs 192 ± 40, p<.001) and had less neuronal injury (7.3 ± 3 Vs 48.3 ± 9, p<.0001). Densitometric RA revealed selective preservation of neuronal NMDA-glutamate receptor expression in Grp 1 only. These results represent the first direct evidence of a role for GE in the development of HCA-induced brain injury and suggest that selective glutamate receptor antagonists may have a neuroprotective role in prolonged periods of HCA.

12:10 p.m. ADJOURN

*By Invitation

WEDNESDAY MORNING, April 28, 1993

9:00 a.m. SIMULTANEOUS SCIENTIFIC SESSION E GENERAL THORACIC SURGERY

Grand Ballroom D

Moderators: Joseph L. Miller, M.D.

Valerie W. Rusch, M.D.

45. Thymomas: Prognostic Factors and Long Term Results of 349 Operated Cases

*PHILIPPE LEVASSEUR, M.D. *,*
*JEAN-FRANCOIS REGNARD, M.D. *,*
*PIERRE MAGDELEINA T, M.D. *,*
*PHILIPPE DARTEVELLE, M.D. *,*
*CHRISTIAN DROMER, M.D. * and*
*JEAN-FRANCOIS LEVI, M.D. **

Plessis Robinson, France

Sponsored by: Willard A. Fry, Evanston, Illinois

Three hundred forty-nine patients (mean age 49) with thymoma were surgically treated over 26 years. Myasthenia gravis was associated in 209 (60%). MASAOKA staging revealed stage I in 154 (45%), stage II in 76 (22%), stage III in 95 (27%), stage IV in 24 (6%). Among the 325 non metastatic tumors, 277 (85%) were totally resected (all stage I and II, 50% of stage III) and 48 (15% had an incomplete resection or biopsy (50% of stage III). Histological findings* were: "spindle cell" tumors 80 (23%), lymphocytic rich tumors 86 (25%), epithelial tumors 183 (52%). Postoperative mortality was 2.3% and was not influenced by myasthenia gravis. Postoperative radiotherapy was performed in 15% of stage I and 75% of stage II and totally resected III, and in 85% of non totally resected III. 90% of stage IV were treated by postoperative radiotherapy and/or chemotherapy. Follow-up was on average 7.5 years and exceeded 10 years in 1/3 of the cases. 8 patients were lost to follow-up. 117 patients died (causes: tumor: 1/3, auto-immune 1/3, miscellaneous 1/3). 21 patients (7.5%) (6 stage I (4%), 8 II (10%) 7 III (15%)) had a recurrence of their thymoma after total excision, and this occurred on average after 6 years (ranged from 2 to 16 years). In calculating by the actuarial method, recurrences were significantly higher in stage III vs II ($p<0.001$) and in stage II vs I ($p<0.01$). Overall actuarial survival at 10 years was 65%. Actuarial survival in stage I, II, III and IV was 80%, 71%, 43%, and 30% respectively. No significant difference was seen between stage I and II, or between III and IV. The only significant difference observed was between stage II and III ($p<0.001$). Regarding the quality of thymoma excision, the 10 year actuarial survival for the group with total excision was 76% vs 28% ($p<0.001$) in the non totally resected group. Actuarial survival was significantly better in totally resected stage III than in non totally resected III ($p<0.001$). Similarly, there was no significant difference between stage II and totally resected III, or between non totally resected III and stage IV. Myasthenia gravis and postoperative radiotherapy did not influence the prognosis in univariate analysis. Additionally, the epithelial tumors have a significantly worse prognosis but the multivariate analysis did not confirm this fact, since histology and MASAOKA staging were closely linked.

According to our results, we propose to modify the MASAOKA classification in order to take into account the quality of thymoma excision.

*By Invitation

46. Video-Thoracoscopic Resection Using the Nd:YAG Laser

*ROBERT J. KEENAN, M.D. *,*
RODNEY J. LANDRENEAU, M.D.,
STEPHEN R. HAZELRIGG, M.D.
and PETER F. FERSON, M.D.

Pittsburgh, Pennsylvania and Milwaukee, Wisconsin

Sponsored by: Hartley P. Griffith, M.D., Pittsburgh,

Pennsylvania

Since January 1991, we have performed 63 thoracoscopic resections, using the Nd:YAG laser, for pulmonary nodular or interstitial disease. Indications included 46 patients with malignancy (27 primary lung; 19 metastatic), 9 with benign nodules, 6 with nodular interstitial processes and 2 with granulomatous disease. There were 30 males and 33 females with a mean age of 62.6 ± 12.5 years. Thirty patients underwent thoracoscopic resection using the Nd:YAG laser alone while 33 had lesions resected with a combination of laser and endoscopic stapling. Ten of 27 patients diagnosed with primary lung malignancies subsequently underwent open anatomic resections. Pulmonary reserve of the other 17 patients was deemed inadequate for further resection. Operating time, number of chest tube days, length of hospital stay and complication rate were compared with 72 patients undergoing thoracoscopic resection of nodules using endoscopic stapling alone.

	Laser	Laser + Stapler	Stapler	
Or Time (min)	159.4 \pm 70.8	160.4 \pm 83.6	83.7 \pm 51.3*	(p<0.05)
Chest Tube Days	3.4 \pm 2.9	4.6 \pm 5.3	3.0 \pm 1.9	(P = NS)
Hospital Stay (days)	5.3 \pm 2.7	6.8 \pm 5.2	5.4 \pm 2.8	(P = NS)
Complications	9/30	9/33	11/72	(P = NS)

Lesions ranged in size from 0.4 to 3.5 cm. Laser resection was performed for lesions deep in the substance of the lung parenchyma or on the effaced surface of the lung; both locations which make stapling alone difficult or impossible. Non-contact laser was used in preference to electrocautery because of the precision of dissection allowing easy identification of vessels which could be clipped and divided safely. Although operating time for laser-assisted procedures was longer (*p<0.05), there were no differences in number of days with chest tubes in place or hospital stay when compared to stapled resections. The complication rate for laser cases was not significantly higher than for stapled resections and consisted primarily of air leaks lasting 2-7 days. The recent use of biologic glue over the raw surface has largely eliminated this problem. There were no bleeding complications or postoperative deaths. Thoracoscopic laser resection is particularly suited for deep lesions and surface nodules where staplers are inadequate. This analysis shows that the Nd:YAG laser is a safe and precise, primary or adjunctive tool for thoracoscopic pulmonary resection.

*By Invitation

47. Thoracoscopic Treatment of Bullous Emphysema Using Sapphire Contact Tip Neodymium Yttrium Aluminum Garnet Laser (Contact YAG): Preliminary Report

*AKIO WAKABA YASHI, M.D., HAROLD PETERS, M.D. *,*

*NARINDAR SINGH, M.D. *, GARY BENNETT, M.D. *,*

*GREGORY BARNES, M.D. *, RICHARD FUJITA, M.D. **

*and JANE CALMESE, R.N. **

Orange, California

We initiated the thoroscopic treatment of bullous emphysema using carbon dioxide (CO₂) laser three and a half years ago with an excellent result. However, it was found ineffective for certain types of bullae and its articulated arm was difficult to maneuver. Therefore, the current study was undertaken to investigate the efficacy of sapphire contact Nd:YAG laser (Contact YAG). From September 21, 1992 to September 18, 1992, 128 patients (95 males & 33 females) with severe bullous emphysema with chronic obstructive pulmonary disease were treated by Contact YAG. Patient selection was based on symptoms (dyspnea), forced expiratory volume for one second (FEV₁), and chest computed tomography (CT). No patients were denied because of the severity of illness. Age ranged from 39 to 79, mean being 65.71 ± 7.38 . Seventy-eight patients were prednisone dependent, 55 oxygen dependent, 42 wheelchair- or bed-bound, 3 had antitrypsin deficiency and 2 had infected bullae. FEV₁ varied from 8 to 72% of predicted values, the mean being 22.69 ± 11.58 . CT showed giant compressive bullae (type I) in 3, diffuse bullae (type III) in 98 and a combination (type IV) in 27 cases. Type III bullae were contracted by contact YAG laser round probe and Type I and IV bullae were excised by contact YAG laser scalpel. In addition, the bronchial communications were closed by sutures and redundant bullae plicated. The anesthesia time ranged from 1.5 to 10.1 hours, the mean being 4.84 ± 1.54 . The ventilatory support ranged from 3 to 504 hours, the mean being 23.66 ± 56.88 hours. The air leak persisted for 3 to 64 days, the mean being 18.59 ± 13.64 days. Seven patients required repeated thoracoscopy for closure of bronchopleural fistulae. Five patients died, thus the overall mortality rate was 4%. Clinical improvement was noted in all but two; 21 (38%) patients were off oxygen and 26 (63%) could walk without wheelchair. In conclusion, thoroscopic ablation of bullae using a sapphire contact tip Nd:YAG laser is an effective treatment with a reasonable risk for bullous emphysema.

*By Invitation

48. Thoroscopic Wedge Excisions for Indeterminate Pulmonary Nodules

*MARK S. ALLEN, M.D. *, CLA UDE DESCHAMPS, M.D. *,*

*ROBERTA E. LEE, M.D. *, VICTOR F. TRASTEK, M.D.*

and PETER C. PAIROLERO, M.D.

Rochester, Minnesota

From June 1991 to July 1992, 119 patients (53 males, 66 females) underwent 121 video thoroscopic surgical (VTS) procedures for indeterminate pulmonary nodules (IPN). Median age was 64 years (range 20-85). Thoracotomy was performed in 29 patients (24%) after thoracoscopy only because the nodule could not be located in 15 patients, appeared malignant in 5, or for other technical reasons in 9. Incisional biopsy revealing metastatic carcinoma made wedge excision unnecessary in 4 patients. Thoracotomy was also required following an unsuccessful attempt at VTS wedge resection in 3 patients. Eighty-five patients underwent 96 wedge resections using VTS. Twenty-one (25%) of these 85 patients were opened, 13 to perform formal lung resection following a diagnosis of bronchogenic carcinoma, 4 for an initial nondiagnostic pathology report, 2 to locate a second nodule, and 1 each for a positive metastatic cancer margin, and stapler malfunction. The pathology of the remaining wedge excisions was granuloma in 29, metastatic cancer in 25, other benign lesions in 9, hamartoma in 8, and lymphoma in 3. In those undergoing VTS alone, there was no mortality, and there were 4 (6.2%) complications. Postoperative analgesic requirements were less in the VTS only group when compared to the open group. The average postoperative hospital stay in the VTS only group was 4 days.

