1995 ANNUAL MEETING PROGRAM

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DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

Back to Annual Meeting Program

AATS Postgraduate Course **Congenital**	*Objectives* The 1995 Postgraduate Course on Congenital Heart Disease will address the following topics: Catheter Intervention or Surgery, Myocardial Protection of the Immature Heart, and Fetal and Premature Surgery/Pediatric Cardiac Transplantation. This course will provide attendees with the opportunity to interact with recognized

Heart **Disease** **Sunday, April 23,1995** **8:00 a.m.-4:30 p.m.** **Independence Ballroom** **Sheraton Boston** **Hotel and Towers** **Boston, MA**	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 topic areas, with ample time provided during each session for discussion of specific questions from the audience. **Registration** Enrollment in this course will be by pre-registration until March 27, 1995. The registration fee is $50 per person and includes the PG Course, coffee break, and lunch. *Accreditation* The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing medical education activity for 6 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

Postgraduate Course on Congenital Heart Disease

Independence Ballroom, Sheraton Boston Hotel

7:00-8:00 a.m. REGISTRATION/CONTINENTAL BREAKFAST

Session I CATHETER INTERVENTION OR SURGERY

Moderators: Richard A. Jonas, M.D.

James E. Lock, M.D.

8:00 a.m. **PATENT DUCTUS ARTERIOSUS**

Devices and Coils

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

Video Assisted Thoracoscopic Surgery

Redmond P. Burke, M.D., Boston, Massachusetts

Thoracotomy for PDA: Ligation and Division

Constantine Mavroudis, M.D., Chicago, Illinois

Panel Discussion

8:40 a.m. **AORTIC VALVE STENOSIS**

Balloon Dilation

James E. Lock, M.D., Boston, Massachusetts

Surgical Valvotomy

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

Panel Discussion

9:10 a.m. **COARCTATION**

Balloons and Stents

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

Surgery

Jan Quaegebeur, M.D., New York, New York

Panel Discussion

9:40 a.m. **ASD and VSD**

Devices

James E. Lock, M.D., Boston, Massachusetts

Surgery

Carl Backer, M.D., Chicago, Illinois

Panel Discussion

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

Session II MYOCARDIAL PROTECTION OF THE IMMATURE HEART

Moderator: Pedro del Nido, M.D.

10:45 a.m. **Differences Between Mature and Immature Myocardium**

Pedro del Nido, M.D., Boston, Massachusetts

11:05 a.m. **Calcium and Immature Myocardium**

Ivan M. Rebeyka, M.D., Toronto, Ontario, Canada

11:25 a.m. **Blood Cardioplegia and Substrate Enhancement**

David C. Drinkwater, Jr., M.D., Los Angeles, California

11:45 a.m**. The Role of Vascular Events in Hypothermic Ischemia/Reperfusion**

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

12:15-1:30 p.m. **LUNCHEON**

Session III FETAL AND PREMATURE SURGERY/PEDIATRIC CARDIAC TRANSPLANTATION

Moderator: John E. Mayer, M.D.

1:30 p.m. **Cardiac Surgery in the Fetus and Premature Neonate**

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

1:55 p.m. **Cardiac Surgery in the Low Birth Weight Neonate**

Roger B.B. Mee, M.B., Ch.B., FRACS, Cleveland, Ohio

2:20 p.m. **Immunosupression Regimens for Pediatric Cardiac Transplantation**

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

2:45 p.m. **Coronary Atherosclerosis and Pediatric Cardiac Transplantation**

Leonard L. Bailey, M.D., Loma Linda, California

3:10 p.m. **Neoplasm after Pediatric Cardiac Transplantation**

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

3:35-4:00 p.m. **Panel Discussion and Summary Remarks**

5:00-7:00 p.m. **RECEPTION**

Hynes Convention Center

1995 Symposium on Transplantation **Immunotherapy for Thoracic**	*Objective* The 1995 Symposium on Transplantation entitled "Immunotherapy for Thoracic Transplantation" will address various issues related to thoracic transplantation including early and late results of cardiac and pulmonary transplantation, Cyclosporin and FK-506 based immunosuppression, total lymphoid irradiation, phototherapy, Mycophenolic acid, rapamycin and methotrexate for immunosuppression, chimerism and tolerance, and prophylaxis for infection following thoracic transplantation. This symposium will provide attendees with the opportunity to interact with recognized experts involved in the research and

Transplantation **Sunday, April 23,1995** **8:00 a.m.-4:30 p.m.** **Republic Ballroom** **Sheraton Boston** **Hotel and Towers** **Boston, Massachusetts**	development of new techniques and procedures in transplantation. The format of the course will include lectures on current issues within each of the topic areas, with ample time provided during each session for discussion of specific questions from the audience. *Registration* Enrollment in this Symposium will be by pre-registration until March 27,1995. After March 25, 1994, participants may register on site at the Sheraton Boston Hotel and Towers. The registration fee is $50.00 per person and includes the symposium, coffee breaks, and lunch. *Accreditation* The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing medical education activity for 6 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

Immunotherapy for Thoracic Transplantation SymposiumRepublic Ballroom, Sheraton Boston Hotel

7:00-8:00 a.m. **REGISTRATION/CONTINENTAL BREAKFAST**

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

Robert L Hordesty, M.D.

Pittsburgh, Pennsylvania

8:30 a.m. **Early and Late Results of Cardiac Transplantation**

Bruce A. Reitz, M.D.

Stanford, California

9:00 a.m. **Early and Late Results of Pulmonary Transplantation**

Alec Patterson, M.D.

St. Louis, Missouri

9:30 a.m. **Cyclosporin-Based Immunosuppression**

Alan H. Menkis, M.D.

London, Ontario, Canada

10:00 a.m. **FK-506-Based Immunosuppression**

Robert J. Keenan, M.D.

Pittsburgh, Pennsylvania

10:30-11:00 a.m. **REFRESHMENT BREAK**

11:00 a.m. **Total Lymphoid Irradiation**

Daniel Bernstein, M.D.
Palo Alto, California

11:30 a.m. **Phototherapy for Immunosuppression**

Robert E. Michler, M.D.

New York, New York

12:00-1:30 p.m. **LUNCHEON**

1:30 p.m. **Chimerism and Tolerance**

Yolonda L. Colson, M.D., Ph.D

Pittsburgh, Pennsylvania

2:00 p.m. **RS-164443 (Mycophenolic Acid) for Immunosuppression**

James K. Kirklin, M.D.

Birmingham, Alabama

2:30-3:00 p.m. **REFRESHMENT BREAK**

3:00 p.m. **Rapamycin for Immunosuppression**

Randall E. Morris, M.D.
Stanford, California

3:30 p.m. **Methotrexate for Immunosuppression**

R. Morton Bolman, III, M.D.

Minneapolis, Minnesota

4:00 p.m. **Prophylaxis for Infection following Thoracic Transplantation**

Penny Williams, M.D.

Pittsburgh, Pennsylvania

5:00-7:00 p.m. **RECEPTION**

Hynes Convention Center

MONDAY MORNING, APRIL 24, 1995

Back to Annual Meeting Program

AMERICAN ASSOCIATION FOR

THORACIC SURGERY

75TH ANNUAL MEETING
HYNES CONVENTION CENTER and
SHERATON BOSTON HOTEL and TOWERS

APRIL 23-26, 1995

MONDAY MORNING, APRIL 24, 1995

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

8:45 a.m. PLENARY SESSION

Auditorium, Hynes Convention Center
Moderators: Robert B. Wallace, M.D.

James L. Cox, M.D.

1. LONG-TERM SURVIVAL BENEFIT OF CABG AND PTCA IN PATIENTS WITH CORONARY ARTERY DISEASE

Robert H. Jones, M.D., Karen Kesler, M.S.*, Harry R. Phillips, III, M.D.*, Daniel B. Mark, M.D.*, Peter K. Smith, M.D.*, Charlotte L. Nelson, M.S.*, Mark F. Newman, M.D.*, Joseph G. Reves, M.D.*, Robert W. Anderson, M.D and Robert M. Califf, M.D.*

Durham, North Carolina

The purpose of this study was to evaluate long-term survival benefit of CABG and PTCA in 9263 patients with symptomatic coronary artery disease confirmed to involve one, two, or three vessels by cardiac catheterization at a single academic medical center between 1984 and 1990. Clinical data was pro-spectively entered into an established cardiovascular database, and annual follow-up was 97% complete for a minimum and mean interval of 2.5 and 5.4 years. Outcomes were analyzed by intention to treat by medicine (n = 3038), PTCA (n = 2799) or CABG (n = 3426). Differences among treatment groups in nine baseline characteristics known to influence outcome (ejection fraction, age, gender, acute myocardial infarction, comorbid disease, congestive heart failure, chest pain, peripheral vascular disease, mitral regurgitation) were adjusted by statistical models previously shown to be valfd. A severity of coronary artery stenosis variable, defined by the number of 75% stenoses, presence of any 95% stenoses, and LAD and proximal LAD location of stenosis, best defined survival benefit from CABG and PTCA compared to medical treatment. Adjusted hazard ratios for the three treatment pairs are illustrated for nine discrete anatomic groups comprising this anatomic severity index.

Trends apparent in our previous report of intermediate-term outcomes in this patient group are more definitive and statistically valid in these long-term outcome data. These data justify the following generalizations regarding selection of treatment in patients with symptomatic coronary artery disease: 1) either PTCA or CABG confers long-term survival benefit over medical treatment in all patients; 2) all patients with single-vessel disease, except those with >95% proximal LAD stenosis, benefit from PTCA in comparison to CABG; 3) all three-vessel patients and two-vessel patients with severe LAD stenosis benefit from CABG in comparison to PTCA; 4) other two-vessel patients and one-vessel patients with >95% proximal LAD stenosis can be treated with either PTCA or CABG; 5) these relative benefit ratios must be interpreted in light of the absolute rate of death in each anatomic subgroup which is least in patients with single-vessel disease and greatest in patients with severe three-vessel disease.

*By invitation

2. COMPARISON OF EARLY FUNCTIONAL RESULTS AFTER VOLUME REDUCTION OR LUNG TRANSPLANTATION FOR COPD

Henning Gaissert, M.D.*, Elbert P. Trulock, M.D.*, Sudhir Sundaresan, M.D.*, Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.

St. Louis, Missouri

Bilateral lung volume reduction is designed to improve pulmonary function by restoration of diaphragmatic and chest wall mechanics. For selected patients with end stage emphysema this procedure may offer a treatment alternative to lung transplantation. Functional performance and survival in the first six months after volume reduction (VR) were compared to single (SLT) and bilateral lung transplantation (BLT) in a group of patients with emphysema due to chronic obstructive pulmonary disease. Following evaluation, patients were enrolled in a supervised intensive pre- and postoperative program of pulmonary rehabilitation. Supplemental oxygen was administered as needed to keep Sa02 greater than 90%. Functional assessment including PFT, room air ABG, and six minute walk test was obtained before operation (Pre) and at three and six months postop. Twenty-three patients underwent VR (mean age 55 years), 40 patients SLT (55 years), and 27 patients BLT (49 years).

Early mortality was 0, 0 and 2/27 in the VR, SLT and BLT groupsm respectively. Six month mortality was 0, 3/40 and 2/27 in the VR, SLT and BLT groups, respectively.

	VR		SLT		BLT	
Time	$FEV_{1,}$L(%)	room air pO_2	FEVi.L(%)	room air pO_2	FEV_1,L(%)	room air pO_2
Pre	076 ± 033	65 8 ± 8 0	047 ± 0 13	52.5 ± 9.4	0.51 ± 0 18	552 ± 85
	(25 ± 8)		(18 ± 4)		(17 ± 9)	
3 mo	1.21 ± 060	71 6 ± 12	1 51 ± 0.50	80 6 ± 9 8	2 66 ± 0.82	88. 7 ± 15
	(39 ± 16)		(56 ± 13)		(83 ± 14)	
6 mo	1.36 ± 0.70	75 8 ± 10	1 57 ± 0.45	80 9 ± 9 1	2.9 ± 0 74	90 2 ± 8 5
	(43 ± 17)		(58 ± 12)		(90 ± 12)	
Data are presented as mean ± standard deviation, Values in parentheses are percent of predicted normal						

At six months FEV, was improved by 79% (VR), 234% (SLT) and 469% (BLT) over preoperative values. Exercise endurance as measured by six minute walk test increased by 22% (VR), 47% (SLT) and 84% (BLT) and room air pC>2 increased by 15, 54, and 63% from baseline, respectively. At six months all SLT and BLT patients and 21 of 23 VR patients were free of supplemental oxygen. Although single and bilateral lung transplantation result in superior pulmonary function, volume reduction achieves satisfactory improvement of disabling symptoms early after operation while avoiding immunosuppression and transplant-specific complications.
*By invitation

3. VIDEO THORACOSCOPIC SURGICAL INTERRUPTION: THE TECHNIQUE OF CHOICE FOR PATENT DUCTUS ARTERIOSUS. ROUTINE EXPERIENCE IN 201 PEDIATRIC CASES

Francois Laborde, M.D.*, Thierry A. Folliguet, M.D.*, Alain Batisse, M.D.*, Alain Dibie, M.D.*, Edouardo Da-Cruz, M.D.* and Daniel Carbognani, M.D.*

Paris, France

Sponsored by: J. N. Cunningham, M.D., Brooklyn, New York

Video Thoracoscopic Surgical Interruption (VTSI) for patent ductus arteriosus (PDA) is a well-standardized procedure, already described. We present our entire series of VTSI from the first case performed on 9/5/91 to 10/31/94.

201 patients underwent VTSI in a variety of age groups: < 6 months (53 pts, 26.4%), 6-48 months (116 pts, 57.7%), > 48 months (32 pts, 16%). The mean weight was 12.6 Kg (1.3 Kg - 65 Kg). 35 patients exhibited asymptomatic pulmonary hypertension, while the remainder were asymptomatic. Associated intracardiac anomalies included ASD (3), VSD (5) and APVR (1).

All patients underwent VTSI of their PDA using two titanium clips. Assessment of closure was evaluated by post-operative echocardiogram prior to extubation. 2.5% (5 pts) had a patent ductus following VTSI, all occurring in our early experience and related to insufficient dissection resulting in inadequate clip placement. Four pts had immediate clip repositioning (3 by VTSI, 1 by thoracotomy). Subsequent echocardiogram revealed persistent closure in

these patients. A persistent PDA with minimal flow was discovered in one asymptomatic patient following discharge. Recurrent laryngeal nerve dysfunction was noted in 5 pts (4 transient, 1 persistent). There was no mortality, hemorrhage, transfusion requirement or chylothorax in this series. Mean operative time was 20 mn ± 10 mn, and hospital stay averaged 48 hr > 6 mos, 72 hr < 6 mos.

In conclusion, this is a safe, rapid, cost effective technique resulting in excellent results and a shortened hospital stay. VTSI represents the technique of choice for PDA closure.

9:45 am INTERMISSION - VISIT EXHIBITS

*By invitation

10:30 am PLENARY SESSION

Auditorium, Hynes Convention Center
Moderators: Mortimer J. Buckley, M.D.

James L. Cox, M.D.

4. TUMOURS OF THE ESOPHAGOGASTRIC JUNCTION (EGJ): LONG-TERM SURVIVAL IN FUNCTION OF LYMPH NODE METASTASIS PATTERNS. REFLECTION ON TNM CLASSIFICATION

Toni E. Lerut, M.D., Willem H. Steup, M.D.*, Paul De Leyn, M.D.*, Dirk van Raemdonck, M.D.* and Willy Coosemans, M.D.*

Leuven, Belgium

Introduction: EGJ tumours still give reason for debate as they may be considered as a separate entity with a specific behavior, not compatible to pure esophageal or gastric tumours and because of their poor survival after surgical resection.

Material and methods: From 1983 to 1993, 259 patients with EG-junction tumours, i.e. tumours with their centre located at the Z-line, were operated. A retrospective study was made in the group of patients from 1983 to 1989 (n=95), in order to have a minimum of follow-up of 5 years and a maximum follow-up of 10 years. Results were analysed in function of lymph node metastasis pattern.