We conclude that VTS wedge resection is a safe and effective procedure in carefully selected patients with IPN. A significant number (42%) required an open procedure to ensure an adequate resection of malignancy or to accomplish a safe operation.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

**11:05 a.m. SIMULTANEOUS SCIENTIFIC SESSION E GENERAL THORACIC
SURGERY Grand Ballroom D**

49. Short Segment Intestinal Interposition of the Esophagus

*DOUGLAS J. MATHISEN, M.D. *,*

*HENNING A. GAISSERT, M.D. *, HERMES C. GRILLO, M.D.,*

*JOHN C. WAIN, M.D. *, ASHBY C. MONCURE, M.D.,*

*RONALD A. MALT, M.D. * and*

*LESLIE W. OTTINGER, M.D. **

Boston, Massachusetts

Esophageal replacement remains a challenge. For some conditions the stomach is not a suitable substitute. Short segment colon and jejunal interposition are alternative conduits, however, limited information is available on the results of their use. Between 1971 and 1991, 41 patients underwent short segment interposition of the esophagus with jejunum or colon. Indications were failed antireflux procedures (21 patients), nondilatable stricture (11), achalasia (2), Barrett's esophagus (2), hemorrhagic esophagitis after esophagogastrectomy (1), motility disorder (1), instrumental perforation (1), carcinoma (1), and leiomyosarcoma (1). Thirty-one patients (75.6 percent) had prior surgical procedures. Eleven males and 11 females (mean age 56.9 years) had colon grafts, 12 males and 7 females (mean age 65.0 years) underwent jejunal interposition. Median hospital stay was 17 days for colon and 21 days for jejunum. Major complications after colon interposition consisted of pneumonia (3 patients), sepsis (1), ARDS (1), graft perforation (1), sub-phrenic abscess (1), chylothorax (1), pulmonary edema (1), pulmonary embolus (1), and DVT (1), and in-hospital mortality was 4.5 percent (sepsis-1). Major complications following jejunal interposition included pneumonia (3 patients), graft necrosis (1), MI (1), iatrogenic gastric perforation (1), and in-hospital mortality was 10.5 percent (graft necrosis-1, MI-1). One anastomotic leak was contained. Two colon and one jejunal grafts required late revision. One or more dilatations were performed after 4 jejunal and 5 colon interpositions. Mean follow-up for 36 patients is 77.9 months. Twenty-two patients responded to a questionnaire. All tolerate a regular diet. Dysphagia is mild (8) or absent (14). Regurgitation is described as mild in 11 and severe in 4 patients. Six patients underwent manometry and barium food study. Two colon segments were aperistaltic by manometry and emptied by gravity. Three jejunal interpositions were hypoperistaltic by manometry with slow emptying of contrast and 1 was aperistaltic with a distended afferent loop on fluoroscopy. When stomach is not available, successful palliation of swallowing can be accomplished with either jejunum or colon. Surgeons involved in the management of esophageal disease should be familiar with the demanding technical details of both procedures.

*By Invitation

50. Extended Esophagectomy in the Management of Esophageal Carcinoma of the Upper Thoracic Esophagus

*WICKII T. VIGNESWARAN, M.D. **

*VICTOR F. TRASTEK, M.D., MARK S. ALLEN, M.D. *,*

*CLAUDE DESCHAMPS, M.D. * and*

PETER C. PAIROLERO, M.D.

Rochester, Minnesota

Resection of cancers of the upper thoracic esophagus often require a preliminary thoracotomy to accomplish resection. Between January 1985 and July 1992, 50 consecutive patients underwent extended esophagectomy for cancer of the upper thoracic esophagus where the neoplasm was resected through a concomitant laparotomy, right thoracotomy, and cervical incision. There were 39 males and 11 females. Ages ranged from 40 to 80 years (mean 63 +/- 9.5 years). Thirty-three patients (66%) had squamous cell carcinoma, and 17 (33%) had adenocarcinoma arising in Barrett's esophagus. Complications occurred in 31 patients and included pneumonia in 19 patients; anastomotic leak in 17, vocal cord paralysis in 13, atrial ar-rhythmia in 10, wound infection in 5, renal failure in one, and postoperative bleeding in one. Four patients required tracheostomy. There was one perioperative death (2%). Median survival was 1.6 years. Twenty-five patients are currently alive, 22 without evidence of cancer. Cause of death was recurrent disease in 20 patients. The overall two- and four-year actuarial survival was 48% and 31%, respectively. Survival data by stage will be presented. Nine patients developed late dysphagia; 4, gastroesophageal reflux; and 3, symptoms of dumping. Although associated with significant morbidity, we conclude that extended esophagectomy is the procedure of choice for neoplasms of the upper thoracic esophagus. Mortality is low and long-term results are excellent.

*By Invitation

51. Superiority of Extended En Block Esophagogastrectomy for Carcinoma of the Lower Esophagus and Cardia

*TOM R. DeMEESTER, M.D., JEFF A. HAGEN, M.D. **

*and JEFF H. PETERS, M.D. **

Los Angeles, California

To investigate the relationship between the type of resection performed and survival in patients with esophageal cancer, we reviewed our experience with esophagogastrectomy for carcinoma arising in the distal esophagus and gastric cardia. Between July 1983 and January 1992, 69 patients underwent resection of distal esophageal or gastric cardia cancers. Preoperative and in-traoperative staging defined 3 distinct subgroups of patients. Those with apparently limited disease and good general health (Group 1, N = 30) underwent en bloc resection. Those with apparently limited disease but poor physiologic reserve (Group 2, N = 16) underwent transhiatal resection, as did those with evidence of more advanced disease (Group 3, N = 23). The mean follow-up time of surviving patients was 26 months. Survival curves were calculated by the Kaplan Meier method and reported as the percent of patients surviving at the end of the observation period. Comparison between groups were made using the log-rank method.

Univariate analysis revealed no difference in survival based on tumor cell type, but that the depth of tumor invasion and the type of resection were predictive of long-term survival. Overall, survival was significantly better in the 30 patients having en bloc resection (41%), than in the 39 patients having transhiatal resections (14%) ($p < 0.001$, log-rank).

Clinical staging revealed apparently limited disease in 46 patients (Groups 1&2). These groups differed only in the presence of complicating medical problems in Group 2, as the percentages of patients with transmural tumors (Gp.1 17/30, Gp. 2 10/16) and extensive nodal disease (Gp.1 11/30, Gp. 2 2/16) were not significantly different. Survival following en bloc resection was, however, significantly better (41% vs 21%, $p<0.05$, log-rank).

Utilizing a modified WNM system of postoperative histologic staging, 19 patients had early lesions defined as intramural lesions associated with 4 or fewer nodes, 26 had intermediate lesions defined as either transmural or associated with >4 lymph nodes, and 24 had late lesions defined as both transmural and associated with >4 lymph nodes. Survival was significantly better for patients with early lesions following en bloc resection, compared to transhiatal resection (75% vs 20%, $p<0.01$), as well as in those with advanced lesions (27% vs 9%, $p<0.01$). For intermediate lesions, the survival was similar (14% vs 20%), although the median survival following en bloc resection was longer (24 mo. vs 8 mo.).

Conclusions: Survival following resection of carcinoma of the distal esophagus and gastric cardia is dependent upon the method of resection. Patients with early lesion had significantly better survival after an extended en bloc resection.

12:10 p.m. ADJOURN

*By Invitation

WEDNESDAY MORNING, April 28, 1993

9:00 a.m. SIMULTANEOUS SCIENTIFIC SESSION F CONGENITAL HEART DISEASE

Grand Ballroom F

Moderators: Hillel Laks, M.D.

John E. Mayer, M.D.

52. Transposition With Intact Ventricular Septum: Arterial Switch After 21 Days of Age

*TOM R. KARL, M.D. *, ANDREW M. DAVIS, FRACP*,*

JAMES L. WILKINSON, FRACP and*

ROGER B.B. MEE, M.B., Ch.B., FRACS

Victoria, Australia

Sponsored by: John W. Kirklin, M.D.,

Birmingham, Alabama

Although results of arterial switch for TGA.IVS have improved worldwide, controversy still exists regarding the upper age limit for one stage operation. This presentation reviews our experience with primary arterial switch for infants 21 days or older. As of December 1990, 118 arterial switch operations have been performed for TGA.IVS. 18/118 patients had operation after 21 days of age (mean = 38 days, range = 21-118 days). 14/18 had echocardiographic and/or cardiac catheter evidence of low left ventricular pressure. None had a preliminary PA band.

All patients had alpha blockade with phenoxybenzamine + / - other systemic and pulmonary vasodilators, as well as inotropic support for 3-5 days. Operative mortality was 1/18 (5.6%, CL = 0.8-17%) for the >21 days group, versus 0/100 (0%, CL = 0-1.8%) for the <21 days patients ($p<.33$). The single death was probably due to a technical problem with a coronary anastomosis. One patient in the >21 days age group required LVAD for 48 hours postoperatively. There have been no late deaths, and late hemo-dynamics are similar in the two groups. We conclude that although operation in the first two weeks of life remains preferable,

with appropriate peri-operative management one stage arterial switch for TGA.IVS can be safely performed up to age 4 weeks and possibly 8 weeks of age.

*By Invitation

53. The Surgeon's "Skill" As a Risk Factor in the Arterial Switch Operation

MARC R. deLEVAL, M.D., KATRIENFRANCOIS, M.D. *,

CATHERINE BULL, MRCP*.

EUGENE H. BLACKSTONE, M.D.

and JOHN W. KIRKLIN, M.D.

London England and Birmingham, Alabama

An experienced pediatric cardiac surgeon prepared himself to perform the arterial switch operation by a series of on-site tutorials and then between 6/16/87 and 9/15/92 performed 93 arterial switch operations for TGA (simple or with VSD). Initial euphoria with 1 death in the first 52 patients gave way to increasing concern when cases 53, 55, 59, 63, 64, 67, and 68 died. No deaths have occurred since 68. Was the surgeon a risk factor? Were the early favorable results misleadingly optimistic? Is the recent experience misleadingly optimistic? Is the outcome variability in the entire experience due to chance alone? What were the procedural risk factors in the experience, and does their variation across time explain the mortality? Can appropriate prospective monitoring techniques for surgeons be derived?

If a prospective, ongoing search for increasing (+) and/or decreasing (-) mortality (logistic regression) had been in place from the beginning, a + trend would have been suspected after 53, and nearly certain after 59 (Fig. 1). A - trend would have been suspected after 76, and considered probable after 80. Prospectively computed differences between actual mortality and that patient-specifically predicted from an equation derived from a multi-institutional study would have given similar inferences (Fig. 2). The surgeon did not make a decision to "retrain" until after 68.

Among the 93, origin of the left or circumflex coronary artery from sinus 2 and the case number were risk factors (Eq. 1; Fig. 3). When case number was withheld, either extensive surgical dissection of the coronary arteries (Eq. 2) or non-use of phenoxybenzamine (Eq. 3) became a risk factor (Table). No deaths have occurred since case 68 and a visit to surgeon C, and in an era characterized by removal of a large aortic button with the coronary, reimplantation into a medially hinged trap door, routine use of phenoxybenzamine, and nonuse of aprotinin and surgical glue.