Results: Hospital mortality rate was 6.2% (6/95) overall. Actuarial survival analysis showed a 5- and 10-year survival rate of 33% and 31%, respectively. Five- (and ten-) year survival of TNM stages I (n=13), II (n=13), III (n=28), and IV (n=40) was 90% (90%), 70% (70%), 28% (28%) and 11% (8%), respectively. For node negative patients (n=26), five- (and ten-) year survival was 72% (72%), compared to 18% (16%) for node positive patients (n=68; p<0005). If lymph node metastases were both abdominal and thoracic (n=28), five- (and ten-) year survival was 13% compared to 26% (26%) if metastases were only confined to the abdomen (n=37; p=>005). When tumours were staged as an esophageal carcinoma, individual patient staging changed, as did the five- and ten-year survival rates. Figures were as follows: Stages I (n=13), II (n=13), III (n=27) and IV (n=41) with 90% (90%), 70% (70%), 37% (37%) and 10% (10%) respectively for five- and ten-years survival. When stage IV organ metastases were excluded, 5- (and 10-) year survival were 16% (11%) and 15% (9%) for patients staged as gastric (n=28) and esophageal (n=28) carcinoma, respectively.

Conclusions: Tumours of the EGJ tend to have a pattern of lymph node metastasis to both abdominal and thoracic cavity. A reasonable 5- and 10-year survival can be obtained even in patients with lymph node metastasis after wide excision and two field lymph node dissections for stage III and IV. When staging the tumours respectively as gastric or oesophageal carcinoma no difference is seen in survival curves. We suggest to include a N2 labeling for thoracic lymphnode metastasis instead of the actual M+Ly in the actual TNM staging which better reflects the potential for curative surgery in this group. Finally, there was almost no further difference in 10-year survival as compared to 5-year survival.

*By invitation

5. THE ROSS PROCEDURE (PULMONARY AUTOGRAFT) INTERNATIONAL REGISTRY: THE FIRST 18 MONTHS RESULTS. CHANGING DEMOGRAPHICS AND OUTCOMES

James H. Oury, M.D., A. Craig Eddy, M.D.,

S. Kathryn Mackey, R.N.*, Joseph C. Cleveland, M.D.,

William W. Angell, M.D., Ronald C. Elkins, M.D. and

Donald N. Ross

Missoula, Montana

In April 1993 the Ross Procedure Registry was established. Last year we reported the preliminary results of this registry which encompassed the initial results of the procedure on the 657 entered patients. This included both Mr. Ross' series of 417 patients as well as 240 cases done by other surgeons since 1986. Since that time post-operative follow-up data on these patients has been gathered and the number of patients entered in the registry has doubled. A total of 1022 patients have been cataloged in this registry by 95 surgeons from 75 centers around the world. The purpose of this report is to compare the demographics and outcomes of the 773 more recent pulmonary autografts which have been done since 1986 with Mr. Ross' original series of 249 cases and to examine the fate of the autograft and the right ventricular outflow tract (RVOT) in these two groups.

Demographics:

			Implant Technique
	Mean Age	Sex (M/F)	Subcoronary/Inclusion/Root
Ross' Data	29 (9-60 yr)	80% / 20%	100% / 0% / 0%
Post 86 Data	21 (1 day=73 yr)	72% / 28%	10% / 15% / 75%

Mortality Data:

	Early (30) Day	Late	Primary Etiology Early	Primary Etiology Late
Ross' Data	6.6%	7.4%	Hemorrhage/Arrhythmia	Cardiac Failure
Post 86 Data	1.6%	0.5%	Cardiac Insufficiency	Cardiac Arrest

Reoperative Incidence (Cause):

Ross' Data	20%	(RVOT 25% / AI 75%)
Post 86 Data	2.3%	(RVOT 6% / AI 94%)

RVOT Status:

	Acceptable	Revised
Ross' Data	95%	5%
Post 86 Data	99.6%	0.4%

Explant Data:

	Early (30 Day)	Late	Etiology Early	Etiology Late
Ross' Data	0	14%	N/A	AI
Post 86 Data	0	2.3%	N/A	AI

Postoperative NYHA Classification Data:

	Class I	Class II	Class III	Class IV
Ross' Data	No Data	No Data	No Data	No Data
Post 86 Data	94%	4%	1.5%	0.5%

Postoperative Echo Data:

	# of pts c echo data	EF>50%	Mean EF	AI (0 Trace/Mod)	Mean Ao Gradient
Ross' Data	No Data	No Data	No Data	No Data	No Data
Post 86 Data	188/605	95%	66%	88% / 12%	4.5 Torr

Conclusions:

1. Implantation of the pulmonary autograft as a root has clearly evolved to be the technique of choice and has resulted in a marked decrease in explantation of the autograft due to technical failure.

2. With advancing technology RVOT homograft problems have nearly disappeared and no longer play a major role in the failure of the Ross procedure.

3. Both early and late mortality have markedly decreased due to improved myocardial preservation techniques and an improved technical understanding of the procedure.

4. This recent data reconfirms the efficacy of the Ross procedure as it has now evolved as a revolutionary solution to aortic valvular disease.

11:15 am PRESIDENTIAL ADDRESS

Reflections-Projections!

Robert B. Wallace, M.D., Washington, D.C.

12:00 pm ADJOURN FOR LUNCH - IN EXHIBIT HALL

*By invitation

MONDAY AFTERNOON, APRIL 24, 1995

Back to Annual Meeting Program

1:30 pm PLENARY SESSION

Auditorium, Hynes Convention Center

Moderators: Aldo R. Castaneda, M.D.

John A. Waldhausen, M.D.

6. LATE HEMODYNAMIC RESULTS FOLLOWING LEFT VENTRICULAR PATCH REPAIR ASSOCIATED WITH CORONARY GRAFTING IN PATIENTS WITH POST-INFARCTION AKINETIC OR DYSKINETIC LEFT VENTRICULAR ANEURYSM

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

Monte-Carlo, Monaco and Florence, Italy

Aim of the study: To evaluate hemodynamic, clinical and electrophysio-logic data one year after intervention of left ventricular patch repair (LVR) and associated coronary revascularization in patients (pts) with postinfarction akinetic and dyskinetic LV aneurysm.

Patients: Among 580 pts who underwent this type of surgery since 1984, we report the results obtained in 141 pts (132 men and 9 women, mean age 58±8 yrs) who accepted to be controlled 1 year postoperatively with the same protocol. All these pts had right and left heart catheterization, ventricular and coronary angiography, programmed ventricular stimulation (PVS) unless

contraindicated, before, one month and one year after surgery. Site of the aneurysm was anterior in 135 and posterior in 6. Mean delay from first infarction was 36 months (range 1-198). Fifty-three pts had one vessel (V), 55 two V and 33 three V disease. Eleven pts were in NYHA class I, 51 in class II, 54 in class III, and 25 in class IV. Clinical ventricular arrhythmias (VA) were present in 35 pts (25%) and ventricular tachycardia was inducible in 43/94 pts who underwent preop PVS (46%). Indications for surgery were a combination of cardiac insufficiency, angina and VA in the majority of pts. Twenty-one pts were operated in emergency.

Results: All pts had LVR by endoventricular patch plasty with synthetic or autologous patch. Associated procedures included subtotal endocardectomy and cryotherapy in pts with clinical or inducible VT; ventricular septal defect closure in two pts; mitral valve replacement in two and repair in six pts; aortic valve replacement in one pt and repair in another patient. One hundred thirty-five pts (96%) had associated myocardial revascularization (IV in 49 pts, 2 V in 57, 3 V in 27 and 4 V grafts in 2). Internal mammary artery was implanted in 120 pts, almost always on LAD, and was found to be patent after one year in 113 (94%). The mean number of by-pass was 1.89±.86. Mean cardiopulmonary by-pass time was 110±33 min. Twenty-two pts required intraaortic balloon pump preoperatively (10 elective and 11 in emergency). Perioperative complications were cardiac insufficiency requiring inotropic drugs in 20 pts; mechanical assistance in 8 pts; renal failure in 6 pts; bleeding requiring blood transfusion in 11 pts; conduction disturbances requiring temporary pacing in 30 pts.

After 1 year from surgery mean EF significantly increased (from 35±13 to 46±12%, $p<.0001$); end diastolic and end systolic volume index significantly decreased (from 116±43 to 96±29 and from 76±41 to 54±26 ml/m2, respectively $p<.001$). Table reports early and late results on the basis of basal value in EF

	EF<30%	30<ef<40%< span=""></ef<40%<>	EF>40%
Base	22±5	37±2	50±7
Early postop	42±10	49±11	57±8
Late postop	41±11	44±11	52±12

All data are significant at two way variance analysis.

Spontaneous VT were significantly reduced (only two pts had documented clinical VTs during 1 year period); VT was inducible in 8/117 pts who underwent late PVS (7%) p<.001. One out of the 8 pts was not inducible before surgery; 76 pts were in NYHA class I; 50 pts in NYHA II; 11 pts in NYHA III and 4 in NYHA IV. Angina was still present in 8 pts (6%) while 102 pts (72%) complained of angina before surgery.

Conclusions: Our results demonstrate that LV aneurysm patch repair associated with complete coronary revascularization and with subtotal endocardectomy and cryotherapy (when indicated), induces: 1) a long-term significant improvement in LV pump function especially in pts with a low basal EF; 2) a significant improvement in clinical status; and 3) a significant reduction of clinical and inducible ventricular arrhythmias.

*By invitation

7. REDUCTION OF PERIOPERATIVE NEUROLOGICAL MORBIDITY IN CORONARY BYPASS SURGERY: A RANDOMIZED TRIAL OF HIGH VERSUS LOW INTRAOPERATIVE BLOOD PRESSURE MANAGEMENT

Jeffrey P. Gold, M.D, Mary E. Charlson, M..D.*, Karl H. Krieger, M.D., Pamela Williams-Russo, M.D.*, Stephen J. Thomas, M.D.*, Ted P. Szatrowski, M.D., Denise Barbut, M.D.*, Janey Peterson,

R.N.*, Paul Pirraglia, M.S.* and O. Wayne Isom, M.D.

New York, New York

Optimal management of the mean arterial blood pressure (MAP) on cardiopulmonary bypass (CPB) during coronary artery bypass grafting (CABG) remains controversial. Patients (248) undergoing elective multivessel coronary artery bypass grafting were randomized preoperatively to a low (50-60 mmHg) or high (80-100mmHg) MAP group. All patients were evaluated preoperatively with a cardiac, neurologic, cognitive, and functional test battery which was repeated one week and six months postoperatively. Intraoperative transesophageal echocardiography (TEE) studies complemented standard monitoring and management. CPB temperatures and flow were fixed, and MAP was adjusted with a standardized regimen of vasoactive drugs.

The study groups did not differ with respect to age, gender, race, cardiac history, extent of coronary artery disease, LV-EF, as well as all preoperative aspects of the neurological, psychometric, functional, and cognitive test batteries. The mean age was 66 years; 80% were male. Preoperatively the mean LV-EF was 48%, and 53% had a previous MI. There were no difference in the operative mortality (1.6%), crossclamp time (41"), pump time (8"), number of grafts (3.1), or in post-operative bleeding. Postoperative cardiopulmonary events were defined as shock, MI, pulmonary edema, and ARDS. Neurologic events were defined as persistent (>24 hr) deficits correllated with CT scan findings (paresis, plegia, aphasia, central sensory, hemianppsia & cortical blindness). Cognitive deterioration was defined as a clinically important decline on three of twelve neuropsychiatric tests, and a functional deterioration as a five point decline in the SF-36 score.

Patients with MAP maintained within the high pressure range had fewer neurological complications (1.6% vs 6.4%, $p<0.001$) than their low MAP counterparts (independent of preoperative risk factors and TEE assessed degree of aortic ASVD). In patients with advanced aortic ASVD on TEE (16%), the perioperative CVA rate was 30% in the low MAP group, compared to 10% in the high MAP group ($p<0.001$). There were no significant differences in the incidence of long term cognitive (11.4% vs. 12.3%) or functional

(13.7% vs. 10.5%) changes. These findings indicated that patients with mean arterial pressure on CPB maintained at a higher range (presumably closer to their auto-regulatory range) have a substantial reduction in perioperative neurological morbidity following elective CABG when compared to patients with conventional intraoperative pressure management.
*By invitation

8. REPAIR OF LONG SEGMENT TRACHEAL STENOSIS IN INFANCY

Robert D.B. Jaquiss, M.D.*,

Rodney M. Lusk, M.D.*, Thomas L.

Spray, M.D. and Charles B.

Huddleston, M.D.*

St. Louis, Missouri

Congenital long segment stenosis of the trachea (LSTS) presents a considerable surgical challenge because of the difficulty in providing distal ventilation during the repair and because the optimal type of repair is not clearly defined. This report summarizes our experience with an anterior rib cartilage tracheoplasty using cardiopulmonary bypass (CPB) to avoid direct intubation of the distal airway. Six patients underwent repair of LSTS between September 1987 and the present. The median age was 14 weeks (range = 1 to 58 weeks) and the mean weight was 4.8 kg (range = 2.9 to 9.9 kg). All patients had involvement of at least 70% of the tracheal length, typically from the thoracic inlet to the carina with complete cartilaginous rings over much of the stenotic segment. Two patients also had involvement of one or both mainstem bronchi. Associated anomalies were present in two patients (tetralogy of Fallot in one and patent ductus arteriosus in one). In all patients a median sternotomy was employed and the anterior surface of the trachea was exposed from the thoracic inlet to the carina. After

identification of the upper extent of the stenosis by rigid bronchoscopy, a vertical incision was made in the anterior trachea through the entire stenotic segment. A previously harvested segment of rib cartilage, with intact perichondrium, was then fashioned to correspond to the long tracheotomy and sewn in place as an augmentation patch with the perichondrium facing the lumen. In order to avoid distal airway intubation, all procedures were performed on CPB with a mean bypass duration of 110 minutes (range = 54 to 175 minutes). Mechanical ventilation was required postoperatively for a median of 11 days (range = 7 to 81 days), and the median postoperative hospital stay was 17 days (range = 12 to 180 days). There were no operative deaths and all patients are long term survivors. Complications include the need for ECMO support to treat unexplained ventricular dysfunction in one patient and graft dehiscence requiring re-exploration and placement of additional sutures in another. The patient who developed graft dehiscence has required several bronchoscopies for removal of granulation tissue. All other patients are asymptomatic without evidence of airway obstruction and have normal growth and development with a mean follow-up of 4.4 years (range = 1 month to 7.3 years). We conclude that rib cartilage tracheoplasty for LSTS provides excellent results in short and intermediate follow-up. In addition, the use of CPB allows an unobstructed view of the tiny infant airway, and thus permits a precise repair.

*By invitation

9. RECURRENCE OF OBLITERATIVE BRONCHIOLITIS AND DETERMINANTS OF OUTCOME IN 139 PULMONARY RETRANSPLANT RECIPIENTS.

Richard J. Novick M.D., Hans-Joachim Schafers, M.D.*, Larry

W. Stitt, M.Sc.*, Walter Kleptko, M.D.*, Bernard Andr6assian, M.D.*, Jean-Pierre Duchatelle, M.D.*, Robert L. Hardesty, M.D., Adaani E. Frost, M.D.*, and G. Alexander Patterson, M.D.

London, Ontario, Canada, Hannover, Germany, Vienna, Austria, Paris, France, Pittsburgh, Pennsylvania, Houston, Texas and St. Louis, Missouri

An international series of pulmonary retransplantation (retx) was updated in order to identify the determinants of outcome and the prevalence and recurrence rate of obliterative bronchiolitis (OB) postoperatively. The study cohort included 139 patients who underwent retx in 34 institutions in North America and Europe from 1985 to 1994. Eighty patients were retransplanted for OB, 34 for acute graft failure, 13 for intractable airway problems, 8 for acute rejection and 4 for other indications. Survivors were followed for a mean of 706 ± 67 days, with 48 patients alive at 1 year, 30 at 2 years and 16 at 3 years after retx. Follow-up was 100% complete. Actuarial survival was 65 ± 4% at one month, 45 ± 4% at 1 year, 38 ± 5% at 2 years and 36 ± 5% at 3 years; nonetheless, of 90 day postoperative survivors, 65 ± 6% were alive 3 years after retx. Most deaths were caused by infection (48/85, 56%), followed by acute failure of the second graft (22%), recurrent OB (14%), an airway complication (4%) or other causes (4%). Life table and univariate Cox analysis of 18 variables revealed that more recent year of retransplantation ($p = 0.009$), ABO blood group identity ($p = 0.01$), absence of a donor-recipient cytomegalovirus mismatch ($p = 0.04$) and being ambulatory immediately prior to retx ($p = 0.04$) were associated with survival. There was a trend toward improved outcome ($p = 0.5610$) in centers with experience in at least 5 pulmonary retx procedures. On multivariate Cox analysis, being ambulatory

prior to retx was the most significant predictor of survival (p = 0.008).

Complete pulmonary function test results were obtained yearly from every survivor of retx. Bronchiolitis Obliterans Syndrome (BOS) stages were assigned according to standard criteria, based on absolute FEV] values (J Heart Lung Transplant 1993; 12:713-6). The percentage of retx patients in each BOS stage is shown in the table:

Postop Interval	No. of Patients	Stage 0	Stage 1	Stage 2	Stage 3
1 year	48	79%	4%	6%	11%
2 years	30	63%	10%	7%	20%
3 years	16	69%	6%	0%	25%

Absolute FEV] values decreased by 6 ± 4% at 1 year and 18 ± 5% at 2 years from postoperative baseline values (p=0.07, year 2 versus year 1). There were no significant differences in BOS stages or the rate of FEVj decrease between patients reoperated for OB versus other conditions or between single or double lung retx patients. We conclude that survival after pulmonary retx is improving with increasing experience. Optimal results can be obtained in patients who are ambulatory prior to retx and receive an ABO-identical graft. OB does not appear to recur in a more accelerated manner after retx. As long as early mortality due to infection can be minimized, pulmonary retx appears to offer a reasonable option in highly selected patients.

2:50 pm INTERMISSION - VISIT EXHIBITS

*By invitation

3:30 pm PLENARY SESSION

Auditorium, Hynes Convention Center
Moderators: Aldo R. Castaneda, M.D.

John A. Waldhausen, M.D.

10. COMBINED LUNG AND LIVER TRANSPLANTATION IN CYSTIC FIBROSIS PATIENTS. A 4.5 YEAR EXPERIENCE

Jean-Paul A. Couetil, M.D.*, Didier Houssin, M.D.*, Patrick Chevalier, M.D.*, Bertrand Dousset, M.D.*, Didier F. Loulmet, M.D.*, Olivier Soubrane, M.D.*, Romain J. Guillemain, M.D.*, Catherine I. Amrein, M.D.*, Alain Guinvarch, M.D.* and Alain F. Carpentier, M.D., Ph.D.

Paris, France

Cystic fibrosis (CF) patients (pts) with end stage respiratory failure and associated liver cirrhosis have been considered as poor candidates for lung transplantation because of high morbidity and mortality due to hepatic insufficiency following the operation. Since June 1990 our policy has been to combine lung or heart-lung and liver transplantation in this group of pts. Among the 15 pts enrolled in the program, 5 died while awaiting transplantation and 10 underwent one of the following procedures: heart-lung-liver (n=5), En bloc double-lung-liver (n=1), sequential double-lung-liver (n=3), bilateral lobar lung from a split left lung and reduced liver (n=1). The latter was performed because of a large size discrepancy between donor and recipient. All pts were infected with persistent *Pseudomonas,* and 2 had in addition *Aspergillus* species. Pre-operative forced vital capacity and forced expiratory volume/sec were 35 ± 8% and 30 ± 5% of predicted values. All pts were on continuous oxygen and all suffered from severe cirrhosis with portal hypertension. Four had a history of esophageal variceal bleeding and two had had previous portosystemic shunts. Selection of donor organs was based on established

criteria for lung and liver transplantation. The operation was performed as a two-stage procedure, the intra-thoracic stage being completed before commencing the abdominal stage. **Results:** 2 pts died in the post-operative period, one from primary liver failure (at 10 days), the second from pulmonary edema (at 21 days). Other complications included tracheal stenosis (n=1), biliary stenosis (n=3) and severe ascites (n=3). All were successfully treated. Four pts developed established diabetes post-operatively. Obliterative bronchiolitis (OB) developed in 2 pts at one year and 1 pt at two years. Of those, 2 pts were successfully treated with FK506. The other pt who was not treated with FK.506 continued to deteriorate and required heart-lung transplantation at 38 months but eventually died from bleeding. Estimated actuarial survival is 68.6 ± 12% at 3 years which is comparable to survival achieved for lung transplantation alone in CF pts in our unit. **Conclusion:** This series, the largest reported, demonstrates that in CF patients suffering from chronic respiratory failure with advanced cirrhosis, and thus having minimal life expectancy, lung transplantation combined with liver transplantation can be performed with satisfactory outcome.

*By invitation

11. PHASE I TRIAL OF FK506 IN CLINICAL HEART TRANSPLANTATION: IMMEDIATE TERM RESULTS

Si M. Pham, M.D.*, Robert L. Kormos, M.D., Brack G. Hattler, M.D.*, Athabassios C. Tsamandas, M.D.*, Anthony J. Demetris, M.D.*, Frederick J. Fricker, M.D.*, Thomas E. Starzl, M.D.* and Hartley P. Griffith, M.D. *Pittsburgh, Pennsylvania*

FK506, a macrolide antibiotic and a potent T-cell suppressor, recently received FDA approval for treatment of liver recipients. A

phase I trial of FK506 in clinical heart transplantation was initiated at our center in 1989. The present study reports the intermediate term results of this trial.

Between 01/01/89 and 10/01/94, 136 primary heart recipients (age=0-65 years) received FK506/steroids/azathioprine regimen (FK), while 121 others were treated with cyclosporine-based immunosuppression (CBIS) consisting of triple drugs (cyclosporine A, azathioprine, steroids) and lympholytic induction with either rabbit antithymocyte globulin (RATG) or OKT3. The actuarial 1-, 2-, and 4-year survival for the FK and CBIS groups were 86%, 83% and 77%, and 93%, 89% and 73%, respectively (p=NS). Renal toxicity was the major side effect of both drugs. Mean serum creatinine values at 1 year were slightly higher for FK506 recipients (FK=2.2±1.2 vs CBIS=1.8±0.6; p=0.06). This probably reflected our learning curve for the use of FK506. By two years, the difference in creatinine disappeared (FK506=2.2±0.9 vs CBIS=2.1±0.9; p=0.5). There was a higher rate of intractable rejection (refractory to steroid bolus and RATG or OKT3) in the CBIS group ($p<0.05$). Fourteen CBIS patients had intractable rejection; all resolved with FK506 rescue. Two FK patients had intractable rejection requiring total lymphoid irradiation and methotrexate rescue. Endomyocardial specimens of 132 adult patients (FK=52, CBIS=50) were available for histopathological analysis. There was a trend toward a greater incidence of ISHLT Grade 2 or higher rejections (57% vs 40%, p=0.07) during the first 30 days in the FK506 arm. In contrast, there was a trend toward more Quilty lesion (27% vs 15%, $p<0.27$) and endocardia! fibrosis in the one-year biopsies of the CBIS recipients. The intermediate term outcome of cardiac recipients under FK506 compares favorably with those under the cyclosporine-based protocol plus lympholytic induction. FK506 is an effective rescue agent for intractable rejection under conventional immunosuppression.

*By invitation

12. COMPARATIVE STUDY OF CONVENTIONAL AND "TOTAL" (WITH CAVAL ANASTOMOSIS)

ORTHOTOPIC HEART TRANSPLANTATION

Dominique R. Metras, M.D., Bernard Kreitmann, M.D.*, Alberto Riberi, M.D.*, Gilbert Habib, M.D.*, Pierre Ambrosi, M.D.* and Adrienne Pannetier, M.D.*

Marseille, France

Conventional orthotopic heart transplantation (OHT) has been reported to modify size and geometry of both atria and to cause mitral and tricuspid regurgitation affecting ventricular filling. Since 1992 we have systematically performed a modified technique of OHT with extensive resection of the recipient's heart, anastomosis of a small peri-venous cuff of left atrium and of both venae cavae. Our total experience includes 120 OHT, with 70 conventional technique (group I) and 50 modified technique (group II). Both groups were similar in age, pre-transplant characteristics, post-operative management including immunosuppression, and routine antithrombotic therapy (low molecular weight heparin followed by anti-agregant). The recipient aortic cross clamping time (i.e., time of heart excision and heart implantation) was longer in Group II (87 ± 19 mn vs 65 ± 15 mn; $p<0.001$). No haemorrhagic complication or surgical problems (such as caval thrombosis and/or stenosis) imputable to the technique were noted. Peri-operative and subsequent mortality were similar in both groups. Five patients in Group I presented a documented systemic embolism one month to five years after OHT, two of them with neurologic sequelae. No thromboembolic episode occurred in Group II.

Two comparable groups of 25 patients were studied at 6 months post-transplant (Group A after conventional OHT; Group B after modified OHT) with EKG, 24 hours EK.G Holter examination, and trans-thoracic and oesophagial echodoppler. A higher number of EKG anomalies, a significant increase in atrial size, a significantly higher proportion of mitral and tricuspid insufficiencies and a significantly higher coefficient of variation of the E/A ratio (parameter of left ventricular filling) were noted in Group A. Spontaneous echocontrast and atrial thrombi were present in the left atrium of several patients of Group A (50% and 20%, respectively) and never noted

in Group B. We conclude that this modification of OHT lengthening surgery by about 20 mn achieves a closer to normal cardiac anatomy and geometry. Less anomalies of the cardiac function were found, the long term significance of which are yet unknown. A lower number of echocardiographic features of left atrial thrombosis and an absence of systemic embolism prompt us to think there is a definite advantage in this technically simple approach.
*By invitation

13. MEDIASTINAL LYMPH NODE STAGING OF NON-SMALL CELL LUNG CANCER: A PROSPECTIVE COMPARISON OF COMPUTED TOMOGRAPHY AND POSITRON EMISSION TOMOGRAPHY

Walter J. Scott, M.D.*, Lisa S. Gobar, M.D.*, John D. Terry, M.D.*, John J. Sunderland, Ph.D.*, Naresh A, Dewan, M.D.* and Jeffrey T. Sugimoto, M.D.*

Omaha, Nebraska

Sponsored by: Alex G. Little, Las Vegas, Nevada

We prospectively compared the abilities of positron emission tomography (PET) and computed tomography (CT) to detect lymph node metastases (N2 or N3) in patients with non-small cell lung cancer (NSCLC). PET detects increased rates of glucose uptake, characteristic of malignant cells, by measuring the uptake of a positron-emitting glucose analogue administered intravenously.

Patients had known or suspected NSCLC and were candidates for surgical staging. Patients with peripheral, <2 cm tumors and a normal mediastinum were ineligible. All patients underwent CT, PET, and surgical staging. The American Thoracic Society lymph node map was used. CT and PET scans were read by separate radiologists blinded to surgical staging results. Lymph nodes were positive by CT if > 1.0 cm in short-axis diameter. The CT, but not the interpretation, was available to the radiologist reading the PET to help localize areas of uptake. Standardized uptake values (SUVs) were recorded from areas on PET

corresponding to those that were biopsied. Node regions with an SUV >4.2 were called positive.

All patients had NSCLC and all primary tumors were detected by PET. A total of 75 lymph node stations (2.8 per patient) were analyzed in 27 patients. Patients underwent mediastinoscopy (9), thoracotomy (19), thoracoscopy (1), and scalene node biopsy (2). N2/N3 metastases were present in 10/75 stations (13.3%), and 9/27 patients (33.3%). CT incorrectly staged 3 patients as positive and 3 as negative for mediastinal metastases. CT sensitivity and specificity were 67% and 83%, respectively. PET correctly staged the mediastinum in all 27 patients, for a sensitivity and specificity of 100%. When analyzed by individual node station, there were 4 false positives and 4 false negatives by CT (sensitivity = 60%, specificity = 93%, positive predictive value = 60%). PET mislabeled one negative node station as positive (100% sensitive, 98% specific, positive predictive value 91%). The stage of the patient was unchanged. The differences between CT and PET were significant when the data were analyzed using the McNemar test (one-sided) for both individual lymph node stations ($p=0.039$) and for patients ($p=0.031$). We conclude that PET supplemented by CT is more accurate than CT in detecting mediastinal lymph node metastases in patients with non-small cell lung cancer.

*By invitation

14. SURFACE-BOUND HEPARIN FAILS TO REDUCE THROMBIN FORMATION DURING CLINICAL CARDIOPULMONARY BYPASS

Robert C. Gorman, M.D.*, Nicholas P. Ziats, Ph.D.*, Nicolas Gikakis, B.S.*, Ling Sun, Ph.D.*, Mohammed M.H. Khan, M.D., Ph.D.*, Nina Stenach, B.A.*, Suneeti Sapatnekar, M.D.*, Matt L. Robertson*, Stefan Niewiarowski, M.D., Ph.D.*, Robert W. Colman, M.D.*, A. Koneti Rao, M.D.*, James L. Anderson, M.D., Ph.D.* and L. Henry Edmunds, Jr., M.D.

Philadelphia, Pennsylvania

The hypothesis that heparin-coated perfusion circuits reduce thrombin formation, fibrinolysis and platelet, complement and neutrophil activation was tested in 20 consecutive, randomized adults who had cardiopulmonary bypass (CPB). Medtronic, Inc. provided 20 identical perfusion systems, but in 10 all blood contacting surfaces were coated with partially degraded heparin (Carmeda process). All patients received 3 mgm/kg heparin; activated clotting times were maintained over 400 seconds; CPB lasted 36 to 244 min.

Blood samples for platelet count: platelet response to ADP; plasma beta thromboglobulin (BTG); Complement 3b; neutrophil elastase; fibrinopeptide A (FPA); prothrombin fragment, F1.2; thrombin-antithrombin III complex (TAT); and D-dimer were obtained after heparin, 5 and 30 min after starting CPB, within 5 min after CPB and 15 min after protamine. After CPB, tubing segments were analyzed for surface adsorbed antithrombin III (At-IH), fibrinogen, Factor XII, and von Willebrand factor by radioimmunoassay (RIA) and by immunogold labeling.

Heparin-coated circuits significantly ($p<0.05$) reduced platelet adhesion during CPB, but did not alter platelet sensitivity to ADP nor reduce the increase in plasma BTG, C3b or neutrophil elastase. There were no significant differences between groups at any time for FPA, F1.2, TAT, or D-dimer. In both groups F1.2 and TAT increased progressively and significantly during CPB and after protamine. After CPB, F1.2=5.8 nmol/L, control; 5.7 nmol/L, Carmeda; TAT=52.1 pmol/L, control; 51.6 pmol/L, Carmeda. FPA increased significantly after protamine in both groups (30.1 ng/ml, control, 28.7 ng/ml, Carmeda). D-dimer increased significantly during CPB, but not after protamine. RIA showed a significant increase in surface adsorbed At-III on coated circuits, but no

significant differences between groups for the other proteins.

We conclude that heparin-coated circuits fail to reduce the significant increases in all markers of thrombin and fibrin formation during CPB and also fail to attenuate activation of complement, platelets and neutrophils. For clinical CPB these hematologic and biochemical data indicate that heparin-coated circuits are not advantageous and that they should not be used with reduced systemic doses of heparin except under extraordinary circumstances.

*By invitation

TUESDAY MORNING, APRIL 25,1995

Back to Annual Meeting Program

TUESDAY MORNING, APRIL 25, 1995
7:00 am FORUM SESSION I

Auditorium, Hynes Convention Center
Moderators: Andrew S. Wechsler, M.D.

Douglas J. Mathisen, M.D.