			Risk-Adjusted	
			Hospital Mortality	
Equation	Coronary	Technique	%	CL (%)
2	Usual Cor	Exten. dissec N	1	0.5-3
		Exten.dissec Y	18	9-34
	L or Cx Sinus 2	Exten dissec N	9	5-16
		Exten.dissec Y	62	44-78

3	Usual	Phenoxy. Y	1	0.5-3
		Phenoxy. N	17	8-31
L or Cx Sinus 2		Phenoxy. Y	9	5-16
		Phenoxy. N	61	43-77

Thus, if "skill" is a risk factor, this can change across time and be neutralized. Prospective analyses can anticipate problems.

*By Invitation

54. Long-Term Outcome of Myocardial Revascularization in Children With Kawasaki Coronary Artery Disease

*SOICHIRO KITAMURA, M.D. *, YOICHIKAMEDA, M.D. *,*

*TOSHIO SEKI, M.D. *, MASAHIRO ENDO, M.D. *,*

*YASUO TAKEUCHI, M.D. *,*

TOMISAKU KAWASAKI, M.D. and*

YASUNARU KAWASHIMA, M.D.

Nara, Japan

The long-term outcome of myocardial revascularization by coronary artery bypass grafting (CABG) in children with severe obstructive coronary artery sequelae of Kawasaki disease (KD) is largely unknown. Multicenter postoperative data was acquired in 1991 with the help of the Bureau of Health and Welfare of Japan. A total of 168 children with a mean age of 10.6 ± 8.1 years, 127 male (75.6%) and 41 female (24.4%) who had undergone surgery was analyzed. Obstructive coronary artery disease was identified in 11.8% of the left main trunk (LMT), 77.6% of the RCA, 87.6% of the LAD and 25.9% of the CX arteries. Old myocardial infarction was present in 46.0% of the patients (pts). Among them, 54 pts (32%, 12.5 ± 9.7 y/o) underwent CABG with saphenous vein grafts (SVG) alone. The remaining 114 pts (68%, 9.8 ± 7.1 y/o) had grafting of at least one internal thoracic artery (ITA) to the LAD artery. The gastroepiploic artery was also utilized in 11 pts. There were no significant differences between the SVG and the ITA groups in the mean age at operation (12.5 vs 9.7 y/o), female ratio (22 vs 25%), previous history of infarction (50 vs 35.2%), impaired left ventricular function ($EF < 0.5$) (13 vs 11%), LMT disease (9.6 vs 10.0%), the number of vessel diseased 2.2 ± 0.8 , 2.0 ± 0.6 /pt) or the mean number of grafts placed (1.7 ± 0.7 vs 1.7 ± 0.7 /pt). The operative death rate was same (1.9 vs 0%), but the late cardiac death rate was significantly higher in the SVG group (13.0%) than in the ITA group (0.9%) ($p < 0.002$). Actuarial analysis showed a significantly higher survival rate in the ITA group (98.7 ± 1.2 vs $81.6 \pm 7.0\%$, $p < 0.05$) 90 months after surgery. The actuarial graft patency rate was higher in arterial grafts (77.1%, $n=151$) than in SVG (46.2%, $n=26$). [Conclusions] ITA grafts had significantly better long-term patency than SVG in children with KD who underwent CABG, and the use of ITA grafts resulted in a reduction in late cardiac death following surgery.

*By Invitation

55. Ventricular Function After Anatomical Repair in Patients With Atrioventricular Discordance

*YASU HARU IMAI, M.D., KAZUO SAWATARI, M.D. *,
SHUICHI HOSHINO, M.D. *, KAZUAKI ISHIHARA, M.D. *,
MAKOTO NAKAZAWA, M.D. *,
and KAZUO MOMMA, M.D. **

Tokyo, Japan

Double-switch operation for atrioventricular discordance (AVD) has an advantage of creating nearly normal atrioventricular and ventriculoarterial relationships, in which the anatomical left ventricle serves as a systemic ventricle postoperatively. Since June, 1989 to May, 1992, 16 patients with AVD ranging in ages from 1 to 12 years underwent double-switch operation consisting of 10 Mustard or 6 Senning procedures at atrial level, and of 2 arterial switch operations or 14 external conduit repairs at ventriculoarterial level. Mustard procedure with patch-augmentation of functional systemic atrium was selected for small right atrium, and arterial switch in cases with intact pulmonary valve. Prior to the anatomical correction, 17 palliative shunt procedures were performed in 10 cases, and pulmonary artery banding was done for preparation of anatomical left ventricle in one. All but one had ventricular septal defect, and 14 had pulmonary atresia or stenosis. Systemic tricuspid regurgitation (TR) was seen in 6 and was repaired by annuloplasty in 5. Situs solitus was seen in 10 and inverted situs in 6. Left and Right ventricular function were evaluated in the cineangiogram before and one month after operation. All but one survived the operation. One died of septicemia 33 days after operation. Cardiothoracic ratio increased from $50.6 \pm 6.1\%$ to $59.5 \pm 4.2\%$ postoperatively ($P < .0001$). Anatomical RV end-diastolic volume (RVEDV) showed significant reduction from $121 \pm 49\%$ of normal to $78 \pm 28\%$ due to unloading of the ventricle after operation ($P < .0009$) and RV ejection fraction (RVEF) remained unchanged ($57 \pm 0.08\%$ to $57 \pm 0.07\%$). Postoperatively systolic pressure in RV showed a marked decrease to 48 ± 17.4 mmHg and RV to LV systolic pressure ratio decreased to 0.50 ± 0.19 . Anatomical LVEDV and LVEF remained unchanged before and after operation; $132 \pm 52\%$ of normal to $133 \pm 35\%$ and LVEF from $60.9 \pm 0.09\%$ to $55.9 \pm 0.08\%$. Cardiac index was averaged 3.2 ± 0.5 after surgery ranging from 2.6 to 4.4. Although longer follow-up was warranted, double-switch operation would be best indicated for AVD especially in presence of systemic tricuspid regurgitation, or in cases with poor systemic RV.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

11:05 a.m. SIMULTANEOUS SCIENTIFIC SESSION F CONGENITAL HEART DISEASE

Grand Ballroom F

56. Results of Biventricular Repair for Double Outlet Right Ventricle

*JOSEPH M. FORBESS, M.D. *, MITSURU AOKI, M.D. *,
RICHARD A. JONAS, M.D., JOHN E. MAYER, JR., M.D.*

and ALDO R. CASTANEDA, M.D.

Boston, Massachusetts

From 10/1981 to 12/1991, 73 patients (pts) underwent biventricular repair for double outlet right ventricle (DORV, defined by the 50% rule) with AV concordance. Bilateral conus was documented in 51 pts. There were 8 hospital deaths (11 %), 2 late deaths (3%) and 18 pts (28%) required reoperations during 212 pt-year follow-up. The various anatomical subsets were correlated with surgical options and outcome. Numbers of hospital deaths are shown in parentheses.

Location of VSD	<i>n</i>	Associated lesions				Type of repair			
		SAS	SPS	AAO	IVR	ASO	Rastelli	DKS	Sen/Mus
subaortic	31(3)	4(1)	13(0)	1(0)	29(3)	□	2(0)	□	□
subpulmonary	27(3)	5(1)	4(0)	15(1)	5(0)	9(1)	3(1)	6(1)	4(0)
doubly committed	5(0)	3(0)	2(0)	2(0)	5(0)	□	□	□	□
noncommitted	10(2)	2(0)	5(1)	2(1)	4(1)	1(0)	2(1)	1(0)	1(0)
total	73(8)	14(2)	24(1)	20(2)	43(4)	10(1)	8(2)	7(1)	5(0)

SAS: subaortic stenosis, SPS: subpulmonary stenosis, AAO: aortic arch obstruction, IVR: intraven-tricular rerouting, ASO: arterial switch operation, DKS: Damus-Kay-Stansel type procedure, Sen/Mus: Senning or Mustard operation
Forty-five pts were operated during the 1st year of life and all 6 pts were operated upon during the 1st month of life had VSDs not committed to the aorta and also left-sided obstructions (hypoplastic ascending aorta in 2, SAS in 4, and coarctation in all). Anatomy which precluded IVR included SPS with subpulmonary coronary artery, insufficient tricuspid-pulmonary annular distance, abnormal tricuspid valve insertion to conal septum, hypoplastic aortic valve, and severely restrictive subpulmonary VSD. Two of the pts who had ASO required also a RV-PA conduit for RV outflow obstruction with coronaries crossing the infundibulum. SPS, hypoplastic aortic annulus with pulmonary hypertension, or unfavorable coronary anatomy for ASO were the reasons for Rastelli or DKS operations. Sen/Mus were done mostly for the pts with complex coronary anatomy precluding right ventriculotomy and ASO. Reasons for the 21 reoperations (in 18 pts) included recurrent SAS in 8 (none with subaortic VSD), extracardiac conduit obstruction in 5, residual VSD (multiple) in 3, and SPS in 2. Two pts after a DKS developed significant regurgitation of the native aortic valve, and 1 pt after a Senning operation developed severe tricuspid regurgitation. This pt underwent preliminary PA banding in preparation for ASO.

The majority of pts with DORV and subaortic and doubly committed VSDs can have IVR at low risk for total mortality and reoperations. Pts with DORV and subpulmonary or noncommitted VSDs have a high incidence of significant associated intra- and extracardiac pathology and complex coronary patterns, which require detailed preoperative evaluation in order to select the most appropriate operation.

*By Invitation

57. Correction of Total Anomalous Pulmonary Venous Connection in Infancy

FLAVIAN M. LUPINETTI, M.D. *, THOMAS J. KULIK, M.D. *,

ROBERTH. BEEKMAN, M.D. *,

DENNIS C. CROWLEY, M.D. *

and EDWARD L. BOVE, M.D.

From 1985 through 1992, 39 patients under one year of age underwent operative correction of isolated total anomalous pulmonary venous connection (TAPVC). Twenty-two patients were male and 17 were female. The median age at operation was 10 days (range 1-282 days) and weight was 3.7 kg (2.5-5.2 kg). Connection was to the supracardiac innominate vein in 19, to the coronary sinus or right atrium in eight, infradiaphragmatic in ten, and mixed supracardiac and cardiac in two. Obstruction of the pulmonary veins was severe in 23, mild in three, and absent in 13. Preoperative stabilization included mechanical ventilation for 14 patients for a mean duration of 2.6 days and extracorporeal membrane oxygenation (ECMO) for one patient for one day. All operations were performed with deep hypothermia and circulatory arrest (mean arrest time 33 minutes). Supracardiac connections were repaired by performing a side-to-side anastomosis between the pulmonary venous confluence and the dome of the left atrium using a superior approach between the superior vena cava and the aorta. Coronary sinus connections were repaired by enlarging the atrial septal defect and the coronary sinus communication with the left atrium, closing the atrial defect with a large patch. Infracardiac repairs included elevation and rotation of the heart to the right and an elongated side to side anastomosis between the common venous confluence and the body and appendage of the left atrium sewn from outside the heart. Following operation, median duration of inotropic support was three days, duration of ventilatory support was three days, and duration of hospitalization was ten days. There were no hospital or late deaths (70% CI: 0-27%). One patient required two subsequent reoperations for persistent pulmonary venous obstruction and another patient developed superior vena cava obstruction requiring reoperation. A third patient, who was on ECMO preoperatively, has remained ventilator dependent, probably as a consequence of pulmonary lymphangiectasia. All other patients are alive and well with a mean follow-up of 22 months (range 1-76 months). Operative treatment of TAPVC in infants can be performed with low mortality and an infrequent need for reoperations.