F1. INHALED NO FAILS TO CONFER THE PULMONARY PROTECTION PROVIDED BY DISTAL STIMULATION OF THE NO PATHWAY AT THE LEVEL OF CGMP

Yoshifumi Naka, M.D., Ph.D.*, Dillip K. Roy, M.D.*, Arthur J. Smerling, M.D.*, Robert E. Michler, M.D.*, Craig R. Smith, M.D., David M. Stern, M.D.*, David J. Pinsky, M.D.* and †Mehmet C. Oz, M.D.*

New York, New York

Inhalation of nitric oxide (NO) gas effectively lowers pulmonary vascular resistance (PVR) in the adult respiratory distress syndrome, and some have suggested that it may be beneficial following lung transplantation. However, during reperfusion, NO combines rapidly with

superoxide to form highly toxic peroxynitrite. We hypothesized that distal stimulation of the NO/cGMP pathway at the level of cGMP might confer the beneficial vascular effects of NO without its potential toxicities. To test this hypothesis, we used a recently described orthotopic rat left lung transplant model in which hemodynamic and survival measurements can be obtained independent of the native lung. Three conditions were tested: (1) **Control** (EC preservation solution), (2) Inhaled **NO** (EC preservation solution but inhaled NO given to recipient), and (3) EC preservation solution supplemented with the membrane permeable, nonhydrolyzable **cGMP** analog [8-(4-chlorophenylthio)-guanosine-3',5'-cyclic monophosphate; cGMP, 250 |iM]. The left lung was harvested from 22 male Lewis rats, preserved for 6 hrs at 4° C, and transplanted into isogenic recipients using cuff technique for all anastomoses. All recipients were ventilated with 70% ©2 and 30% N2 (as the carrier gas for NO was N2), which was supplemented with NO (65 ppm, monitored by chemiluminescence) in the second group. Gas mixtures were begun immediately prior to cross-clamp release and continued throughout reperfusion. In all experiments, the native (right) PA was ligated immediately after placement of a PA flow probe and Millar catheters into the LA and PA. PA flow (mL/min), arterial oxygenation (pO2, mm Hg) graft neutrophil infiltration (myeloperoxidase activity; MPO, A absorbance/min at 460 nm) and recipient survival (%) at 30 min. were measured. PVR (WU x 1000) was calculated. (Means ± SEM are shown; *=p<0.05 vs control, **=p<0.01 vs control; t=p<0.05 vsNO).

	PVR	pO_2	MPO	Survival
Control (n=6)	12.1 ± 2.6	82.2 ± 20	3.1 ± 0.2	0%
NO (n=12)	6.5 ± 1.4	170 ± 50	2.9 ± 0.2	33%
cGMP (n=4)	1.1 ± 0.1*	369 ± 28*†	1.7 ± 0.1*†	100%**†

Only the cGMP analog was associated with reduced PVR, improved pO2, reduced graft neutrophil infiltration, and improved recipient survival. These data suggest that stimulation of the NO/cGMP pathway at the level of cGMP has a protective effect that is not seen with inhaled NO in the immediate pulmonary reperfusion period.

†Robert E. Gross Research Scholar

*By invitation

F2. DIRECT EFFECTS OF 3,5,3' TRIIODO-L-THYRONINE (T_3) ON MYOCYTE CONTRACTILE PROCESSES: INSIGHTS INTO MECHANISMS OF ACTION

‡Jennifer D. Walker, M.D.*, Rupak Mukherjee, M.S.*, Fred A. Crawford, M.D. and Francis G. Spinale, M.D., Ph.D.*

Charleston, South Carolina

Administration of 3,5,3' triiodo-L-thyronine (T_3) in the setting of congestive heart failure and cardiopulmonary bypass has been suggested to improve left ventricular (LV) function; however, the cellular basis for this improvement remains unknown. Accordingly, the present study examined the direct effects of T_3 administration on myocyte function and the contributory mechanisms for these effects. In isolated porcine LV myocytes (n=81), velocity of shortening increased in the presence of 80 pM T_3 compared to baseline (77.1±3.3 vs 117.0±5.0 jam/sec, p<0.05). In a separate series of experiments (n=29), pre-treatment with 80 pM T_3 increased P-adrenergic response (pAR: 25 nM isoproterenol) compared to PAR alone (203.7±16.2 vs 274.3±16.9 |im/sec, p<0.05). In isolated myocyte preparations (n=9), cyclic-AMP production (fmols/myocyte) was measured. Cyclic-AMP was unchanged with T_3, but T_3 pretreatment with PAR increased cyclic-AMP compared to PAR alone.

	Baseline	80 pM T_3	ÃŸ
Cyclic-AMP	24.7±5 8	39 1±8 3	120 1
L-type Ca^{2+} Current Density	-3 63±0.28	-3.76±0 18	-6.27
Peak Intracellular Ca^{2+}	159.9±3.9	159.6±5.7	179.9

*p<0 05 vs baseline, $^{+}$p<0.05 vs

Since cyclic-AMP modulates intracellular Ca^{2+} processes, L-type Ca^{2+} processes, L-type Ca^{2+} channel density (patch clamp methods; pA/pF, n=15) and peak Ca^{2+} release from the sarcoplasmic reticulum (Fura-2 ionic measurement, pM, n=46) were measured. T_3pretreatment followed by PAR increased L-type Ca^{2+} channel current and intracellular Ca^{2+}compared to PAR alone. The unique findings from this study are twofold. First, T_3 stimulates steady state myocyte contractile function by a cyclic-AMP independent mechanism. Second, T_3 potentiates the effects of PAR by increasing cyclic-AMP and subsequent phosphorylation of L-type Ca^{2+} channels. Thus, T_3 enhanced PAR transduction with resultant increased Ca^{2+}availability and improved myocyte inotropic

response. These findings suggest the T_3 may provide a unique adjunct to conventional P-adrenergic agonist therapy in the clinical setting.

‡Nina S. Braunwald Research Fellow

*By Invitation

F3. INFLUENCE OF VEIN VALVES IN THE DEVELOPMENT OF ARTERIOSCLEROSIS IN RABBIT VENO-ARTERIAL GRAFTS

Aurelio Chaux, M.D.*, Xin Min Ruan, M.D.*, Michael C. Fishbein, M.D.* and Jack M. Matloff, M.D.

Los Angeles, California

Purpose: To study the influence of vein valves and define possible mechanisms for the formation of arteriosclerotic lesions in veno-arterial grafts.

Methods: Jugular vein grafts were interposed into the carotid circulations of 48 hypercholesterolemic rabbits. In 24 animals (Group A), vein segments did not contain vein valves; in another 24 (Group B), a vein valve was present. Subgroups of 6 animals were sacrificed at 2, 4, 6 and 8 weeks. Two mm sections were fixed in 4% paraformaldehyde and placed in sucrose for morphology and morphometry or frozen for immunohistochemistry and in situ hybridization for growth factor expression. Morphometry was done with computer-based image measuring software (Optimas) and validated by manual planimetry in 70% of animals. Cell identification and couting was done by immunohistochemistry. Dependent variables: graft mean area, mean thickness and proliferated cell nuclear antigen. Independent variables: presence or absence of vein valves and time in weeks post surgery. Measurements of mean area and thickness at cross sections were made just before and after the valve in Group B. Post valve values were also compared with those in the middle of the graft of Group A animals. Paired t-test and regression analysis were used.

Results: At 2 weeks intimal and medial thickening were due to an increase in cell number. From 2 to 6 weeks further intimal and medial thickening occurred without additional increase in cell number. At 6 weeks, foam cells and lipid deposits appeared, and at 8 weeks changes identical to human arteriosclerotic plaques were evident.

Changes developed sooner and more intensely in Group B animals (p<001), and segments immediately after the valve developed them faster and more severely than before the valve (p<.001). PCNA staining showed greater cell proliferation in the post-valve segments (p<.001). Endothelial and intimal growth factor expression was strong for IGF-1 and EOF, mild for PDGF.

Conclusions: The presence of vein valves augments and accelerates intimal hyperplasia and arteriosclerosis, and at least 3 growth factors may be involved in the development of these lesions. To our knowledge, this is the first controlled experiment that demonstrates this phenomenon.

*By invitation

F4. MYOCARDIAL STUNNING: A THERAPEUTIC CONUNDRUM

Zhandong Zhou, M.D.*, Robert D. Lasley, Ph.D.*, Rolf Bunger, M.D, Ph.D.*, Julia Hegge, B.S.* and Robert M. Mentzer, Jr., M.D. *Madison, Wisconsin and Bethesda, Maryland*

Dobutamine (DOB) and pyruvate (PYR) are two potent inotropic agents with different mechanisms of action. While both can be used to treat post-ischemic myocardial dysfunction, the potential deleterious effects of augmenting myocardial contractility in the setting of myocardial stunning has not been addressed. To test the hypothesis that these agents may have adverse effects on the stunned heart, systolic wall thickening (SWT), myocardial phosphorylation potential index (CrP/CrxPj), interstitial fluid adenosine (ISF [ADO]) and myocardial oxygen consumption (MV02) were measured in open chest pigs. SWT was measured using sonomicrometry, and microdialysis probes were inserted to estimate ISF [ADO]. Stunning was induced with a 10 min occlusion of the anterior descending coronary artery. After 30 min reperfusion, pigs were treated with either DOB (10 jig/kg/min) or PYR (1 ml/min or a 150 mM solution). Results expressed as mean±SEM; *p<0.05 vs control, Ñ,,p<0.05 vs stunned.

Dobutamine Treatment (N=7) Pyruvate

	SWT (% Baseline)	ISF[ADO] (μM)	MVO_2 (ml/min/100g)
Control	100	0.47 ± 0.05	7.27 ± 0.22

	SWT (% Baseli
Control	100

Stunned	31.2 ± 1.0*	0.42 ± 0.04	7.17 ± 0.7
10'DOB	57.3 ± 5.4*Ñ„	7.5 ± 0.09*Ñ„	12.03 ± 1.4*
20' Post DOB	23.7 ± 1.8*Ñ„	0.45 ± 0.71	7.57 ± 0.71

Stunned	31.0 ± 4
10' PYR	75.2 ± 10.
20' Post PYR	30.0 ± 4

DOB produced myocardial de-energization, as the CrP/ $CrxP_i$. ratio decreased from 0.17±0.02 to 0.09±0.02 ($p<0.02$) in the stunned hearts. In contrast, PYR enhanced myocardial energy status, since the ratio increased from 0.20±0.03 to 0.55±0.08($p<0.01$).

Infusion of both inotropic agents resulted in a marked improvement in regional SWT. The dobutamine effect, however, produced a marked increase in MVO_2 associated with an expected rising ISF [ADO] due to the decline in the $CrP/CrxP_i$. This energetic stress may account for the further deterioration in cardiac function observed after cessation of DOB. In contrast, PYR improved postischemic contractile function and enhanced myocardial energetics. These experimental findings suggest that under certain circumstances the clinical use of ÃŸ receptor agonists to treat myocardial stunning after cardiac surgery or heart transplantation may be suboptimal, if not undesirable. Further investigation is warranted to determine the optimum threapy for the stunned heart.

*By invitation

F5. PRECONDITIONING WITH POTASSIUM CHANNEL OPENERS: A NEW CONCEPT FOR ENHANCING CARDIOPLEGIC PROTECTION

Philippe Menasche, M.D., Ph.D., Egidijus Kevelaitis, Ph.D.*, Christian Grousset, M.D., Ph.D.*, Armand Piwnica, M.D. and Gerard Bloch, M.D.*

Paris, France

Background. Preconditioning (PC) is now recognized as one of the most effective means of improving the tolerance of myocardium to a sustained episode of ischemia. It has been suggested that this protection could result from an adenosine-induced opening of adenosine triphosphate-sensitive potassium channels and the related limitation of calcium overload.

Objective. This study was therefore designed to assess whether the protective effects of ischemic PC can

be mimicked by the administration of a potassium channel opener in the surgically relevant setting of global myocardial ischemia.

Methods. Forty isolated isovolumic rat hearts were subjected to 45 minutes of normothermic potassium arrest followed by 1 hour of reperfusion. Hearts were randomly divided into four equal groups that only differed by the modalities of the prearrest treatment protocol: group 1 had no prearrest intervention (controls); group 2 hearts were preconditioned with 5 minutes of global ischemia followed by 5 minutes of buffer reperfusion prior to the onset of potassium arrest; in group 3, the preconditioning stimulus consisted of a 5-minute infusion of the potassium channel opener nicorandil (lOpM) followed by 5 minutes of drug-free buffer perfusion prior to potassium arrest; in group 4, the same protocol as in group 3 was used except that the administration of nicorandil was preceded by a 5-minute infusion of the selective potassium channel blocker glibenclamide ([glib]10|aM). During arrest, the left ventricular balloon was kept inflated to monitor the time course of ischemic contracture.

Results. They are summarized below (data analysis by 2-way ANOVA with repeated measures).

Group	Diastolic Pressure (mmHg)		LV dP/dt (mmHg/sec^{-1})	
	Baseline	Reperfusion	Baselin e	Reperfusion
1 (controls)	9.6 ± 0.6	53.5 ± 2.9	4,745 ± 170	1,599 ± 99
2 (ischemic PC)	9.2 ± 0.7	29.7 ± 3.1*°	4,570 ± 167	2,970 ± 141**
3 (nicorandil PC)	9.1 ± 0.3	29.0 ± 1.4*#	4,767 ± 161	2,724 ± 105**
4 ((nicorandil+ glib)	11. 3 ± 0.9	46.2 ± 2.4	5,002 ± 117	1,965 ± 77
*P<0.0001 vs group 1	° P<0.0002 vs group 4		** P<0.0001 vs groups 1 & 4	
#P<0.0001 vs group 4				
All data are mean ± SEM				

Pretreatment with nicorandil also lowered peak contracture during arrest significantly (40.4 ± 2.1 mmHg vs 51.8 ± 3.0 mmHg in controls [P<0.01 vs nicorandil], 50.0 ± 2.8 mmHg in ischemically preconditioned hearts [P<0.01

vs nicorandil] and 54.9 ± 5.3 mmHg in nicorandil+glibenclamide-treated hearts [P<0.01 vs nicorandil]) and lengthened the time to peak contracture, as compared with the control and nicorandil+glibenclamide groups (2,066 ± 45 sec vs 1,806 ± 91 sec [P<0.03] and 1,573 ± 104 sec [P<0.001], respectively).

Conclusion. The protective effects of ischemic PC can be mimicked by a pharmacological opening of adenosine triphosphate-sensitive potassium channels. The surgical relevance of these data stems from (1) the possibility to plan the onset of aortic cross-clamping and, consequently, to implement a PC stimulus in a timely appropriate fashion, and (2) the approval of nicorandil for clinical use in patients with coronary artery disease.

*By invitation

F6. THE ROLE OF NITRIC OXIDE IN CEREBRAL RECOVERY AFTER CARDIOPULMONARY BYPASS WITH DEEP HYPOTHERMIC CIRCULATORY ARREST IN INFANT PIGLETS

Takeshi Hiramatsu, M.D.*, Takuya Miura, M.D.*, Adre Duplessis, M.D.*, Masahiro Tanji, M.D.*, Joseph M. Forbess, M.D.*, Miles K. Tsuji, M.D.*, David Holtzman, M.D.* and Richard A. Jonas, M.D.

Boston, Massachusetts

Although nitric oxide (NO) is neurotoxic in vitro, there is continuing controversy regarding the role of NO in global cerebral ischemia. The effects on brain metabolic recovery of L-arginine (L-arg), a NO precursor, and L-Nitro-Arginine Methyl Ester (L-NAME), a NO synthase inhibitor, have been studied in an infant piglet model of deep hypothermic circulatory arrest (DHCA).

METHODS: Forty 2-week old piglets underwent core cooling to 15°C, 1 hour of DHCA at 15°C, 45 minutes of rewarming and 3 hours of normo-thermic reperfusion. Group L-arg (n=10) received 30 mg/kg of L-arginine IV before cardiopulmonary bypass (CPB) and 10 mg/kg/min infusion during the first 1 hour of reperfusion (Rep). Group L-NAME 10 mg/kg (n=10) received 10 mg/kg of L-NAME IV before CPB. Group L-NAME+L-arg (n=10) received both L-NAME and L-arg as in the first two groups.