*By Invitation

58. AV Valve Competence After Takedown to Improve Exposure During VSD Repair

*ARA K. PRIDJIAN, M.D. *, BENNETT F. PEARCE, M.D. *,*

*WALTER S. CULPEPPER, M.D. *,*

*LUTHER C. WILLIAMS, M.D.**

and JOHN L. OCHSNER, M.D.

New Orleans, Louisiana

Although the AV valve and its attachments can sometimes obscure the superior margin of a VSD, concern for valvular competence has made surgeons hesitant to take down the AV valve. From May, 1982 to the present, we have taken down the right AV valve to improve exposure for VSD repair in selected patients. Medical records of 40 patients repaired in this manner were reviewed and follow-up echocardiographic studies, available in 32 patients, were examined to determine the degree of tricuspid regurgitation. Patients ranged in age from 2 months to 4.5 years (mean 18 months), having weights of 2.7 to 26 kg (median 5.5 kg.). There were 32 patients with isolated perimembranous defects, 1 with an isolated inlet defect, 4 with subaortic defects and DORV, 2 with perimembranous defects and DCRV, and 1 with subaortic defect and subaortic membrane. Mean preoperative pulmonary to systemic blood flow ratio was 4.5:1, and pulmonary vascular resistance ranged from 0.5 to 6.6 Woods units (mean 2.9).

Contiguous portions of the anterior and septal leaflets of the valve were taken down at the annulus. Valve leaflets were re-suspended after VSD repair with running polypropylene. The degrees of valvular regurgitation determined by echo were graded as none, trivial, moderate,

or severe based on the area of the color-flow jet. Valvular regurgitation was graded as none in 22 and trivial in 10. No patient had moderate or severe regurgitation. There were no early or late deaths in any patients undergoing VSD repair by this approach. Takedown and re-suspension of the AV valve is a safe and effective technique which improves exposure for VSD repair and does not adversely affect valve competence.

12:10 p.m. ADJOURN

*By Invitation

GEOGRAPHICAL ROSTER

NECROLOGY

Nina S. Braunwald, M.D. Boston, MA

Cecil M. Couves, M.D. Kelowna, BC

E. Stanley Crawford, M.D. Houston, TX

Ralph Deterling, Jr., M.D. Boston, MA

Sanford French, III, M.D. San Antonio, TX

Farouk S. Idriss, M.D. Chicago, IL

John L. Keeley, M.D. Maywood, IL

Philip J. Kunderman, M.D. New Brunswick, NJ

Hiram T. Langston, M.D. Savannah, GA

Georg Rodewald, M.D. Hamburg, Germany

Robert R. Shaw, M.D. Dallas, TX

Joseph L. Timmes, M.D. Short Hills, NJ

John S. Vasko, M.D. Columbus, OH

Elton Watkins, Jr., M.D. Burlington, MA

The American Association for Thoracic Surgery

(Listed by Countries, States, Provinces and Cities)

Geographical - UNITED STATES

1992-1993

USA

ALABAMA

Birmingham

Blackstone, Eugene H

Blakemore, William S

Kahn, Donald R

Kessler, Charles R

Kirklin, James K

Kirklin, John W

McElvein, Richard B

Pacifico, Albert D

Montgomery

Simmons, Earl M

ARIZONA

Green Valley

McClenathan, James E

Mesa

Fisk, R Leighton

Paradise Valley

Nelson, Arthur R

Phoenix

Brown, Lee B

Cornell, William P

Scottsdale

Pluth, James R

Sun City

Read, C Thomas

Tucson

Burbank, Benjamin

Copeland, Jack G, III

Sanderson, Richard G

Sethi, Gulshan K

CALIFORNIA

Anaheim

Main, F Beachley

Capistrano Beach

Flynn, Pierce J

Chico

Becker, Ronald M

Coronado

Silver, Arthur W

Covina

Carter, P Richard

El Cajon

Long, David M, Jr

El Macero

Andrews, Neil C

Escondido

Mannix, Edgar P, Jr

Flintridge

Penido, John R F

Fresno

Evans, Byron H

Guernsey, James M

Indian Wells

Salyer, John M

Inglewood

Lee, Myles E

Irvine

Connolly, John E

La Canada

Meyer, Bert W

La Jolla

Baisch, Bruce F

ARKANSAS

Jasper

Hudson, W A

Little Rock

Campbell, Gilbert S

Read, Raymond C

Williams, G Doyme

Fosburg, Richard G

Hutchin, Peter

Loma Linda

Bailey, Leonard L

Gundry, Steven R

Wareham, Ellsworth E

Long Beach

Bloomer, William E

Carlson, Herbert A

Stemmer, Edward A

Los Angeles

Buckberg, Gerald D

Davis, Lowell L

DeMeester, Tom R

Fonkalsrud, Eric W

Holmes, E Carmack

Kay, Jerome Harold

Khonsari, Siavosh

Laks, Hillel

Lindesmith, George G

Longmire, William, Jr

Maloney, James V, Jr

Mandal, Ashis K

Matloff, Jack M

Mulder, Donald G

Waters, Paul F

Los Osos

Aronstam, Elmore M

Montebello

Lui, Alfred H F

Newport Beach

Boyd, Thomas F

Oakland

Sacramento

Benfield, John R

Berkoff, Herbert A

Follette, David M

Harlan, Bradley J

Hurley, Edward J

Miller, George E, Jr

Smeloff, Edward A

San Bernardino

Misbach, Gregory A

San Diego

Baronofsky, Ivan D

Chambers, John S, Jr

Daily, Pat O

Jamieson, Stuart W

Lamberti, John J, Jr

Moreno-Cabral, Ricardo

Trummer, Max J

San Francisco

Ellis, Robert J

Gardner, Richard E

Grimes, Orville F

Hill, J Donald

Leeds, Sanford E

McEnany, M Terry

Rankin, J Scott

Richards, Victor

Ecker, Roger R	Roe, Benson B
Iverson, Leigh I G	Thomas, Arthur N
May, Ivan A	Turley, Kevin
Orange	Ullyot, Daniel J
Gazzaniga, Alan B	San Jose
Wakabayashi, Akio	Oakes, David D
Oxnard	San Marino
Dart, Charles H, Jr	Tsuji, Harold K
Palm Springs	Santa Ana
Goldman, Alfred	Pratt, Lawrence A
Palo Alto	Santa Barbara
Cohn, Roy B	Higginson, John F
Fogarty, Thomas J	Jahnke, Edward J, Jr
Jamplis, Robert W	Lewis, F John
Peters, Richard M	Love, Jack W
Wilson, John L	Santa Cruz
Palos Verdes Estates	Fishman, Noel H
Stiles, Quentin R	Santa Monica
Pasadena	Morton, Donald L
Hughes, Richard K	Nelson, Ronald J
Ingram, Ivan N	Robertson, John M
Newman, Melvin M	Santa Rosa
Pebble Beach	Neville, William E
Ramsay, Beatty H	St Helena
	Dugan, David J
Stanford	Hartford
Mark, James B D	Kemler, R Leonard
Miller, D Craig	New Haven
Oyer, Philip E	Baldwin, John C
Reitz, Bruce A	Glenn, William W L
Shochat, Stephen J	Hammond, Graeme L
Shumway, Norman E	Kopf, Gary S
Starnes, Vaughn A	New Miltord
Stinson, Edward B	Okinaka, Arthur J
Tiburon	Norwich
Heydorn, William H	Kelley, Winfield O
Torrance	Sharon

Carey, Joseph S
Cukingnan, Ramon A
Moore, Thomas C
State, David
Victorville
Jurado, Roy A
COLORADO
Aspen
Zaroff, Lawrence I
Colorado Springs
Burrington, John D
Denver
Brown, Robert K
Clarke, David R
Condon, William B
Eiseman, Ben
Grover, Frederick L
Grow, John B
Harken, Alden H
Hopeman, Alan R
Paton, Bruce C
Pomerantz, Marvin
Rainer, W Gerald
Wright, George W
Englewood
Kovarik, Joseph L
Lakewood
Swan, Henry
Littleton
Pappas, George
Pueblo
Hartley, Thomas D
Vail
Fuller, Josiah
CONNECTICUT
Avon
Maier, Herbert C

Wylie, Robert H
Wilton
Pool, John L
Woodbridge
Lindskog, Gustaf E
Stansel, Horace C, Jr
Stern, Harold
DELAWARE
Newark
Lemole, Gerald M
Wilmington
Pecora, David V
DISTRICT OF COLUMBIA
Washington
Aaron, Benjamin L
Edwards, Fred H
Gomes, Mario N
Katz, Nevin M
Keshishian, John M
Lefemine, Armand A
Midgley, Frank M
Randolph, Judson G
Simmons, Robert L
Wallace, Robert B
FLORIDA
Atlantic Beach
Stranahan, Allan
Belleair
Lasley, Charles H
Boca Raton
Seley, Gabriel P
Clearwater
Wheat, Myron W, Jr
Coconut Grove
Center, Sol
Coral Gables
Cooke, Francis N