Control (n=10) received no medication. Recoveries of cerebral high energy phosphates and pHi were assessed by magnetic resonance spectroscopy in half of the animals in each group. Cerebral blood flow (CBF) and cerebral vascular resistance (CVR) by microspheres, cerebral metabolic rates of oxygen and glucose, and the redox state of cytochrome a,a3 by near infrared spectroscopy were assessed in the rest. Brain water content was measured in all animals after the experiments.

RESULTS: Results are given as mean±SEM. *p<0.05 vs. Control by Student-Newman-Keuls test

	min. Rep	Control	L-arginine
ATP	45	73.7 ± 15.9	83.5 ± 6.2
(% recovery)	225	71.5 ± 5.0	84.3 ± 2.7*
pHi	45	6.75 ± 0.05	6.79 ± 0.05
(Unit)	225	7.23 ± 0.11	7.28 ± 0.04
Cytochrome a,a3	45	-7.0 ± 1.1	-2.3 ± 1.3*
(⎮iM x DPF)	225	0.4 ± 0.8	1.0 ± 1.4
Mean arterial	45	45.2 ± 4.1	44.2 ± 3.7
Blood pressure (mmHg)	225	121 ± 8	112 ± 7
CBF	45	17.3 ± .3	32.6 ± 5.9*
(mL/min/100g)	225	50.6 ± 4.7	59.3 ± 9.6
CVR	45	3.20 ± 0.43	2.02 ± 0.35
(unitx 100g)	225	2.14 ± 0.17	1.96 ± 0.22
Brain water content		81.8 ± 0.3	81.8 ± 0.2

SUMMARY: L-arginine improved recovery of cerebral ATP, cyto-chrome a,a3 oxidation and cerebral blood flow. L-NAME reduced recovery of cerebral ATP, pHi and cytochrome a,a3 oxidation and increased mean blood pressure, CVR and brain water content. These effects of L-NAME were minimally reversed by L-arginine infusion after reperfusion.

CONCLUSIONS: The net effect of intra-neuronal and endothelial nitric oxide is to promote cerebral metabolic recovery after global ischemia.

*By invitation

F7. THE EFFECTS OF RETROGRADE CEREBRAL PERFUSION AFTER PARTICULATE EMBOLIZATION TO THE BRAIN

Mustafa E. Yerlioglu, M.D.*, Craig K. Mezrow, M.S.*, All M. Sadeghi, M.D.*, Peter S. Midulla, M.D.*, Ning Zhang, M.D.*, Howard H. Shaing, D.V.M.*, Donald J. Weisz, Ph.D.* and Randall B. Griepp, M.D.

New York, New York

Neurologic injury as a consequence of cerebral embolism of air or atherosclerotic debris during cardiac and aortic surgery remains a major cause of morbidity. We have developed a chronic porcine model to evaluate the potential of retrograde cerebral perfusion (RCP) for improving cerebral outcome following cardiac surgery, and have previously demonstrated that it results in a small amount of nutritive flow, and provides cerebral protection superior to prolonged hypothermic circulatory arrest. We designed the current study to evaluate the efficacy of RCP in mitigating the effects of cerebral embolism of paniculate matter during cardiac surgery.

Four groups of pigs (20-28 kg) were assigned to undergo deep hypothermia at an esophageal temperature of 20°C: a retrograde control group (RC, n=5); a retrograde embolism group (RE, n=10); an antegrade control group (AC, n=5); and an antegrade embolism group (AE, n=6). The sagittal sinus (SS) was cannulated in all animals, and flow in the SVC was regulated in order to achieve and maintain a SS pressure of 30 mmHg during RCP. During ante-grade perfusion, the aortic arch pressure was maintained at 50 mmHg. Polystyrene microspheres, (250-750 um, 200 mg), were utilized as the embolic material. In the embolism groups, the spheres were injected via a cannula in the isolated arotic arch; controls were injected with 10 cc of saline. Five minutes after injection, antegrade perfusion was continued in antegrade groups, and retrograde perfusion via an SVC cannula was instituted in the retrograde groups, both for a duration of 25 minutes. Blood returning to the aortic arch was collected and measured in the RCP groups. All animals were allowed to recover for at least five days and were evaluated daily using a quantitative behavioral score, in which 9 indicates complete normalcy, 7 means that the animals were able to stand unassisted

and were likely to recover fully, and lower numbers indicate substantial injury, with 0=death. At the time of elective sacrifice, half of the brain was utilized for recovery of embolized spheres after digestion with ION NaOH.

As shown in the table, neurological recovery in the control groups was complete with retrograde as well as with antegrade perfusion. Following embolization, there was severe neurological injury in both groups, and both antegrade and retrograde flows were reduced compared with controls. Although fewer spheres were present in the brains of the RE group, neurologic outcome was no better in the group as a whole then in the AE group. When the RE group was examined in greater detail, however, it was seen that some animals recovered almost completely after retrograde perfusion (score>7, good outcome). Further scrutiny showed that these good outcome RE animals required significantly lower SVC pressure to achieve adequate SS pressures than the animals that sustained severe neurologic injury following embolization and RCP. Although the mechanism of the injury following use of high SVC pressures during RCP after embolization (but not in controls) remains unclear, our data suggest that RCP during cardiac surgery may mitigate cerebral injury from participate emboli if adequate RCP can be achieved using SVC pressure <40 mmHg.

Group	n	Flow Rate (ml/kg/min)	Aortic Arch Return Rate (ml/kg/min)	SVC Pressure (mmHg)
AC	5	11.0 ± 3.1		
RC	5	34.7 ± 32.7	0.8 ± 0.4	53.8 ± 23.6
AE	6	5.1 ± 1.0[a]		
RE	10	17.5 ± 10.3	0.5 ± 0.2	48.4 ± 19.2
RE Good Outcom	4	10.3 ± 0.4	0.5 ± 0.1	33.7 ± 3.9
RE Poor Outcome	6	22.3 ± 11.1	0.4 ± 0.2	58.2 ± 19.1

Mean values ± STD Deviation

a: p<0.05 vs. AC

b: p<0.05 vs. RC

F8. EFFECTS OF CARDIOPLEGIA ON VASCULAR FUNCTION AND THE NO-REFLOW PHENOMENON FOLLOWING ISCHEMIA-REPERFUSION: STUDIES IN THE ISOLATED BLOOD-PERFUSED RAT HEART

Vincenzo Argano, M.D., FRCS*, Manuel Galinanes, M.D., Ph.D.*, Stephen J. Edmondson, MRCP, FRCS* and David J. Hearse, Ph.D., D.Sc.*

London, United Kingdom

Sponsored by: Mark V. Braimbridge, London, United Kingdom

The protective role of cardioplegia against post-ischemic cardiac dysfunction is well established. However, the effect of cardioplegia on vascular function and its repercussion on the no-reflow phenomenon are controversial. We have investigated the influence of St. Thomas' cardioplegic solution on: (i) endo-thelium-dependent and endothelium-independent vascular function (EDVF

and EIVF), and (ii) the extent of the no-reflow phenomenon. Isolated rat hearts (n=16/group) perfused with blood at 60 rnmHg, were subjected to 10, 20, 30 or 40 min of global ischemia and 40 min of reperfusion (37°C). Eight hearts in each group also received cardioplegia (40 mmHg for 2 min) before ischemia. At the end of reperfusion, a bolus of 250ug (3mM) of nitro-L-arginine methyl ester was infused to assess EDVF. After 20 min washout, 25ug (0.3mM) of sodium nitroprusside was infused to assess EIVF. Fluorescein (1 ml, 1% w/v) was then infused to assess no-reflow. Hearts were frozen, cut into transverse sections (lOxlmm), video-recorded under UV light, and images digitised and analysed for density of fluorescence. No-reflow was defined as a flow <5% (corresponding to 171-256 grey-scale density). EIVF (percent of non-ischemic control) was severely decreased only after 30 min and 40 min of ischemia (45. ± .6% and 31. ± 2.5%; $p<0.05$), but was significantly protected by cardioplegia (62. ± .7% and 57. ± .9%). A significant reduction in EDVF was only observed after 40 min of ischemia (69. ± 0.6%; $p<0.05$) and again this was improved by cardioplegia (89. ± .8%). Areas of no-reflow were present after 30 and 40 min of ischemia (11. ± .8% and 33. ± 4.1% of LV mass) and they were significantly decreased by

cardioplegia (0. ± .4% and 3. ± .6%; p<0.05). In conclusion, cardioplegia protects against post-ischemic endothelium-dependent and endothelium-independent vascular dysfunction and reduces the extent of no-reflow.

*By invitation

9:00 am PLENARY SESSION

Auditorium, Hynes Convention Center
Moderators: Robert B. Wallace, M.D.

James L. Cox, M.D.

15. SURGICAL MANAGEMENT OF EXTENSIVE

CALCIFICATION OF THE MITRAL VALVE ANNULUS

Alain F. Carpentier, M.D., Ph.D., Jean-FranÃ§ois Fuzelier, M.D.*, John Y.M. Relland, M.D.* and Michel Pellerin, M.D.*
Paris, France

Extensive calcification of the mitral valve annulus (MVA) is a pathological entity frequently associated with degenerative valvular disease. The calcification process involves at least 1/3 of the circumference of the MVA and usually not the valvular tissue. It may extend however to the underlying myocardium. Whenever an operation is necessary for an associated valve insufficiency, the question arises whether it is preferable to repair or to replace the valve and how to manage the calcification. This paper reports the technical details, the recent improvement and the mid-term results of a reconstructive operation proposed in 1986 which up to now has only been the object of early and brief reports on small series. This operation comprises the temporary detachment of the corresponding leaflets, en bloc resection of the calcium deposit with its surrounding fibrous capsula, annular reconstruction and valve repair. For patients in whom the calcification process extends to the myocardium, a modified operation has been more recently developed which comprises in addition a "sliding

plasty" of the left atrium over the area of resected calcium.

Between 1986 and 1994, among 63 patients (pts) with extensive calcification of the annulus and severe mitral valve insufficiency, 62 benefited from these techniques of repair. Ages ranged from 18 to 81 years (mean 62). Twenty-eight pts had a billowing mitral valve (Barlow), 32, a fibroelastic deficiency and 2, Marfan's disease. The calcification involved 1/3 of the annulus in 26 pts, 2/3 in 34 pts and the whole annulus in 2 pts. In 8 pts, the calcification process extended to the papillary muscles (4 pts) and/or deeply within the myocardial wall (6 pts). There were 2 hospital deaths (3.2%) and no early reoperation. The follow-up period extended from 4 months to 8 years (mean 3 years 8 months). There were 2 late deaths, 2 and 17 months after the operation (one valve related) for an actuarial survival of 95.7% at 7 years.

Late reoperation (3 to 62 months) was necessary in 4 pts (6.4%) for residual mitral valve incompetence (MVI) (n=2), hemolysis (n=1) or endocarditis (n=1). In one of these pts, a new repair was possible, whereas the 3 other pts required a valve replacement. All patients but one survived the reoperation. Actuarial freedom from reoperation was 88% at 7 years.

All 56 patients with valve repair were reviewed for this study by clinical examination and echocardiography. All but one were in functional chiss I or II. There was no MVI in 19, trivial MVI in 17 and moderate MVI in 7. No thromboembolic complications have been recorded.

This study shows that complete annulus decalcification and valve repair can be carried out safely in patients with mitral valve insufficiency and associated extensive calcification of the annulus, even when the calcification process deeply involves the myocardium. It also demonstrates that an initially good result remained stable up to 7 years.

*By invitation

16. POSTINFARCTION VENTRICULAR SEPTAL RUPTURE: REPAIR BY ENDOCARDIAL PATCH WITH INFARCT EXCLUSION

Tirone E. David, M.D., Laura Dale, R.N.*,
Robert J. Cusimano, M.D.* and Zhao Sun,
B.A.*

Toronto, Ontario, Canada

Because the extent of right ventricular infarction has been identified as an important determinant in the outcome of pts with postinfarction ventricular septal defect (VSD), an operation that avoids further damage to the right ventricle was developed and it has been applied in 43 pts during the past 8 years. The procedure is performed by opening the left ventricle through the infarcted wall and by suturing a pericardial patch (fresh autologous or bovine pericardium) to the endocardium of the left ventricle with a continuous 3-0 polypropylene suture to exclude the infarcted muscle from the left ventricular cavity. The ventriculotomy is closed primarily and no infarctectomy is performed.

There were 21 men and 22 women, ages 48 to 84 years, mean 68. Twenty-seven pts were in cardiogenic shock when operated on and all had an intraaortic balloon pump inserted preoperatively; 7 of them also required assisted ventilation and 13 were anuric. Doppler echocardiography was used to confirm the diagnosis and assess left and right ventricular function in all pts. The VSD was anterior in 22 pts and posterior in 21. All pts had coronary angiography. Pts in shock were operated on as an emergency and most pts who were hemodynamically stable were operated on within 3 days of diagnosis. In addition to the endocardial pericardial patch, 29 pts had coronary artery bypass and one pt also had mitral valve replacement. There were 6 operative deaths (14%); 3 in pts with anterior VSD and 3 in pts with posterior VSD. Eleven pts required renal dialysis and 15 required assisted ventilation for more than 48 hours. ICU stay (mean ± SD) was 7.8 ± 10.3 days and hospital stay was 20 ± 18 days. Only one pt had a recurrent VSD but it closed spontaneously within one month. Operative survivors have been followed from 3 to 95 months, mean of 42. There were 7 late deaths, 4 cardiac. The actuarial survival at 7 years was 64% ± 8%. The only two factors that were predictive of mortality were renal failure and left ventricular ejection fraction <35%, by a stepwise logistic regression analysis. All pts are in NYHA class I or II. Doppler echocardiography was performed at least once a year in all survivors and revealed mild impairment of left ventricular function in 18 pts, moderate in 11 and severe

in 1. All pts have normal or near normal right ventricular function.

This new operative technique is relatively simple and appears to improve the outcome of pts with postinfarction VSD.

*By invitation

17. AORTIC VALVE REPAIR IN ACUTE TYPE A DISSECTION: IS IT SOUND?

Ludwig K. von Segesser, M.D.*, Urs Niederhauser, M.D.*, Marietta Schonbeck, M.D.*, Paul Vogt, M.D.* and Marko I. Turina, M.D.

Zurich, Switzerland

The last 200 consecutive patients with acute Stanford type A dissection (157 men [78%], 43 women [22%]) were analyzed in order to assess the validity of aortic valve repair whenever possible. Indication for surgery was in most cases based on echocardiographic examination only in order to reduce the doctors' delay. In the majority of patients (111/200: 56%), the incompetent aortic valve was resuspended and repaired. Aortic root replacement with a compositd graft was performed in 66/200 patients (33%) mainly because of enlarged aortic anulus and sinus portion. Replacement of the aortic valve and the supracoronary ascending aorta was performed in 23/200 patients (12%) with diseased aortic valve (e.g., bicuspid valve) but acceptable aortic sinus portion. Follow-up totalized 656 patient

years (maximum 14 years). Actuarial analyses as a function of aortic valve type provided the following probabilities ± errors (95%).

	interval	all	repaired
type of valve/patients		200/200 (100%)	111/200(56%)
survival	30 days	78.3 ± 2.9	72.8 ± 4.3
	1 year	74.9 ± 3 1	68.5 ± 4.6
	5 years	67.9 ± 3.6	60.8 ± 5.2
	1 0 years	48.5 ± 6.1	43.4 ± 8.2
freedom from valve failure	30 days	100.0 ± 0.0	100.0 ± 0.0
	1 year	100.0 ± 0.0	100.0 ± 0.0
	5 years	99.1 ± 0.9	97.7 ± 2.3
	10 years	99.1 ± 0.9	97.7 ± 2.3
freedom from reoperation	30 days	100.0 ± 0.0	I00.0 ± 0.0
(same segment)	1 year	99 3 ± 0.7	98.6 ± 1 3
	5 years	97 5 ± 1.5	96.7 ± 2.3
	10 years	95.1 ± 2.8	91.2 ± 5.8

During long term follow-up, there was no significant difference between groups with regard to structural deterioration, valve thrombosis, thrombo-embolic complications, anticoagulation-induced hemorrhage, and endocarditis. Freedom from valve failure and valve-related complications are similar for repaired, mechanical, and biological valves. Consequently, valve-related reoperations are rare during at least five years of follow-up. Hence, aortic valve repair in acute type A dissection can be recommended in the majority of patients.

10:05 am INTERMISSION - VISIT EXHIBITS

*By invitation

10:50 am PLENARY SESSION

Auditorium, Hynes Convention Center

Moderators: Robert B. Wallace, M.D.

James L. Cox, M.D.

18. PREDICTORS OF SURVIVAL IN MALIGNANT TUMORS OF THE STERNUM

Nael Martini, M.D., Andrew G. Huvos, M.D.*, Michael E. Hurt, M.D., Ph.D., Robert Heelan, M.D.*, Manjit S. Bains, M.D., Patricia M. McCormack, M.D., Valerie W. Rusch, M.D., Michael Weber, S.A.C.* and Robert J. Ginsberg, M.D.

New York, New York

From 1930 to 1994, 54 patients with primary malignant tumors of the sternum were seen. The median age was 54 years (range: 19-78 years). Fifty patients presented with a mass, one-half of them also had pain in the sternal region. Two patients had no symptoms at presentation. Of 39 solid tumors, 26 were chondrosarcomas, 10 osteosarcomas, 1 fibrosarcoma, 1 angiosarcoma and 1 malignant fibrous histiocytoma. Of these, 25 were low grade and 14 were high grade tumors. Of 15 small cell tumors, 8 were plasmacytomas, 6 malignant lymphomas, and 1 Ewing's sarcoma.

The size of the tumor ranged from 3 to 24 cm. in maximum diameter (median 7 cm.). Twenty-one patients had only plain chest roentgenograms, 26 had CT scans, 15 plain tomograms and 7 had MR scans. Tissue diagnosis was established by needle aspiration in 3 patients, open biopsy in 38 and at surgery in 13.

Partial or subtotal sternectomy was done in 36 patients and total sternectomy in 3 (34/39 sarcomas and 5/15 small cell tumors). Of the remaining 15 patients, 4 had local excision, 10 external radiation and/or chemotherapy and one had no treatment. All but one patient treated by wide resection (N=39) had some form of skeletal reconstruction of the chest wall defect. Twenty-three were repaired with marlex mesh and methylmethacrylate, 7 with marlex mesh, 3 with ox fascia, 2 with fascia lata, 1 with rub struts + fascia lata, 1 with tentalum mesh, and 1 by shifting pectoral muscles to cover the defect. The skin edges were closed by primum in 37 patients; 2 required skin grafts.

There were 2 deaths secondary to respiratory failure (one due to inadequate stabilization following total sternectomy and one due to pneumonia). Two infections necessitated removal of the methylmethacrylate prosthesis.

In solid tumors, adverse prognostic factors were high grade tumor, pulmonary metastases, inadequate or no resection and local recurrence.

Resection in chondrosarcomas yielded a 5-year survival (Kaplan-Meier) of 80% (median follow-up: 17 years). The 5-year survival in osteosarcomas was 14%. Resection was curative in 54% of low grade sarcomas but in only 7% of high grade sarcomas. The local recurrence rate following complete resection was 14%. In small cell tumors, resection and radiation were helpful for local control; all failures were due to distant metastases.

We conclude that primary sarcomas of the sternum though uncommon are potentially curable by wide surgical excision. With rigid prostheses to repair the skeletal defects, the surgical complication rates are minimized and survival following treatment remains tumor dependent.

*By invitation

19. TRANSMYOCARDIAL LASER REVASCULARIZATION: INITIAL CLINICAL EXPERIENCE

Denton A. Cooley, M.D., O.H. Frazier, M.D., Kamuran A. Kadipasaoglu, Ph.D.*, Seckin Pehlivanoglu, M.D.*, Eddy Barasch, M.D.*, Matthias Lindenmeir, M.D.*, Dena P. Houchin, R.N.*, Wanda Samuels, R.N.*, K. Lance Gould, M.D.* and Susan Wilansky, M.D.*

Houston, Texas

Transmyocardial laser revascularization (TMLR) with an 850-W CO_2 laser involves drilling 1-mm diameter channels into a beating heart after left thoracotomy. Clotting acutely occludes the channels on the

subepicardium (SEp) and, in the long term, camerosinusoidal connections improve subendo-cardial (SEn) perfusion. We performed TMLR in 21 consecutive patients (pts; mean age, 63 y; 3 women) who had hibernating myocardium and/or reduced coronary flow reserve by positron emission tomography (PET) and low-dose dobutamine echocardiography (DE). All pts had distal diffuse coronary artery disease refractory to antianginal therapy. The mean angina class (AC_m, Canadian Cardiovascular Society) was 3.76 (4 pts with unstable angina) and the mean resting left ventricular ejection fraction (LVEF) by MUGA was 47%.

During follow-up, AC_m was 2.40 at 3 mo (n=15 pts, $p<0.01$), and 1.5 at 6 mo (n=l 1 pts, $p<0.001$). METs (ml Oj/kg/min) on treadmill improved from 3.2 at baseline (BL) to 4.4 at 3 mo (p=NS), and to 5.6 at 6 mo ($p<0.05$). Resting LVEF increased but not significantly at either time. On PET analysis at 3 mo, the mean SEn/SEp perfusion ratio increased by 8% in lased regions but decreased by 4% in nonlased septal regions (n=10 patients, $p<0.01$). On DE, the percentage of normokinetic segments (15-segment analysis) had increased from 46% at BL to 57% at 3 mo (p=NS) and to 62% at 6 mo ($p<0.05$). Of 7 (33%) patients who had an adverse event after TMLR (5 deaths, 2 re-revascularizations), 4 (57%) had a history of anterior/anterolateral myocardial infarction (vs 0% of 14 non-event pts, $p<0.001$), 6 (86%) were being treated for congestive heart failure (vs 36%, $p<0.001$), and 5 (71%) had regurgitant mitral valve disease (vs 29%, $p<0.05$). Histopathologic examination of myocardium from one of the patients who died revealed multiple patent, endothelium-lined neochannels connected to the native sinusoidal vasculature.

These results suggest that, in patients with reversible myocardial ischemia, the laser channels remain functional in the long term and augment subendo-cardial perfusion. We conclude that TMLR produces immediate clinical benefit and improves cardiac function at 6 mo.

***11:30 am ADDRESS BY HONORED SPEAKER**

Medical Ethics in the 21st Century: DNR or CPR?
Edmund D. Pellegrino, M.D.,
Washington, D.C.

12:10 pm ADJOURN FOR LUNCH - IN EXHIBIT HALL

12:10 pm CARDIOTHORACIC RESIDENTS'
LUNCHEON Independence
Ballroom, Sheraton Boston Hotel

TUESDAY AFTERNOON, APRIL 25,1995

Back to Annual Meeting Program

TUESDAY AFTERNOON, APRIL 25, 1995
1:45 am SIMULTANEOUS SCIENTIFIC SESSION A ADULT CARDIAC SURGERY

Auditorium, Hynes Convention Center

Moderators: Tirone E. David, M.D.

Bruce A. Reitz, M.D.

20. HOMOGRAFT MITRAL VALVE REPLACEMENT: MEASUREMENT TECHNIQUES FOR GRAFT SELECTION AND IMPLANTATION

Christophe Acar, M.D.*, Jullien Gaer, M.D.*, Alain Berrebi, M.D.*, Denis Tixier, M.D.*, Christian Brizard, M.D.*, Arnaud Farge, M.D.*, Alain Deloche, M.D.* and Alain F. Carpentier, M.D., Ph.D.

Paris, France and London, England

Homograft replacement of the mitral valve (HRMV) has experienced a renewal of interest since the introduction of improved methods of homograft preservation. However, the reliability of this technique remains a major concern due to the complexity and the frequent variations of the subvalvular apparatus. Our experience of 40 HRMV (19 total, 21 partial) carried out in the past 3 years made it possible to define precise measurement criteria which proved to be useful to achieve predictable results. The indications for HRMV were bacterial endocarditis in 16 patients and rheumatic valvular disease in 24 patients.

Measurement on the homograft were the following: 1) Circumference (assessed by prosthetic ring sizers), 2) Height of the anterior leaflet (average 23 ± 3 mm), 3) Distance from annulus to apex of the anterior papillary muscle (PM) (average 23 + 4 mm). In addition, the morphology of each papillary muscle was classified as follows: Type I, simple head (n=20); Type II, bifid head (n=52); Type III, complex PM (n=8).

Measurements on the recipient were obtained by transesophageal echocardiography (TEE): 1) Circumference of the annulus, 2) Distance from annulus to apex of the anterior PM which averaged 20 ± 3 mm in the bacterial endocarditis group and 8 ± 2 mm in the rheumatic valve group.

The homograft valve was selected to match as much as possible these measurements. Further adjustments were made by systematic use of a Carpentier-Edwards ring selected from the size of the anterior leaflet of the homograft and by side-to-side implantation of the graft PM to the recipient PM. Associated procedures comprised aortic valve repair (n=3), or replacement using homograft (n=2) and tricuspid valve repair (n=1), or replacement using homograft (n=1).

There has been 1 hospital death due to low cardiac output and 1 reopera-tion 10 days following the operation due to annulus dehiscence. The follow-up extended from 3 months to 34

months (average 8 months). There has been 1 late death due to lung cancer. All the remaining 37 patients were reviewed for this study and a TEE was performed. All patients were in NYHA class I (n=33) or II (n=4). TEE showed no mitral valve regurgitation (MVR) in 18 pts, trivial MVR in 15 and moderate MVR in 4. The valve surface area was 2.0 ± 0.3 cm2 for partial HRMV and 2.4 ± 0.2 cm^2 for total HRMV with an average transvalvular gradient of 3 ± 2 mmHg.

This study shows that reliable results can be obtained with HRMV, a technique particularly useful for bacterial endocarditis in the adult and rheumatic valvular disease in children.

21. RECONSTRUCTION OF THE MITRAL ANNULUS - A TEN-YEAR EXPERIENCE

Christopher M. Feindel, M.D.*, Tirone E. David, M.D., Susan Armstrong, M.Sc.* and Zhao Sun, B.A.*

Toronto, Ontario, Canada

The mitral annulus (MA) may be damaged by extensive calcification, infective endocarditis or repeated mitral valve (MV) replacement making MV surgery hazardous. It is possible to reconstruct the MA with either autologous or bovine pericardium. All calcific or infected tissue is excised and a properly tailored pericardia! patch is sutured to the endocardium of the left ventricle posteriorly and to fibrous tissue underneath the aortic valve superiorly. If this fibrous tissue is also diseased, it can be replaced with pericardium, too. Thus, the MA can be repaired segmentally, circumferentially or along the entire base of the left ventricle.

Seventy-six pts have undergone reconstruction of the MA since 1984. There were 35 men and 41 women, ages 17 to 86 years, mean 58. The indications for reconstruction of the MA were: calcification in 19 pts (22%), MA abscess in 23 (30%), redo MV surgery in 30 (39%), and type I rupture of the left ventricle after MV replacement in 4 (5%). Three-fourths of the pts were in NYHA functional class IV and 18 were in septic and/or cardiogenic shock when operated on; 11 pts had coronary artery disease; and 48 pts had had at least one previous MV operation. The reconstruction of the MA was segmen-tal in 40 pts (52%), circumferential in 21 (27%), and involved both aortic and mitral annuli in 15 (20%). The MV was replaced in 70 pts and repaired in 6. There were 4 early reoperations in 3 pts: 1 for dehiscence of the patch, 1 for thrombosis of the valve, and 2 for new onset endocarditis. The operative mortality was 9% (7 pts). Pts have been followed up from 3 to 114 months, mean of 34. Every pt has had Doppler echocardiography annually. There has been no late patch dehiscence. Four pts have required reoperation: 2 for endocarditis, and 2 for failed bioprosthesis. There have been 13 late deaths, 8 cardiac and 3 valve-related. The actuarial survival at 5 years was 74% ± 4%.

Reconstruction of the MA with pericardium is an extremely useful operative technique in pts with damaged annulus, and provides excellent late results.

*By invitation

22. ATRIOVENTRICULAR VALVE REPAIR USING EXTERNALLY ADJUSTABLE FLEXIBLE RINGS

JoÃ£o Q. Melo, M.D.*, JosÃ© S. Neves, M.D.*, Regina Ribeiras, M.D.*, Miguel Abecasis, M.D.*, Manuel Canada, M.D.*, Maria J. Rebocho, M.D.*, Narciso C. Andrade, M.D.* and Manuel M. Macedo, M.D.

Lisbon, Portugal

Mitral and tricuspid valve repair using annuloplasty rings have a significant incidence of residual regurgitation which leads either to suboptimal results or to the need of a reoperation. We have developed a new technique for partial annuloplasty using a flexible externally adjustable ring. If required, it is adjusted from outside the heart after hemodynamic stabilization is obtained following interruption of extracorporeal circulation. Adjustment of the ring is controlled using transesophageal echocardiography. Twenty patients (pts), with a mean age of 45 years, ranging from 7 to 70 years, had 25 rings implanted, 14 in mitral position and 11 in tricuspid position. Most pts (12) were rheumatic in origin. Eight pts had pure mitral insufficiency and the other mixed lesions. All tricuspid lesions were regurgitation. Eight pts had complex mitral repairs and 8 pts had associated procedures: aortic repair (1 pt), aortic replacement (3 pts), maze 3 operation (2 pts), left atrial isolation (2 pts), coronary artery bypass (1 pt). Mitral regurgitation before surgery was grade 4 in 5 pts, grade 3 in 6 pts and grade 2 in 3 pts. After surgery it reduced to grade 2 (1 pt), grade 1 (5 pts) or none (8 pts). Tricuspid regurgitation reduced from grade 4 (4 pts), grade 3 (3 pts) and grade 2 (3 pts) to grade 1 (3 pts) and no regurgitation (7 pts). Mean pulmonary artery pressure was reduced from 67 to 40 mmHg.

External adjustment was performed in 10 mitral rings and in 8 tricuspid rings, improving the regurgitation by 1 or 2 grades. There was no hospital or late mortality. At 6 months all pts are in functional Class I. One pt had a change of its residual mitral regurgitation from grade 1 to grade 2. The other had the same mitral or tricuspid evaluation as at hospital discharge.

We conclude that flexible, externally adjustable rings are safe and provide excellent early results. The external adjustment feature provides the possibility of a significant reduction of the incidence of residual regurgitation after mitral and/or tricuspid annuloplasty repair.

2:45 pm INTERMISSION - VISIT EXHIBITS

*By invitation

3:15 pm SIMULTANEOUS SCIENTIFIC SESSION A ADULT CARDIAC SURGERY

Auditorium, Hynes Convention Center
Moderators: Tirone E. David, M.D.

Bruce A. Reitz, M.D.

23. 1026 PATIENTS UNDERGOING COMBINED

CORONARY AND VALVULAR SURGERY: EARLY AND LATE PHASE EVENTS

Glenn W. Laub, M.D.*, Bridget M. Bailey, B.S.N.*, Chao L. Chen, Ph.D.*, Robert S. Clancy, B.S.N.*, Javier Fernandez, M.D., William A. Anderson, M.D.*, Joseph T. Costic, D.O.* and Lynn B. McGrath, M.D.