Bridgeport
Rose, Daniel M

Delray Beach
Shumacker, Harris B, Jr

Hartford

Gainesville
Alexander, James A

GEORGIA
Atlanta

Jacksonville

Koster, J Kenneth, Jr

Stephenson, Sam, Jr

Graver, Joseph M

Guyton, Robert A

Hatcher, Charles, Jr

Hopkins, William A

Jupiter

Gerbasi, Francis S

Jones, Ellis L

Lakeland

Brown, Ivan W, Jr

King, Richard

Lee, Arthur B, Jr

Marathon

Mangiardi, Joseph L

Mansour, Kamal A

Miller, Joseph I

Marco

Schuster, Samuel R

Rivkin, Laurence M

Symbas, Panagiotis

Miami

Bolooki, Hooshang

Williams, Willis H

Augusta

Chesney, John G

Daughtry, Dewitt C

Ellison, Robert G

Rubin, Joseph W

Greenberg, Jack J

Chickamanga

Jude, James R

Hall, David P

Kaiser, Gerard A

Macon

MacGregor, David C

Dalton, Martin L, Jr

Papper, Emanuel M

Sealy, Will C

Reis, Robert L

Van De Water, Joseph M

Ripstein, Charles B

Marietta

Subramanian, S

Bailey, Charles P

Thurer, Richard J

Savannah

Wilder, Robert J

Yeh, Thomas J

Miami Beach
Spear, Harold C

HAWAII
Honolulu

Naples

Battersby, James S

Ching, Nathaniel P

Gebauer, Paul W

Linberg, Eugene J

McNamara, Joseph J

Smyth, Nicholas P D

IDAHO

North Palm Beach

Dorsey, John M

Orlando

Scott, Meredith L

Sherman, Paul H

Ponte Vedra Beach

Gilbert, Joseph, Jr

Punta Gorda

Taber, Rodman E

St Petersburg

Daicoff, George R

DeMatteis, Albert

Tallahassee

Kraeft, Nelson H

Tampa

Angell, William W

Seiler, Hawley H

Winter Haven

Maurer, Elmer P R

Winter Park

Bloodwell, Robert D

Michaelis, Lawrence

Najafi, Hassan

Raffensperger, John

Shields, Thomas W

Tatooles, Constantine

Thomas, Paul A, Jr

Vanecko, Robert M

Warren, William H

Downers Grove

Leininger, Bernard J

Evanston

Anderson, Robert W

Fry, Willard A

Glencoe

Boise

Herr, Rodney H

ILLINOIS

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Barker, Walter L

Breyer, Robert H

Campbell, Charles D

Ebert, Paul A

Faber, L Penfield

Ferguson, Mark K

Goldin, Marshall D

Hanlon, C Rollins

Hartz, Renee S

Head, Louis R

Hunter, James A

Karp, Robert B

Kittle, C Frederick

Mavroudis, Constantine

Council Bluffs

Sellers, Robert D

Des Moines

Dorner, Ralph A

Phillips, Steven J

Zeff, Robert H

Iowa City

Behrendt, Douglas M

Ehrenhaft, Johann L

Rossi, Nicholas P

Stanford, William

KANSAS

Cunningham

Allbritten, Frank F, Jr

Rubenstein, L H
Harvey
Norman, John C
Replogle, Robert L
Hines
Mason, G Robert
Maywood
DeLeon, Serafin Y
Montoya, Alvaro
Pifarre, Roque
Sullivan, Henry J
Oak Brook
Hudson, Theodore R
Ilbawi, Michel N
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
Indianapolis
Brown, John W
King, Harold
King, Robert D
Mandelbaum, Isidore
Siderys, Harry
IOWA
Cedar Rapids
Lawrence, Montague S
Levett, James M

Lawrence
Miller, Don R
Shawnee Mission
Adelman, Arthur
Wichita
Tocker, Alfred M
KENTUCKY
Lexington
Crutcher, Richard R
Dillon, Marcus L, Jr
Todd, Edward P J
Louisville
Gray, Laman A, Jr
Mahaffey, Daniel E
Ransdell, Herbert, Jr
LOUISIANA
Alexandria
Knoepp, Louis F
Baton Rouge
Berry, B Eugene
Beskin, Charles A
Metairie
Ochsner, Alton, Jr
New Orleans
Blalock, John B
DeCamp, Paul T
Hewitt, Robert L
Lindsey, Edward S
McFadden, Paul M
Mills, Noel L
Moulder, Peter V
Ochsner, John L
O'Neill, Martin J, Jr
Pearce, Charles W
Rosenberg, Dennis M
Schramel, Robert J
Webb, Watts R

MAINE

Liberty

Hurwitz, Alfred

Portland

Bredenberg, Carl E

Drake, Emerson H

Rockport

Swenson, Orvar

Windham

Hiebert, Clement

MARYLAND

Baltimore

Attar, Safuh

Baker, R Robinson

Baumgartner, William A

Blair, Emil

Dodrill, Forest D

Gardner, Timothy J

Gott, Vincent L

Haller, J Alex, Jr

McLaughlin, Joseph S

Michelson, Elliott

Salomon, Neal W

Turney, Stephen Z

Watkins, Levi, Jr

Bethesda

Jones, Michael

Pass, Harvey I

Chevy Chase

Iovine, Vincent M

Ellicott City

MacManus, Joseph E

Towson

Brawley, Robert K

Worton

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

Mathisen, Douglas J

Mayer, John E

Moncure, Ashby C

Neptun.-, Wilford B

Rheinlander, Harold F

Russell, Paul S

Scannell, J Gordon

Shemin, Richard J

Starkey, George W B

Thurer, Robert L

Vlahakes, Gus J

Weintraub, Ronald

Boylston

Okike, Okike N

Brookline

Madoff, Irving M

Burlington

Shahian, David M

Cambridge

Harken, Dwight E

Malcolm, John A

Chestnut Hill

Laforet, Eugene G

Strieder, John W

Concord

Walkup, Harry E	Soutter, Lamar
MASSACHUSETTS	Dover
Boston	Black, Harrison
Akins, Cary W	Lynnfield
Austen, W Gerald	Wesolowski, Sigmund A
Barsamian, Ernest M	Medford
Berger, Robert L	Desforges, Gerard
Bougas, James A	Methuen
Buckley, Mortimer J	Wilson, Norman J
Burke, John F	North Andover
Castaneda, Aldo R	Cook, William A
Cleveland, Richard J	Shrewsbury
Cohn, Lawrence H	Moran, John M
Collins, John J	Springfield
Daggett, Willard M	Engelman, Richard M
Daly, Benedict D T	Rousou, John A
Diehl, James T	Vineyard Haven
	Malm, James R
West Roxbury	Rochester
Bernhard, William F	Bernatz, Philip E
Khuri, Shukri F	Danielson, Gordon K
Westport Harbor	McGoon, Dwight C
Findlay, Charles W, Jr	McGregor, Christopher G A
Williamstown	Olsen, Arthur M
Wilkins, Earle W, Jr	Orszulak, Thomas A
Worcester	Pairolero, Peter C
Vander Salm, Thomas J	Payne, W Spencer
MICHIGAN	Puga, Francisco J
Ann Arbor	Schaff, Hartzell V
Bartlett, Robert H	Trastek, Victor F
Bove, Edward L	St Paul
Gago, Otto	Lillehei, C Walton
Greenfield, Lazar J	Miller, Fletcher A
Kirsh, M.Tvin M	Waubun
Morris, Joe D	DeNiord, Richard N

Neerken, A John	MISSISSIPPI
Orringer, Mark B	Carthage
Prager, Richard L	Logan, William D, Jr
Sloan, Herbert	Jackson
Birmingham	Hardy, James D
Timmis, Hilary H	Johnston, J Harvey, Jr
Detroit	Netterville, Rush E
Arbulu, Agustin	MISSOURI
Silverman, Norman A	Bridgeton
Steiger, Zwi	Codd, John E
Stephenson, Larry W	Chesterfield
Wilson, Robert F	Bergmann, Martin
Grand Rapids	Columbia
Harrison, Robert W	Bryant, Lester R
Meade, Richard H	Curtis, Jack J
Rasmussen, Richard A	Silver, Donald
Tomatis, Luis A	Kansas City
Grosse Pointe	Ashcraft, Keith W
Benson, Clifford D	Benoit, Hector W, Jr
West Bloomfield	Borkon, A Michael
Arciniegas, Eduardo	Holder, Thomas M
MINNESOTA	Killen, Duncan A
Minneapolis	Mayer, John H, Jr
Arom, Kit V	Padula, Richard T
Bolman, R Morton, III	Piehler, Jeffrey M
Emery, Robert W	Reed, William A
Foker, John E	Van Way, Charles W, III
Gannon, Paul G	Mount Vernon
Garamella, Joseph J	Campbell, Daniel C, Jr
Helseth, Hovald K	St Louis
Humphrey, Edward W	Baue, Arthur E
Kaye, Michael P	Connors, John P
Riser, Joseph C	Cooper, Joel D
Molina, J Ernesto	Cox, James L
Nicoloff, Demetre M	Ferguson, Thomas B

Flye, M Wayne	Moorestown
Johnson, Frank E	Morse, Dryden P
Kaiser, George C	Morristown
Kouchoukos, Nicholas T	Parr, Grant V S
Lewis, J Eugene, Jr	Mount Laurel
Naunheim, Keith S	Pierucci, Louis, Jr
Pasque, Michael K	Neptune
Patterson, G Alec	Roberts, Arthur J
Pennington, D Glenn	New Brunswick
Roper, Charles L	Lewis, Ralph J
Spray, Thomas L	MacKenzie, James W
Weldon, Clarence S	Scholz, Peter M
Willman, Vallee L	Newark
MONTANA	Abel, Ronald M
Missoula	Donahoo, James
Oury, James H	Gielchinsky, Isaac
Twin Bridges	Hochberg, Mark S
Lower, Richard R	Swan, Kenneth G
NEBRASKA	Paterson
Omaha	Bregman, David
Fleming, William H	Tenaflly
Schultz, Richard D	Gerst, Paul H
NEVADA	NEW MEXICO
Las Vegas	Albuquerque
Little, Alex G	Edwards, W Sterling
Swain, Julie A	Las Vegas
NEW HAMPSHIRE	Thai, Alan P
Franconia	Santa Fe
Taylor, Warren J	Davila, Julio C
Jaffrey	Silver City
Woods, Francis M	Waddell, William R
NEW JERSEY	NEW YORK
Alpine	Albany
Holswade, George R	Foster, Eric D
Bellville	Bay Shore
Gerard, Franklyn P	Ryan, Bernard J
Browns Mills	Bronx

Fernandez, Javier	Altai, Lari A
McGrath, Lynn B	Brodman, Richard F
Camden	Fell, Stanley C
Camishion, Rudolph C	Ford, Joseph M
DelRossi, Anthony J	Prater, Robert W M
East Orange	Gay, William A, Jr
Auerbach, Oscar	Hirose, Teruo
Hackensack	Brooklyn
Hutchinson, John E, III	Cunningham, Joseph N, Jr
Jersey City	Levowitz, Bernard S
Demos, Nicholas J	Sawyer, Philip N
Millburn	Buffalo
Parsonnet, Victor	Adler, Richard H
	Andersen, Murray N
Bhayana, Joginder N	Redo, S Frank
Hoover, Eddie L	Reemtsma, Keith
Lajos, Thomas Z	Rose, Eric A
Cooperstown	Rusch, Valerie W
Blumenstock, David A	Skinner, David B
East Meadow	Smith, Craig R
Strevey, Tracy E, Jr	Spencer, Frank C
Fayetteville	Spotnitz, Henry M
Bugden, Walter F	Steichen, Felicien M
Effler, Donald B	Subramanian, Valavanur A
Floral Park	Tice, David A
Crastnopol, Philip	Tyras, Denis H
Lido Beach	Veith, Frank J
Hines, George L	Wichern, Walter, Jr
Loudonville	Wolff, William I
Alley, Ralph D	Patchogue
New Hyde Park	Finnerty, James
Amato, Joseph J	Plattsburg
Earnar, Hendrick B	Potter, Robert T
New Rochelle	Rochester
Rubin, Morris	Craver, William L