Browns Mills, New Jersey

Between January 1981 and December 1991, combined coronary and valve surgery was perfomed in 1,026 patients. Coronary revascularization was combined with aortic valve (AV) surgery in 551 (53.7%), mitral valve (MV) in 354 (34.5%), multiple valve in 119 (11.6%) and isolated pulmonic or tricuspid in 1 (0.2%) each, with valve repair being performed in 21% of procedures. Mean age was 66.7 years (range 31-90 yrs), 61% were male, and 56% were in CHF Class III and IV. There were 117 (11.4%) hospital deaths, with surgery on the MV being the most important risk factor, increasing mortality to 16% as compared to 7% without (p=0.001). CHF, cross clamp time, female gender, and previous surgery were also predictors of mortality, while extent of coronary disease, NYHA Class, and ejection fraction were not. Total follow-up was 5,021 pt-years (mean 5.5, range 1-13.3 yrs) and 99.9% complete. Actuarial survival at 5 and 10 yrs was 70 ± 2% and 42 ± 2% (70% CL), with survival of AV (74 and 48%) significantly longer than MV (67 and 32%, p=0.002). Risk factors for late death included MV surgery, preop NYHA and CHF Class, and liver disease. Survival with a mechanical valve was better than with a tissue valve at 10 yrs for AV (61 vs 34%, p=.0001), but not different for MV. The use of the internal mammary artery to the left anterior descending conferred a survival advantage at 10 yrs (59 vs 42%, p=0.02), while the number of vessels bypassed showed no effect. Of 415 late deaths, 33 (7.9%) were valve related: thromboembolic (TE) 10, prosthetic valve endocarditis 10, anticoagulant bleed (ACRH) 6, structural valve dysfunction (SVD) 6 and paravalvular leak (PVL) 1. Cardiac, non-valve related deaths (229) were comprised predominately of progressive heart failure or myocardial ischemia in 203 patients (89%). Postoperative angina occured in 85 (9.4%) and graft occlusion in 29 (3%) patients. Late survivors fared well as 453 (92%) were in CHF Class I and II. Overall actuarial freedom from complications at 10 years was 86.8% for TE, 84.2% from ACRH, 94.5% for PVL, 88.5% for SVD, 99.6% for valve thrombosis, and 84.1% for MI. Freedom for SVD at 8 yrs was far better with mechanical than tissue (98 v 71%, p=.01) as was freedom for re-operation at 10 yrs (AV:91 v 72%, p=.002; MV:79 v 64%, p=.04). Tissue valves had a superior freedom from ACRH at 5 yrs for AV (94 v 89%, p=.002) and MV (94 v 84%, p=.007), but TE rates were similar. Valve repair was not associated with a reduction in early or late phase events.

We conclude that although late survival was markedly lower than in an age, sex, race matched population (42 v 65%, 10 yr), the survivors' functional results are good. In addition, the use of the IMA and mechanical valve in the aortic position confer substantial long term survival advantages and freedom from reoperation in combined procedures.
*By invitation

24. REVASCULARIZATION OF THE CIRCUMFLEX ARTERY WITH THE PEDICLED RIGHT INTERNAL MAMMARY ARTERY: CLINICAL, FUNCTIONAL AND ANGIOGRAPHIC MIDTERM RESULTS

Michel Buche, M.D.*, Erwin Schroeder, M.D.*, Patrick Chenu, M.D.*, Olivier Gurne, M.D.*, Giulio Pompilio, M.D.* Baudouin Marchandise, M.D.*, Philippe Eucher, M.D.*, Yves Louagie, M.D.*, Robert Dion, M.D.* and Jean-Claude Schoevaerdts, M.D.*

Yvoir, Belgium

Sponsored by: Albert Starr, M.D., Portland, Oregon

Retroaortic crossing of the pedicled Right Internal Mammary Artery (RIMA) for revascularization of the Circumflex Artery (Cx) combined with a pedicled Left Internal Mammary Artery (LIMA) to the Left Anterior Descending Artery (LAD) and its branches is an attractive technique to achieve a complete arterial revascularization of the left ventricle. However there is

a suspicion that pulling the RIMA through the transverse sinus could compromise its blood flow capacity and patency.

Material: Between January 1990 and July 1994, this technique was applied in 256 patients (202 male; 54 female; average age 62 years; range 31 to 80 years). 61 patients had a 2-vessel disease and 195 had a 3-vessel disease. 17 patients were undergoing a reoperation. 22 had a left ventricular ejection fraction <40%. 30 were diabetecs. The RIMA was directed to the Cx artery through the transverse sinus (259 anastomoses) and the LIMA was anastomosed to the LAD and its branches (375 anastomoses) in all patients. 195 patients with a 3-vessel disease received additional coronary artery bypasses to the Right Coronary Artery (93 Saphenous Vein grafts; 89 free Inferior Epigastric Artery; 12 pedicled Right Gastroepiploic Artery). In total, the patients received an average of 3.2 distal anastomoses (2.4 IMA anastomoses).

Clinical results: 3 patients died early and 8 had a nonfatal myocardial infarction (MI). 7 patients required postoperative intraaortic balloon pumping. 6 patients underwent early reoperation for excessive bleeding. Sternal wound complications occurred in 4 patients. One of these 4 patients died of the complication 10 months after the operation. No patient was lost for follow-up (average 28 months). During follow-up, one sudden death and 4 noncardiac deaths occurred. 2 patients experienced a nonfatal MI and 12 had recurrence of angina. There was no late reoperation.

Graft patency and functional assessment: 73 patients enrolled in a prospective angiographic study agreed to undergo a postoperative angiogram (average 12.6 months, range 6 to 24 months). 72/73 of the RIMA were patent. In comparison 73/73 of the LIMA (104/105 LIMA anastomoses) were patent. Stress Thallium Scintigraphy obtained in 25 of those patients did not reveal ischemia in the Cx area.

Conclusions: Complete arterial revascularization of the left ventricle by means of both pedicled IMA can be performed with acceptable mortality and morbidity.

The midterm patency rate of the pedicled RIMA passed through the transverse sinus and anastomosed to the Cx artery is excellent and does not differ of the patency rate of the LIMA anastomosed to the LAD.

*By invitation

25. RISK FACTORS FOR POSTOPERATIVE MORBIDITY

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

Albany, New York

Sponsored by: L. Henry Edmunds, Jr., M.D., Philadelphia, Pennsylvania

Background: Analysis of outcomes after coronary artery bypass grafting (CABG) has focused on risk factors for operative motality. Non-fatal peri-operative morbidity is far more costly and more common after CABG and the factors that predispose to morbid events are not well documented. In order to identify the risk factors that lead to postoperative morbidity, we evaluated 938 patients undergoing CABG at our institution during 1993.

Methods: Univariate statistical analysis was performed on 46 patient variables in order to identify risk factors for either serious postoperative morbidity (stroke, perioperative Q-wave myocardial infarction, deep sternal infection, life-threatening postoperative hemorrhage) or increased hospital length-of-stay (LOS). Variables were considered both individually and in combination. For example, age was considered individually or in combination with other variables including parameters of blood volume (i.e., age divided by red blood cell volume or Age/RBCVOL), renal function, cardiac function, etc. Univariate analysis of these 46 variables revealed 16 that were significant risk factors for serious postoperative morbidity or increased LOS. These 16 variables were entered into multivariate analyses to determine independent risk factors for serious postoperative morbidity (logistic regression) or increased LOS (Cox proportional hazards regression). Similar multivariate analysis was performed to identify independent risk factors for hospital mortality (logistic regression).

Results: Hospital length-of-stay (LOS) was correlated with serious postoperative morbidity (Spearman rank correlation coefficient = -0.31, $p<0.001$) but not mortality (correlation coefficient = 0.03, $p=0.38$). In order of decreasing importance, the following patient variables were significantly associated with increased LOS by stepwise Cox proportional hazards analysis: Age/RBCVOL ($p<0.001$), history of CHF ($p<0.001$), CPB time ($p<0.001$), hypertension ($p=0.004$), chronic obstructive lung disease ($p=0.002$), and previous stroke ($p=0.041$). In order of decreasing importance the following patient variables were significantly associated with the occurrence of any serious postoperative morbidity by logistic regression: history of CHF ($p<0.001$), Age/RBCVOL: ($p=0.001$), hypertension ($p=0.004$), CPB time ($p=0.003$), and previous stroke ($p=0.03$). The ratio of age divided by the RBCVOL (blood volume multiplied by hematocrit) was an important risk factor for both increased LOS and serious postoperative morbidity. Interestingly, variables that were significant independent predictors of increased mortality such as, preoperative shock, previous open heart operation, renal failure, ejection fraction less than 20%, and MI within 6 hours of operation, were not risk factors for either serious morbidity or increased LOS. Using the variables and relative risk values identified in this analysis, it is possible to develop a scoring system that assigns morbidity risk scores to patients before operation.

Conclusions: We conclude that: 1) hospital length-of-stay is a continuous variable that correlates with postoperative morbidity but not with mortality, 2) advanced age and low red blood cell volume are very important risk factors for serious postoperative morbidity and increased LOS, and 3) risk factors for postoperative morbidity are different than those for postoperative mortality. These results suggest that older patients with preoperative anemia and low blood volume, who also have other co-morbidities (CHF, stroke, COPD or hypertension), are at increased risk for postoperative complications. These studies allow identification of a high-risk cohort of patients who are likely candidates for interventions to lessen postoperative morbidity.

*By invitation

26. IS BODY SIZE THE CAUSE FOR POOR OUTCOMES OF BYPASS SURGERY IN FEMALES?

George T. Christakis, M.D., Karen Buth, M.Sc.*, Richard D. Weisel, M.D., Vivek Rao, M.D.*, Kostas P. Panagiotopoulos, B.Sc.*, Joan Ivanov, R.N.* and Stephen E. Fremes, M.D.,

Toronto, Ontario, Canada

Females have a higher incidence of operative mortality (OM), low output syndrome (LOS), myocardial infarction (MI), postoperative bleeding (BLD), and infection (INF) than males (M) following CABG. The poor outcomes have been attributed to the smaller size of females and (by extension) to smaller coronary diameter. In order to assess risk factor differences between males and females, and the influence of body size on postoperative outcome, data were gathered prospectively on 7,025 patients (M=5,694, F=l,331) proceeding to isolated CABG between January 1990 and June 1994. Univariate and multivariate statistics were used to determine risk factors for CABG in M and F separately. Body size was assessed by both body mass index (BMI) and body surface area (BSA).

		% OM		% LOS		% MI		% BLD		% INF	
BMI ($kg/m^{2)}$	"pts"	M	F	M	F	M	F	M	F	M	F
<20	125	3.5	7.5	13.0‡	20.0	3.5	5.1	3.5‡	2.5	2.3‡	15.0
20-25	1700	2.5	3.8	8.0	15.7*	3.2	5.8*	2.2	2.0	2.3	3.5
25-30	3169	1.8	3.9*	6.3	14.3*	2.8	5.5*	1.3	2.4*	3.5	5.1
>30	2031	1.6	3.1*	5.1	12.1*	2.8	5.3*	1.0	2.4	6.3	9.9*

*=M diff than F p<0.05 ‡=<20 diff than >30, p<0.05 for M

Small body size (BMI<20) resulted in increased morbidity postoperatively for both sexes. However, within each classification of body size females continued to have a higher morbidity and mortality. In patients with heights less than 164 cm, the incidence of OM (M=2.2%, F=3.7%) and LOS (M=7.9%, F=14.2%) was higher (p<0.05) for females. In patients with BSA<1.7 m^2, the incidence of OM (M=3.8%, F=3.8%) and LOS (M=l 1.4%, F=15%) were similar for both sexes. BSA<1.7m^2 accounted for 47% of F but only 8% of M. For patients with BSA>1.7m2, OM (M=1.7%, F=3.8%) and LOS (M=6.3%, F=14%) were higher (p<0.0001) in females. The multivariate risk factors (odds ratios) for OM in M were: age>70 (2.7), LVEF<40% (2.6), diabetes mellitus (DM) (1.6), peripheral vascular disease (PVD) (2.3), smoking (1.9), renal failure (2.5), preoperative MI (PMI) (2.0) and for F the predictors were: age>70 (2.0), urgent surgery (US) (2.5), LVEF<40% (3.2), PVD (2.8). The predictors of LOS in M were: age>70 (1.6), LVEF<40% (2.2), reoperative surgery (REDO) (5.6), left main stenosis (LMS) (1.3), endarterectomy (EA) (1.8), DM (1.6), congestive failure (2.1), PMI (2.0). LOS predictors for F were: US (1.6), LVEF<40% (2.5), LMS (1.7), REDO (4.2), EA (3.2).

CONCLUSION: Risk factors for OM and LOS were similar for males and females. Small body size increases morbidity following isolated CABG. Independent of body size, females still have a higher operative mortality and morbidity. Increased risk of surgery for F may be due to differences in atherogenesis.

4:35 pm EXECUTIVE SESSION (Limited to Members)

Republic Ballroom, Sheraton Boston Hotel

6:30 pm MEMBER RECEPTION

Museum of Fine Arts

*By invitation

1:45 pm SIMULTANEOUS SCIENTIFIC SESSION B GENERAL THORACIC SURGERY

Republic Ballroom, Sheraton Boston Hotel
Moderators: J. Kent Trinkle, M.D.

Valerie W. Rusch, M.D.

27. MUTATION IN THE P53 TUMOR SUPPRESSOR GENE IN BARRETT'S ESOPHAGUS IS A MARKER FOR DEVELOPMENT OF ADENOCARCINOMA: RESULTS OF AN INTERNATIONAL MULTI-INSTITUTIONAL PROSPECTIVE STUDY

Paul M. Schneider, M.D.*, Alan G. Casson, M.D.*, Harinder S. Garewal, M.D.*, Bernard Levin, M.D.*, A.H. Hoelscher, M.D.*, J. Rudinger Stewart, M.D.* and Jack A. Roth, M.D.

Munich, Germany, Toronto, Ontario, Canada,

Houston, Texas and Tucson, Arizona

Current histologic criteria are not adequate to predict which patients with Barrett's esophagus (BE) are at high risk of developing invasive cancer and thus are candidates for esophagectomy. The purpose of this study was to determine the value of p53 mutation in BE as a marker for the development of adenocarcinoma. The p53 gene, a tumor suppressor gene whose function is critical to cell cycle control and DNA repair, is the most commonly mutated gene in human cancers. We had previously identified p53 mutations in BE and therefore began a multi-institutional study to determine their significance as a marker for malignancy. Ninety-seven patients from four institutions were studied. Forty-seven (37 males and 10 females, mean age 55) had BE with metaplasia or dysplasia but no evidence of malignancy at a mean follow-up of 2.2 ± 0.2 years. The BE was classified as metaplasia in 30, low-grade dysplasia in 12, and high-grade dysplasia in 5. The other 50 patients (46 males and four females, mean age 63) had adenocarcinoma arising in BE (BC). Tissue for DNA analysis, including normal stomach or esophagus, tumor, and BE, was obtained by endoscopic biopsy in patients with BE or BC or during surgery in some patients with BC. Exons 5-9 of the p53 gene were studied for mutations by single-strand conformational polymorphism analysis (SSCP) after polymerase chain reactions. Mutations detected by SSCP were confirmed by direct DNA sequencing. None of the tissue samples from patients with BE alone with metaplasia or low-grade dysplasia had a p53 mutation, and only one of five patients with high-grade dysplasia but no evidence of invasive malignancy had a p53 mutation. However, 23 of the BC patients (46%) had a p53 mutation in Barrett's epithelium, tumor, or both. Twenty of these had a p53 mutation in the tumor only (N=16) or in both (N=4), suggesting a direct role for the mutation in carcinogenesis. In the other three, a mutation was present in the Barrett's epithelium but not the tumor, suggesting that p53 mutation may be a marker for genetic instability predisposing to tumor formation. Biopsies of BE were classified as metaplasia or low-grade dysplasia for 16 patients with a p53 mutation and cancer, indicating that p53 mutation is associated with cancer regardless of the degree of clinically detectable dysplasia. We conclude that mutation in the p53 gene identifies a subset of BE patients at increased risk for developing cancer.
*By invitation

28. ERYTHROMYCIN STIMULATES GASTRIC EMPTYING STATUS POST-ESOPHAGECTOMY: A RANDOMIZED CLINICAL TRIAL

Michael Burt, M.D., Ph.D., William Williard, M.D.*, Andrew Scott, M.D.*, Samuel Yeh, M.D.*, Manjit Bains, M.D., Allan Turnbull, M.D.*, Joseph Former, M.D.*, Patricia McCormack, M.D. and Robert Ginsberg, M.D.