New York

Acinapura, Anthony J

Adams, Peter X

Anagnostopoulos, C E

Bains, Manjit S

Seattle, Edward, Jr

Bloomberg, Allan E

Boyd, Arthur D

Burt, Michael E

Cahan, William G

Clauss, Roy H

Conklin, Edward F

Cracovaner, Arthur J

Culliford, Alfred T

Ergin, M Arisan

Friedlander, Ralph

Ginsberg, Robert J

Green, George E

Oriepp, Randall B

Holman, Cranston W

Isom, O W<.yne

Jaretzki, Allied, III

King, Thomas C

Kirschner, Paul .'*

Krieger, Karl H

Lambert, Adrian

Litwak, Robert S

Martini, Nael

McCord, Colin W

McCormack, Patricia M

Nealon, Thomas F, Jr

Quaegebeur, Jan M

DeWeese, James A

Hicks, George L

Schwartz, Seymour I

Stewart, Scott

Roslyn

Thomson, Norman B, Jr

Wisoff, B George

Saranac Lake

Decker, Alfred M, Jr

Scarsdale

Robinson, George

Scottsville

Emerson, George L

Slingerlands

Kausel, Harvey W

Staten Island

Garzon, Antonio A

Stony Brook

Dennis, Clarence

Soroff, Harry S

Syracuse

Brandt, Berkeley, III

Meyer, John A

Parker, Frederick, Jr

Valhalla

Moggio, Richard A

Reed, George E

NORTH CAROLINA

Asheville

Belts, Reeve H

Scott, Stewart M

Takaro, Timothy

Chapel Hill

Bowman, Frederick, Jr

Grondin, Claude M

Groves, Laurence K

Keagy, Blair A	Kay, Earle B
Starek, Peter J K	Loop, Floyd D
Wilcox, Benson R	Lytle, Bruce W
Charlotte	Snow, Norman J
Robicsek, Francis	Van Heeckeren, Daniel W
Selle, Jay G	Columbus
Taylor, Frederick H	Clatworthy, H W, Jr
Durham	Davis, J Terrance
Jones, Robert H	Kakos, Gerard S
Lowe, James E	Kilman, James W
Oldham, H N, Jr	Meckstroth, Charles
Sabiston, David C	Myerowitz, P David
Wolfe, Walter G	Williams, Thomas E, Jr
Young, W Glenn, Jr	Dayton
Greenville	DeWall, Richard A
Chitwood, W Randolph	Pepper Pike
Oriental	Mendelsohn, Harvey J
Deaton, W Ralph, Jr	OKLAHOMA
Pinehurst	Jenks
Fischer, Walter W	LeBeck, Martin B
Sugar Grove	Lawton
Gentsch, Thomas O	Barnhorst, Donald A
Winston-Salem	Oklahoma City
Cordell, A Robert	Elkins, Ronald C
Crosby, Ivan K	Felton, Warren L, II
Hammon, John W, Jr	Fisher, R Darryl
Hudspeth, Allen S	Greer, Allen E
Meredith, Jesse H	Munnell, Edward R
Mills, Stephen A	Williams, G Rainey
NORTH DAKOTA	Zuhdi, M Nazih
Grand Forks	OREGON
James, Edwin C	Days Creek
OHIO	Miller, Arthur C
Chagrin Falls	Portland
Cross, Frederick S	Cobanoglu, Adnan
Cincinnati	Krause, Albert H
Albers, John E	Okies, J Edward

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
Ankeney, Jay L
Cosgrove, Delos M
Geha, Alexander S

Camp Hill
Pennock, John L
Carlisle
DeMuth, William, Jr
Darby
McKeown, John J, Jr
Erie
Kerth, William J
Hershey
Campbell, David B
Myers, John L
Pae, Walter E, Jr
Pierce, William S
Waldhausen, John A
Johnstown
Kolff, Jacob
Lancaster
Bonchek, Lawrence I
Rosemond, George P
Witmer, Robert H
Philadelphia
Addonizio, Paul V

Poppe, J Karl
Starr, Albert
PENNSYLVANIA
Abington
Frobese, Alfred S
Ardmore
Hargrove, W Clark, III
Bethlehem
Snyder, John M
Bryn Mawr
Haupt, George J
Mundth, Eldred D

Wynnewood
Wallace, Herbert W
Yardley
Sommer, George N, Jr

RHODE ISLAND
Providence

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

SOUTH CAROLINA

Charleston

Bradham, R Randolph
Crawford, Fred A, Jr
Parker, Edward F
Sade, Robert M

Columbia

Almond, Carl H

Isle of Palms

Mullen, Donald C

Landrum

Stayman, Joseph W

Spartanburg

Bowles, L Thompson
Brockman, Stanley K
DiSesa, Verdi J
Dunn, Jeffrey M
Edie, Richard N
Edmunds, L Henry, Jr
Fineberg, Charles
Kaiser, Larry R
Levine, Frederick H
MacVaugh, Horace, III
Nemir, Paul, Jr
Norwood, William I
Pittsburgh
Bahnson, Henry T
Clark, Richard E
Ford, William B
Griffith, Hartley P
Hardesty, Robert L
Magovern, George J
Pontius, Robert G
Rams, James J
Rosemont
Sink, James D
Templeton, John, III
Sayre
Sewell, William H
Wayne
Lemmon, William M
Wyncote
Mendelssohn, Edwin

TEXAS
Amarillo

Sutherland, R Duncan
Austin

Utley, Joe R
TENNESSEE
Knoxville
Blake, Hu Al
Brott, Walter H
Domm, Sheldon E
Memphis
Cole, Francis H
Eastridge, Charles E
Garrett, H Edward
Howard, Hector S, Jr
Hughes, Felix A, Jr
McBurney, Robert P
Pate, James W
Robbins, S Gwin, Sr
Rosensweig, Jacob
Skinner, Edward F
Watson, Donald C
Nashville
Alford, William, Jr
Bender, Harvey W, Jr
Gobbel, Walter G, Jr
Merrill, Walter H
Sawyers, John L
Scott, Henry W, Jr
Stoney, William S
Thomas, Clarence, Jr
Sparta
Labrosse, Claude C

Kaufman
Davis, Milton V
Lubbock
Bricker, Donald L

Hood, R Maurice
Burnet
Ross, Raleigh R
Coppell
McPhail, Jasper L
Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Lambert, Gary J
Mills, Lawrence J
Paulson, Donald L
Platt, Melvin R
Razzuk, Maruf A
Ring, W Steves
Seybold, William D
Sugg, Winfred L
Urschel, Harold, Jr
Dilley
Hood, Richard H, Jr
El Paso
Glass, Bertram A
Fort Sam Houston
Cohen, David J
Zajтчuk, Rostik
Galveston
Conti, Vincent R
Derrick, John R
Tyson, Kenneth R T
Zwischenberger, Joseph B
Houston
Beall, Arthur C, Jr
Burdette, Walter J
Cooley, Denton A
DeBakey, Michael E
Frazier, Oscar H

Feola, Mario
Wallsh, Eugene
San Antonio
Dooley, Byron N
Heaney, John P
Treasure, Robert L
Trinkle, J Kent
Temple
Brindley, G V, Jr
Woodville
Harrison, Albert W
UTAH
Salt Lake City
Cutler, Preston R
Doty, Donald B
Liddle, Harold V
Mortensen, J D
Nelson, Russell M
Wolcott, Mark W
VERMONT
Burlington
Coffin, Laurence H
Miller, Donald B
Chester Depot
Adams, Herbert D
West Dover
Humphreys, G H, II
White River Junction
Tyson, M Dawson
VIRGINIA
Annandale
Akl, Bechara F
Lefrak, Edward A
Arlington
Conrad, Peter W
Klepser, Roy G

Hallman, Grady L	Charlottesville
Henly, Walter S	Dammann, John F
Jones, James W	Kron, Irving L
Lawrie, Gerald M	Minor, George R
Mattox, Kenneth L	Muller, William, Jr
Morris, George C, Jr	Nolan, Stanton P
Mountain, Clifton F	Spotnitz, William D
Ott, David A	Lynchburg
Overstreet, John W	Moore, Richmond L
Reul, George J, Jr	McLean
Roth, Jack A	Mills, Mitchell
Walker, William E	
Wukasch, Don C	
Richmond	Huntington
Bosher, Lewis H, Jr	Gonzalez-Lavin, Lorenzo
Brooks, James W	Morgantown
Cole, Dean B	Graeber, Geoffrey M
Gwathmey, Owen	Gustafson, Robert A
Wechsler, Andrew S	Murray, Gordon F
WASHINGTON	Warden, Herbert E
Bellingham	Parkersburg
Varco, Richard L	Tarnay, Thomas J
Friday Harbor	WISCONSIN
Lawrence, G Hugh	Delafield
Issaquah	Hausmann, Paul F
Jarvis, Fred J	La Crosse
Kirkland	Gundersen, Erik A
Mills, Waldo O	Madison
Poulsbo	Chopra, Paramjeet S
Malette, William G	Kroncke, George M
Seattle	Mentzer, Robert M, Jr
Anderson, Richard P	Young, William P
Ashbaugh, David G	Marshfield
Dillard, David H	Myers, William O
Hill, Lucius D, 111	Ray, Jefferson F, III

Jones, Thomas W
Li, Wei-I
Manhas, Dev R
Mansfield, Peter B
Miller, Donald W, Jr
Rittenhouse, Edward A
Sauvage, Lester
Thomas, George I
Verrier, Edward D
Spokane
Berg, Ralph, Jr
WEST VIRGINIA
Charlestown
Walker, James H

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

CANADA

ALBERTA
Calgary
Bharadwaj, Baikunth
Miller, George E
Edmonton
Callaghan, John C
Gelfand, Elliot T
Sterns, Laurence P
BRITISH COLUMBIA
Vancouver
Allen, Peter
Ashmore, Phillip G
Jamieson, W R Eric
Tyers, G Frank O
Victoria
Stenstrom, John D
West Vancouver
Robertson, Ross
MANITOBA

Sudbury
Field, Paul
Walker, George R
Toronto
Baird, Ronald J
Bigelow, Wilfred G
Coles, John G
David, Tirone E
Delarue, Norman C
Goldberg, Melvyn
McKneally, Martin F
Mickleborough, Lynda L
Pearson, F Griffith
Salerno, Tomas A
Scully, Hugh E
Trimble, Alan S
Trusler, George A
Weisel, Richard D
Williams, William G

Winnipeg

Barwinsky, Jaroslaw

Cohen, Morley

NOVA SCOTIA**Halifax**

Landymore, Roderick W

Murphy, David A

Mabou

Thomas, Gordon W

ONTARIO**Collingwood**

Heimbecker, Raymond

London

McKenzie, F Neil

North York

Goldman, Bernard S

Nottawa

Key, James A

Ottawa

Keon, Wilbert J

Todd, Thomas R J

Westbrook

Lynn, R Beverley

QUEBEC**Montreal**

Blundell, Peter E

Chiu, Chu-Jeng (Ray)

Cossette, Robert

Dobell, Anthony R

Duranceau, Andre C H

Lepage, Gilles

MacLean, Lloyd D

Morin, Jean E

Mulder, David S

Pelletier, Conrad L

Scott, Henry J

Sainte-Foy

DesLauriers, Jean

Sillery

Grondin, Pierre

OTHER COUNTRIES**AFGHANISTAN****Kabul**

Hankins, John R

ARGENTINA**Buenos Aires**

Favaloro, Rene G

AUSTRALIA**SOUTH AUSTRALIA****Piccadilly**

Sutherland, H D'Arcy

VICTORIA**Melbourne**

Nossal, Gustav J V

FRANCE**Bordeaux**

Couraud, Louis

Meudon

Cachera, Jean Paul

Montpellier

Thevenet, Andre A

Paris

Binet, Jean-Paul

Blondeau, Philip

Cabrol, Christian E A

Carpentier, Alain F

Piwnica, Armand H

AUSTRIA

Puchenau

Bruecke, Peter E

Salzburg

Unger, Felix H

Vienna

Wolner, Ernst

BRAZIL

Rio de Janeiro

Meier, Milton A

Sao Paulo

Jatene, Adib D

Zerbini, E J

ENGLAND

Bath, Avon

Belsey, Ronald

Cambridge

Kennedy, John H

Herefordshire

Smith, Roger A

London

Braimbridge, Mark V

deLeval, Marc R

Lennox, Stuart C

Lincoln, Christopher R

Ross, Donald N

Stark, Jaroslav F

Taylor, Kenneth M

Thompson, Vernon C

Yacoub, Magdi

FINLAND

Kauniainen

Mattila, Severi P

Osaka

Kawashima, Yasunaru

Pessac, Bourdeaux

Fontan, Francis M

Suresnes

Bachet, Jean E

GERMANY

Aachen

Messmer, Bruno J

Hannover

Borst, Hans G

Munich

Sebening, Fritz

Neuss

Bircks, Wolfgang H

GUATEMALA

Guatemala City

Herrera, Rodolfo

INDIA

Raiputana

Van Allen, Chester M

IRELAND

Dublin

O'Malley, Eoin

ITALY

Bergamo

Parenzan, Lucio

Milan

Peracchia, Alberto

Rome

Marcelletti, Carlo

JAPAN

Kanazawa

Iwa, Takashi

Kitakyushu

Miyamoto, Alfonso T

SCOTLAND

Edinburgh

Sendai	Logan, Andrew
Mohri, Hitoshi	Glasgow
Tokyo	Wheatley, David J
Imai, Yasuharu	SPAIN
Wada, Juro J	Madrid
MONACO	Rivera, Ramiro
Monte Carlo	SWEDEN
Dor, Vincent	Sollentuna
NEW ZEALAND	Bjork, Viking O
Auckland	SWITZERLAND
Barratt-Boyes, Sir Brian	Arzier
P.R. OF CHINA	Hahn, Charles J
Beijing	Fully
Ying-Kai, Wu	Naef, Andreas P
PORTUGAL	Zurich
Lisbon	Senning, Prof Ake
Macedo, Manuel E M	Turina, Marko I
ROMANIA	UNITED ARAB EMERIT
Tirgu-Mures	Abu Dhabi
Deac, Radu C	Brom, A Gerard
RUSSIA	VENEZUELA
Moscow	Caracas
Bockeria, Leo A	Tricerri, Fernando E
Burakovsky, Vladimir I	
SAUDI ARABIA	
Riyadh	
Duran, Carlos Gomez	
Merendino, K Alvin	

**THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY
Charter Members
June 7, 1917**

E. Wyllis Andrews	Arthur A. Law
John Auer	William Lerche
Edward R. Baldwin	Howard Lilienthal
Walter M. Boothby	William H. Lockett
William Branower	Morris Manges
Harlow Brooks	Walton Martin
Lawrason Brown	Rudolph Matas
Kenneth Bulkley	E. S. McSweeney
Alexis Carrel	Samuel J. Meltzer
Norman B. Carson	Willy Meyer (Founder)
J. Frank Corbett	James Alexander Miller
Armistead C. Crump	Robert T. Miller
Charles N. Dowd	Fred J. Murphy
Kennon Dunham	Leo S. Peterson
Edmond Melchior Eberts	Eugene H. Pool
Max Einhorn	Walther I. Rathbun
Herman Fischer	Martin Rehling
Albert H. Garvin	B. Merrill Ricketts
Nathan W. Green	Samuel Robinson
John R. Hartwell	Charles I. Scudder
George J. Heuer	William H. Stewart
Chevalier Jackson	Franz Torek
H. H. Janeway	Martin W. Ware
James H. Kenyon	Abraham O. Wilensky
Adrian V. S. Lambert	Sidney Yankauer

BY-LAWS OF
THE AMERICAN ASSOCIATION
FOR THORACIC SURGERY

ARTICLE I. Name

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

ARTICLE II. Purpose

The purposes of the Association shall be:

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

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

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

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

ARTICLE III. Membership

Section 1. There shall be four classes of members: Honorary, Senior, Active and, for a time, Associate. 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 the 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 9 following. There shall be no further additions to the Associate Membership. All new members shall be elected directly to Honorary or Active status.

Section 6. Associate Membership for those members elected after 1960 shall be limited to a five year period. During this limited period, an Associate Member, if properly qualified, may be elected to Active Membership. After the expiration of this limited period an Associate Member, if not yet qualified for Active Membership, must either be re-elected to an additional period of Associate Membership or dropped from the rolls of the Association.

Section 7. 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 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 this 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 8. 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, Associate Members for re-election; members dropped from the rolls of the Association.

Section 9. Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a 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 10. The Council shall recommend that any Active or Associate 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 11. Notwithstanding Section 10, 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 9.
- d. It may not deplete the principal of the Endowment Fund.

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 a 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 IX, give status as officers of the Association to the individual members of any *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 and shall also serve as custodian of the Endowment Fund.

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 six members: the President, the Vice President, the Secretary and at least six members-at-large, two 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 two 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 years. 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 the 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 Committee on Manpower 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 make its recommendations to the Council to assist in meeting the educational mission of the Association.

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 for current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact, and to be known as the Endowment Fund. The Council is responsible for the proper management of the Endowment Fund, and may divert any surplus in the current funds of the Association into this fund, but may not withdraw any of the principal of the Endowment Fund except in accordance with the provisions of Section 6, following.

Section 5. The income from the Endowment Fund shall be expended as the Council directs.

Section 6. The principal of the Endowment Fund may be withdrawn, in whole or in part, under the following conditions only: The amount of principal to be withdrawn shall have been approved by the Council; it shall have been approved by a majority of the members present and voting at a regularly convened annual meeting; it shall have been tabled for one year; it shall have been finally passed by a three-fourths vote of the members present and voting at the next regularly convened annual meeting.

Section 7. In the event of the dissolution of the Association, the Endowment Fund shall be distributed among national institutions of the United States and Canada in a proportion equal to the then existing ratio between the numbers of citizens of the two nations who are members of the Association.

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 these 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 and Associate Members.

Section 5. Each annual meeting shall have at least two executive sessions.

Section 6. When the Association convenes for its annual meeting, it shall immediately go into the first executive session, but the business at this session shall be limited to:

1. Appointment of necessary committees.
2. Miscellaneous business of an urgent nature.

Section 7. The second executive session of the Association shall be held during the afternoon of the second day of the meeting. The business at this session shall include, but is not limited to:

1. Reading or waiver of reading of the minutes of the preceding meetings of the Association and the Council.
2. Report of the Treasurer for 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 or these By-Laws, all questions at a meeting of the members shall be decided by a majority vote of the members present in person and voting. Voting by proxy is not permitted.

Section 9. Fifty voting members present in person shall constitute a quorum at a meeting of members.

Section 10. While the scientific session of the annual meeting is held primarily for the benefit of the members of the Association, it may be open to non-members who are able to submit satisfactory credentials, who register in a specified manner, and who pay such registration fee as may be determined and published by the Council from year to year.

Section 11. There shall be an annual meeting of the Council held during the annual meeting of the Association. Additional meetings of the Council may be called on not less than seven days' prior written or telephonic notice by the President, the Secretary or any three members of the Council.

Section 12. Five members of the Council shall constitute a quorum for the conduct of business at any meeting of the Council, but a smaller number may adjourn any such meeting.

Section 13. Whenever any notice is required to be given to any member of the Council, a waiver thereof in writing, signed by the member of the Council entitled to such notice, whether before or after the time stated therein, shall be deemed equivalent thereto.

Section 14. Any action which may be or is required to be taken at a meeting of the Council may be taken without a meeting if a consent in writing, setting forth the action so taken, shall be signed by all of the members of the Council. Any such consent shall have the same force and effect as a unanimous vote at a duly called and constituted meeting.

ARTICLE IX. Indemnification and Directors and Officers

Section 1. The Association shall indemnify any and all of its Councilors (hereinafter in this Article referred to as "directors") or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against expenses actually and necessarily incurred by them in connection with the defense or settlement of any action, suit or proceeding in which they, or any of them, are made parties, or a party, by reason of being or having been directors or officers or a director or officer of the Association, or of such other corporation or association, provided, however, that the foregoing shall not apply to

matters as to which any such director or officer or former director or officer or person shall be adjudged in such action, suit or proceeding to be liable for willful misconduct in the performance of duty or to such matters as shall be settled by agreement predicated on the existence of such liability.

Section 2. Upon specific authorization by the Council, the Association may purchase and maintain insurance on behalf of any and all of its directors or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against any liability, or settlement based on asserted liability, incurred by them by reason of being or having been directors or officers or 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 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 \$150.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Annual dues for Associate Members shall be \$150.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 4. Senior Members are exempt from dues.

Section 5. The initiation fee for those elected directly to Active Membership shall be \$15.00.

Section 6. If and when an Associate Member is elected to Active Membership, he shall pay an additional \$5.00 initiation fee.

Section 7. Associate and 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, Tuesday, April 28, 1992

Meetings of the American Association for Thoracic Surgery

1918-Chicago.....	President, Samuel J. Meltzer
1919-Atlantic City.....	President, Willy Meyer
1920-New Orleans.....	President, Willy Meyer
1921-Boston.....	President, Rudolph Matas
1922-Washington.....	President, Samuel Robinson
1923-Chicago.....	President, Howard Lilienthal
1924-Rochester, Minn.....	President, Carl A. Hedblom
1925-Washington.....	President, Nathan W. Green
1926-Montreal.....	President, Edward W. Archibald
1927-New York.....	President, Franz Torek
1928-Washington.....	President, Evarts A. Graham
1929-St. Louis.....	President, John L. Yates
1930-Philadelphia.....	President, Wyman Whittemore
1931-San Francisco.....	President, Ethan Flagg Butler
1932-Ann Arbor.....	President, Frederick T. Lord
1933-Washington.....	President, George P. Muller
1934-Boston.....	President, George J. Heuer
1935-New York.....	President, John Alexander
1936-Rochester, Minn.....	President, Carl Eggers
1937-Saranac Lake.....	President, Leo Eloesser
1938-Atlanta.....	President, Stuart W. Harrington
1939-Los Angeles.....	President, Harold Brunn
1940-Cleveland.....	President, Adrian V. S. Lambert
1941-Toronto.....	President, Fraser B. Gurd
1944-Chicago.....	President, Frank S. Dolley
1946-Detroit.....	President, Claude S. Beck
1947-St. Louis.....	President, I. A. Bigger
1948-Quebec.....	President, Alton Ochsner
1949-New Orleans.....	President, Edward D. Churchill
1950-Denver.....	President, Edward J. O'Brien

1951-Atlantic City..... President, Alfred Blalock
 1952-Dallas..... President, Frank B. Berry
 1953-San Francisco..... President, Robert M. Janes
 1954-Montreal..... President, Emile Holman
 1955-Atlantic City..... President, Edward S. Welles
 1956-Miami Beach..... President, Richard H. Meade
 1957-Chicago..... President, Cameron Haight
 1958-Boston..... President, Brian Blades
 1959-Los Angeles..... President, Michael E. De Bakey
 1960-Miami Beach..... President, William E. Adams
 1961-Philadelphia..... President, John H. Gibbon, Jr.
 1962-St. Louis..... President, Richard H. Sweet (Deceased 1-11-62)
 President, O. Theron Clagett
 1963-Houston..... President, Julian Johnson
 1964-Montreal..... President, Robert E. Gross
 1965-New Orleans..... President, John C. Jones
 1966-Vancouver, B. C..... President, Herbert C. Maier
 1967-New York..... President, Frederick G. Kergin
 1968-Pittsburgh..... President, Paul C. Samson
 1969-San Francisco..... President, Edward M. Kent
 1970-Washington, D. C..... President, Hiram T. Langston
 1971-Atlanta..... President, Thomas H. Burford
 1974-Las Vegas..... President, Lyman A. Brewer, III
 1975-New York..... President, Wilfred G. Bigelow
 1976-Los Angeles..... President, David J. Dugan
 1977-Toronto..... President, Henry T. Bahnson
 1978-New Orleans..... President, J. Gordon Scannell
 1979-Boston..... President, John W. Kirklin
 1980-San Francisco..... President, Herbert Sloan
 1981-Washington, D.C..... President, Donald L. Paulson
 1982-Phoenix, Arizona..... President, Thomas B. Ferguson

1983-Atlanta..... President, Frank C. Spencer
 1984-New York..... President, Dwight C. McGoon
 1985-New Orleans..... President, David C. Sabiston
 1986-New York..... President, James, R. Malm
 1987-Chicago..... President, Norman E. Shumway
 1988-Los Angeles..... President, Paul A. Ebert
 1989-Boston..... President, W. Gerald Austen
 1990-Toronto..... President, F. Griffith Pearson
 1991-Washington, D.C..... President, Keith Reemtsma
 1992-Los Angeles..... President, John A. Waldhausen

AWARDS

GRAHAM EDUCATION AND RESEARCH FOUNDATION

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

President Martin F. McKneally, M.D., Toronto, Ontario

Vice President William A. Gay, Jr., M.D., New York, New York

Secretary-Treasurer William T. Maloney, Manchester, Massachusetts

Director Robert A. Guyton, M.D., Atlanta, Georgia

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 North America and 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 the potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 40 young surgeons from 21 countries have completed their training at thoracic surgical centers.

1st 1951-52 L L Whytehead M D F R C S

790 Sherbrooke St., Winnipeg, Manitoba, R3A 1M3
 CANADA

2nd 1953-54 W. B. Ferguson, M.B., F.R.C.S.

		Royal Victoria Infirmary, Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley, M.Chir., F.R.C.S. St. Mary's Hospital, London, W.2, ENGLAND
4th	1955-56	Raymond L. Hurt, F.R.C.S. The White House, 8 Loom Lane, Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth, F.R.C.S. Brompton Hospital, London, S.W. 3, ENGLAND
6th	1957-58	Peter L. Brunnen, F.R.C.S. Department of Thoracic Surgery, Woodend General Hospital Aberdeen, SCOTLAND
7th	1958-59	N. G. Meyne, M.D. University of Amsterdam, Wilhelmina-Gasthuis, Amsterdam, HOLLAND
8th	1960-61	Godrej S. Karai, M.D. Calcutta, INDIA
9th	1961-62	Fritz Helmer, M.D. Second Surgical Clinic, University of Vienna, Vienna, AUSTRIA
10th	1962-63	Theodor M. Scheinin, M.D. Tammisalonitie 20, Helsinki, 00830, Finland
11th	1963-64	Masahiro Saigusa, M.D. National Nakano Chest Hospital, 3-14-20 Egata, Nakano- Ku, Tokyo 165, JAPAN
	1963-64	Adar J. Hallen, M.D. Department of Thoracic Surgery, University Hospital
12th		Uppsala, SWEDEN
13th	1964-65	Stuart C. Lennox, M.D. 18 Alexander Sq., 5W3 2AX, London, ENGLAND
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S. Univ. Hosp. Surgical Clinic, Aristote Univ. of Thessoliniki, Thessaloniki, GREECE

15th	1965-66	Gerhard Friehs, M.D. Chirurgische University Klinik, Graz A-8036, AUSTRIA
16th	1965-66	Ary Blesovsky, M.D. London, ENGLAND
17th	1966-67	C. Peter Clarke, F.R.A.C.S. Ste. #4, 6th Floor, 55 Victoria Parade, Fitzroy 3065 AUSTRALIA
18th	1966-67	G. B. Parulkar, M.D. Wookhardt Heart Inst., Poonam Chambers B, A A Road, Bombay 400 018, INDIA
19th	1967-68	Claus Jessen, M.D. Surg. Dept. D, Rigshospitalet, Blegdamsvej 9, Copenhagen, DENMARK
20th	1969-70	Peter Bruecke, M.D. AM Steinbruch, 29 Linz-Puchenu, A-4040, AUSTRIA
21st	1970-71	Michel S. Slim, M.D. New York Medical College, Division of Pediatric Surgery New York, New York 10595 USA
22nd	1971-72	Severi Pellervo Mattila, M.D. Forsellesintie 5.7. D, Kauniainen, 02700, FINLAND
23rd	1972-73	Yasuyuki Fujiwara, M.D. Department of Cardiovascular Surgery, Tokyo Medical College Hospital, Shinjuku, Tokyo, JAPAN
24th	1973-74	Marc Roger de Leval, M.D. Hosp. for Sick Children, Great Ormond St., Longon, WC1N 3JH, ENGLAND
25th	1974-75	J. J. DeWet Lubbe, M.D. 1406 City Park Medical Center, 181 Longmarket St., Cape Town 8001, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner, M.D. Institute of Surgery, 80-211 U1, Deinsky 7, Gdansk, POLAND
27th	1976-77	Bum Koo Cho, M.D. Yonsei University, P.O. Box 71

Severance Hospital, Seoul, KOREA

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|------|---------|---|
| 28th | 1977-78 | Alan William Gale, M.D., FRACP, FRACS
171 Sutherland, Paddington 2021
Sydney, AUSTRALIA |
| 29th | 1978-79 | Eduardo Otero Coto, M.D.
Servicio de Cirugia Cardiovascular, Ciudad Sanitaria "Le Fe"
Valencia, SPAIN |
| 30th | 1980-81 | Richard K. Firmin, M.D.
"Moss Grove", 5 Knighton Grange Road, Stoneygate,
Leicester LE2 2LF, ENGLAND |
| 31st | 1981-82 | Claudio A. Salles, M.D.
Av Celso Porfirio Machado, 370, Bairro Belvedere
Belo Horizonte MG, BRAZIL |
| 32nd | 1982-83 | Yasuhisa Shimazaki, M.D.
First Dept. of Surgery, Osaka Univ. Medical School
Fukushima-ku, Osaka 553, JAPAN |
| 33rd | 1983-84 | Georg S. Kobinia, M.D.
LKH Klagenfurt, St. Veider Strasse 47, Dept. of Cardiac
Surgery, Klagenfurt, A-9026, AUSTRIA |
| 34th | 1984-85 | Aram Smolinsky, M.D.
Department of Cardiac Surgery, The Sheba Medical
Center
Tel Hashomer, 52621, ISRAEL |
| 35th | 1985-86 | Florentine J. Vargas, M.D.
San Martin 1353, Buenos Aires, ARGENTINA |
| 36th | 1986-87 | Ari L. J. Harjula, M.D.
Helsinki Univ. Hosp. Surgery, Haartmanninkatu, Helsinki,
00290 FINLAND |
| 37th | 1987-88 | Byung-Chul Chang, M.D.
Dept. of Thoracic and Cardiovascular Surgery, Yonsei
University College of Medicine, CPO Box 8044, Seoul,
Korea |

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|------|---------|--|
| 38th | 1988-89 | <p>Wang Cheng, M.D.</p> <p>Department of Cardiac Surgry, Beijing Heart, Lung, Blood Vessel Medical Center & Anzhen Hospital, Andingmenwai, Beijing, PEOPLE'S REPUBLIC OF CHINA</p> |
| 39th | 1989-90 | <p>Christopher John Knott-Craig, M.D.</p> <p>Univ. of Oklahoma, Thoracic and Cardiovascular Surgery, P.O. Box 26901, Oklahoma City, OK 73190</p> |
| 40th | 1991-92 | <p>Ko Bando, M.D., Ph.D.</p> <p>Univ. of Pittsburgh, 1084 Scaife Hall, Pittsburgh, PA 15261</p> |
| 41st | 1992-93 | <p>Timothy E. Oaks, M.D.</p> <p>Department of Surgery, The Milton S. Hershey Medical Center Room #6314, Box 850, Hershey, Pennsylvania 17033</p> |
| 42nd | 1993-94 | <p>Alain E. Serraf, M.D.</p> <p>Hopital Marie - Lannelongue, Universite Paris Sud, 133, avenue de la Resistance, 92350 Le Plessis Robinson</p> |

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 an opportunity for research, training and experience for North American surgeons committed to pursuing an academic career in cardiothoracic surgery. Administered by the Graham Education and Research Foundation, the program is undertaken within the first three years after completion of an approved cardiothoracic residency and is about two years in duration.

EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

1986-1988 Mark K. Ferguson, M.D.
University of Chicago, Department of Surgery, Box 255
5841 South Maryland Avenue, Chicago, Illinois 60637

ALFRED BLALOCK RESEARCH SCHOLARSHIP

"Efficacy and Toxicity of a New Blood Substitute: Polymerized, Ultra-Pure, Stroma-Free Bovine Hemoglobin"

1988-1990 Gus J. Vlahakes, M.D.

Massachusetts General Hospital and Harvard Medical School

Department of Surgery, Boston, Massachusetts 02114

JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

"Load-Independent Assessment of Cardiac Performance by Noninvasive Means"

1990-1992 Donald D. Glower, M.D.

Duke University Medical Center, Box 31064

Durham, North Carolina

ALTON OCHSNER RESEARCH SCHOLARSHIP

"Experimental Cardiac Graft Arteriosclerosis: Pathogenesis and Prevention of Lesion Development"

1992-1994 David H. Adams, M.D.

Department of Surgery

Brigham and Women's Hospital

75 Francis Street

Boston, Massachusetts 02115