New York, New York

Delayed gastric emptying following esophagogastrectomy can pose a significant early post-operative problem. Since erythromycin, which stimulates the gastric antral and duodenal motilin receptor, has been demonstrated to significantly increase gastric emptying in patients with diabetic gastroparesis, we decided to evaluate its effect on gastric emptying after esophagogastrectomy.

Methods: 24 patients (age: 41-79 yr, median 66; 18 men/6 women) were randomized to receive either erythromycin lactobionate (200 mg in 50ml NSIV, n=13) or placebo (50 ml NS IV, n=11) eleven days after esophagogastrectomy (with pyloric drainage procedure). After infusing erythromycin or placebo over 15 min, patients ate a solid meal (scrambled egg with bread) labelled with [99^{m}Tc] sulfur colloid (500 |aCi) over approximately 15 min. Dynamic images of the post-surgical stomach were then acquired over 90 min in the supine position by gamma counter. Results were expressed as percentage of counts retained in the stomach (% gastric retention) over time.

Results: There were no side effects of eryhromycin. The figure displays the gastric emptying over time for both groups.

Analysis of covariance demonstrated that the rate of gastric emptying (slope of the line) was significantly greater in the erythromycin treated group compared to the placebo group ($p<0.0001$).

Conclusion: Early satiety following esophagogastrectomy is secondary to decreased gastric motility and not secondary to a decrease in the gastric reservoir. Intravenous erythromycin significantly improves gastric emptying in patients following esophagogastrectomy by stimulating gastric motility.

*By invitation

29. THE ROLE OF FUNDOPLICATION IN THE TREATMENT OF TYPE II PARAESOPHAGEAL HERNIA

Clark B. Fuller, M.D.*, Jeffrey H. Peters, M.D.*, Manfred Ritter, M.D.*, Cedric G. Bremner, M.D.* and Tom R. DeMeester, M.D.

Los Angeles, California

The role of fundoplication in patients with pure Type II paraesophageal hiatal hernia remains controversial. Conventional thinking suggests that because the lower esophageal sphincter (LES) is located within the abdomen, it is competent, and fundoplication unnecessary. Few studies have used objective evaluation to guide the addition of an antireflux procedure.

Patients and Methods: Sixteen consecutive patients with Type II paraesophageal hernia were treated from May 1991 to July 1994. All demonstrated radiographic criteria of pure Type II hernias. Evaluation before surgery included upper endoscopy, esophageal manometry, and 24-hour ambulatory pH monitoring. The lower esophageal sphincter was considered incompetent if any of the following criteria were present; a resting pressure <6 mmHg, abdominal length <1 cm or total length <2 cm. Primary symptoms responsible for surgery were related to the hernia in 81% of patients postprandial pain (n=7), abdominal distension (n=5), vomiting (n=1), and anemia (n=0). Symptoms typical of gastroesophageal reflux disease were noted in 3 patients heartburn in 2 and regurgitation in 1.

Results: Objective evidence of gastroesophageal reflux disease was present in the majority of patients. Five patients (31%) had evidence of esophageal mucosal injury: esophagitis in 3, stricture and esophageal ulceration in 1 patient each. Eleven of 15 patients (69%) were found to have pathologic esophageal acid exposure on 24-hour pH monitoring. Twelve (75%) patients had a defective LES usually secondary to an inadequate intra-abdominal length (8/12, 66%). Hernia reduction, crural closure and Nissen fundoplication was performed in 14/16 patients (2 patients await definitive surgery). Symptomatic relief has been excellent/good in all patients. No patients has developed hernia recurrence at an average follow-up of 8.8 months (range 2-28 months).

Conclusion: Objective evaluation reveals that gastroesophageal reflux accompanies type II paraesophageal hernia in a high proportion of patients, usually secondary to an incompetent LES. Appropriate treatment includes reduction of the hernia, crural closure and fundoplication in most if not all patients.

2:45 pm INTERMISSION - VISIT EXHIBITS

*By invitation

3:15 pm SIMULTANEOUS SCIENTIFIC SESSION B GENERAL THORACIC SURGERY

Republic Ballroom, Sheraton Boston Hotel
Moderators: J. Kent Trinkle, M.D.

Valerie W. Rusch, M.D.

30. OBJECTIVE ASSESSMENT OF GASTROESOPHAGEAL REFLUX AFTER SHORT ESOPHAGOMYOTOMY FOR ACHALASIA USING MANOMETRY AND PH MONITORING

John M. Streitz, Jr., M.D.*, F. Henry Ellis, Jr., M.D., Ph.D., Warren A. Williamson, M.D.*, Michael E. Glick, M.D.*, Johannes A. Aas, M.D.* and Robert L. Tilden, Dr.P.H.*

Duluth, Minnesota and Burlington, Massachusetts

The role of an antireflux procedure as an adjunct to esophagomyotomy for achalasia remains controversial. In our experience, a short myotomy alone is followed by a low incidence of severe reflux symptoms (4-5%). However, little objective documentation exists of this operation's effect

upon sphincteric competence and the degree of postoperative gastroesophageal reflux. This report provides such documentation.

We performed esophageal manometry and 24 hour pH monitoring on 14 patients with esophageal achalasia who had previously undergone a short esophagomyotomy without antireflux procedure by us. Eight of the 14 were tested to evaluate the postoperative symptom of heartburn, and the remaining six asymptomatic patients were studied to assess postoperative sphincteric function.

Esophagomyotomy reduced lower esophageal sphincter (LES) pressure by 12 to 71% (mean 41%) from a preoperative mean of 27 mmHg to 14 mmHg postoperatively. The number of postoperative acid reflux episodes per patient in 24 hours was below 29 (normal <47) in 12 patients, with a median of 12 episodes for the entire group. Esophageal acid exposure, measured as % total time with pH <4.0 (normal <4.5%), was below 4.5% in 9 patients, 6 of whom had values less than 1%. Of the five patients with values greater than 4.5%, a temporal correlation of symptoms with an episode of acid reflux occurred in only one. Multivariate analysis showed that esophageal acid exposure time correlated only with the level of residual LES pressure during the relaxation phase of deglutition. A pressure less than 8 mmHg was predictive of normal acid contact time ($p<.018$). Age greater than 42 was a covariant and predictive of normal acid exposure by predicting low residual pressure ($p<.001$), irrespective of follow-up duration. Mean LES pressure, percent reduction in LES amplitude, postoperative vector volume and length of the LES did not significantly correlate with amount of esophageal acid exposure.

We conclude that a short esophagomyotomy without an antireflux procedure results in a competent LES in most patients, and that symptomatic gastro-esophageal reflux is unusual. Increased esophageal acid exposure, when it occurs, is due to slow esophageal acid clearance of relatively few reflux episodes, not from too extensive a myotomy, and is more likely to occur when the myotomy results in a high residual pressure during deglutition, a finding more common in young patients. In view of these findings, the addition of an antireflux procedure to a short esophagomyotomy would not be expected to improve results.
*By invitation

31. COMBINED THORACOSCOPIC AND LAPAROSCOPIC LYMPH NODE STAGING IN ESOPHAGEAL CANCER: THE UNIVERSITY OF MARYLAND EXPERIENCE

Mark J. Krasna, M.D.*, John L. Flowers, M.D.*, Safuh Attar, M.D. and Joseph S. McLaughlin, M.D.

Baltimore, Maryland

Unlike mediastinoscopy in lung cancer, there exists no standard minimally invasive test to stage esophageal cancer. If it were possible to obtain exact preoperative staging in esophageal cancer, patients could be separated prospectively to receive adjuvant therapy appropriately. We studied the feasibility and efficacy of thoracoscopic lymph node staging (TSLN) and laparoscopic lymph node staging (LSLN) in esophageal cancer. TSLN was performed in 45 patients with biopsy proven carcinoma of the esophagus. LSLN was done in the last 19 pts (after missing celiac LN in 3 pts). TSLN was aborted in 3 pts due to adhesions. LN stage was NO in 39 pts, N1 in 3 and N3 (celiac LN) in 6 patients. Esophageal resection was performed in 30 patients after TSLN; 17 of these underwent LSLN. These patients form the basis for subsequent data analysis.

TSLN staging showed NO lymph node status in 28 patients and N1 in 2 patients. Two of the 28 NO patients (7%) were found at resection to have para-esophageal lymph node involvement (N1) and were thus understaged by TSLN. One patient with Nl disease at thoracoscopy had no lymph node involvement (NO) after esophagectomy. The other patient remained N1 at the time of resection. Thus TSLN was accurate in detecting the presence of thoracic LN in 28/30 cases (93% accuracy).

LSLN staging found NO disease in 12 pts and N3 in 5 pts. After esophagectomy, final pathology of the 12 NO patients was NO in 11 and N3 in one patient. LSLN missed one pt with N3 disease. Final pathologic staging of the 5 N3 patients revealed N3 status in 2 patients and NO status in 3 patients. Thus, LSLN was accurate in detecting lymph node metastases in 16/17 patients (94%).

TSLN/LSLN staging are more accurate than existing staging methods. 6/18 LSLN pts had unsuspected celiac axis LN involvement missed by standard noninvasive techniques. 3% of thoracic LN and 17% of celiac LN were downstaged after preoperative chemo/radiotherapy. LN near the diagphragmatic hiatus may be difficult to sample without extensive dissection. The role of TSLN/LSLN in staging esophageal cancer should be further evaluated in a multi-institutional trial.

*By invitation

32. CATASTROPHIC COMPLICATIONS ASSOCIATED WITH CERVICAL ESOPHAGOGASTRIC ANASTOMOSIS

Mark D. Iannettoni, M.D.*, Richard I. Whyte, M.D.* and Mark B. Orringer, M.D.

Ann Arbor, Michigan

Recent enthusiasm for the cervical esophagogastric anastomosis (CEGA) has arisen because of its perceived low morbidity. While catastrophic complications of a CEGA are unusual, they can and do occur, and prevention is possible if the potential for them is recognized. In 866 patients undergoing a CEGA after transhiatal esophagectomy, catastrophic complications associated with anastomotic disruption have occured in 12 patients (1.4%): vertebral body osteomyelitis (1); epidural abscess with neurologic impairment (2); pulmonary microabscesses from internal jugular vein abscess (1); tracheogastric fistula (1); and major dehiscence requiring anastomotic takedown (7). These complications became manifest from 8 to 85 days after the esophageal resection and reconstruction (mean 23 days). Leakage from a suspension stitch placed in the anterior spinal ligament over the vertebral bodies resulted in a posterior gastric leak and either osteomyelitis or an epidural abscess in three patients, none of whom had evidence of extravasation on their routine 10 day postoperative barium swallow. Cervical exploration for a presumed anastomotic leak lead to the unexpected discovery of an abscess formed by the stomach and the adjacent wall of the internal jugular vein which was ligated and resected. One asymptomatic patient who was discharged with a contained anastomotic leak on his postoperative barium swallow was readmitted 7 days later with a cervical tracheogastric fistula from which he ultimately died. In 7 patients, 9% of all who have experienced anastomotic leaks, there was sufficient gastric ischemia and/or necrosis to require takedown of the anastomosis and intrathoracic stomach with a plan for later reconstruction. As a result of this experience, it is recommended that cervical gastric suspension sutures either be omitted entirely or placed in the fascia over the longus coli muscles anterior to the spine but not directly into the prevertebral fascia overlying the vertebral bodies or

cervival discs. All cervical anastomotic leaks, even if apparently contained, are best drained rather then treated expectantly. Patients who remain febrile and ill after bedside drainage of a leak should undergo cervical reexploration in the operating room; major gastric ischemia and/or necrosis may warrant takedown of the anastomosis and intrathoracic stomach. Refinement of any operation frequently evolves from complications associated with previous experience. With an awareness of their possibility and with minor technical modifications, the incidence of these catastrophic complications and their morbidity can be reduced.

*By invitation

33. DELAYED PRIMARY REPAIR OF ESOPHAGEAL PERFORATION: IS IT SAFE?

Nan Wang, M.D.*, Ali Safavi, M.D.*, Anees J. Razzouk, M.D.*, Karen Gan, M.D.*, Arthur C. Hill, M.D.*, Bryan Fandrich, M.D.*, Michael J. Wood, M.D.*, Edwin E. Vyhmeister, M.D.*, Changwoo Ahn, M.D.* and Steven R. Gundry, M.D.

Loma Linda and Fontana, California

The management of esophageal perforation with delayed diagnosis (>24 hrs) is controversial. Because of the obvious advantages of primary repair as a simple single-stage operation, this technique was preferentially used to treat 17 of 21 consecutive esophageal perforations. Patients were stratified into three groups according to the time elapsed between perforation and operation:

	n	Age (yrs)	Delay (hrs)	Iatrogenic Perforation	Preop Sepsis
Grp A - <6 hrs	5	77 ± 12	5.2 ± 0.8	4 (80%)	0 (0%)
Grp B - <24 hrs	6	59 ± 12	13.8 ± 2.9	2 (33%)	2 (33%)
Grp C - >24 hrs	6	62 ± 12	46.0 ± 12.8	3 (50%)	3 (50%)

In addition to primary repair in these pts, additional coverage of the sutured site was performed using a fundic wrap in 7 pts (41%), pericardial fat in 2 pts (12%), diaphragmatic flap in 2 pts (12%), pleural patch in 1 pt (6%), and intercostal pedicle in 1 pt (6%). Results:

	n	Leaks	Sepsis	MOF	Mortality
Grp A	5	0 (0%)*	2 (40%)	0 (0%)	1 (20%)
Grp B	6	4 (67%)	4 (67%)	1 (17%)	1 (17%)
Grp C	6	4 (67%)	5 (83%)	2 (33%)	1 (17%)

*p<0.03

In Group C, the only death occurred in a profoundly septic pt whose diagnosis was delayed by more than 2 weeks. Esophageal continuity was maintained in all of the 14 (82%) survivors. We conclude that in the era of advanced intensive-care capabilities, delayed primary repair of esophageal perforation in the properly selected pt can lead to a satisfactory outcome. Although an esophageal leak is common unless immediate repair of the perforation is performed, the leak does not necessarily lead to an adverse outcome.

4:35 pm EXECUTIVE SESSION (Limited to Members)

Republic Ballroom, Sheraton Boston Hotel

6:30 pm MEMBER RECEPTION

Museum of Fine Arts

*By invitation

1:45 pm SIMULTANEOUS SCIENTIFIC SESSION C CONGENITAL HEART DISEASE

Independence Ballroom, Sheraton Boston Hotel
Moderators: Richard A. Hopkins, M.D.

Richard A. Jonas, M.D.

34. APPLICATION OF COMPUTATIONAL FLUID

DYNAMICS TO THE STUDY OF COMPETITIVE FLOWS IN CAVOPULMONARY CONNECTIONS

‡Marc Roger de Leval, M.D., FRCS, Gabriele Dubini, Ph.D.*, Francesco Migliavacca, Ph.D.*, Homayoun Jalali, M.D.*, Riccardo Pietrabissa, Ph.D.* and Andrew Redington, M.D.*

London, England

Computational fluid dynamics (CFD) based on a finite element method were applied to the study of (i) competition of inferior (IVC) and superior vena caval flows (SVC) in the total cavopulmonary connection (TCPC) and (ii) competition between SVC flow and forward flow from a stenosed pulmonary artery (PA) in the bidirectional cavopulmonary anastomosis (BCPA). 3-D parametric models of the connections were created from angiocardiograms and the fluid dynamics (pressure and velocity) were computed by a fluid dynamic computational code.

1. TCPC: 12 models corresponding to various degrees of offsetting and shape of the IVC-PA anastomosis were simulated to evaluate energy dissipation and flow distribution between the two lungs. A minimal energy loss was found with an offset value close to zero and the enlargement of the IVC anastomosis towards the right PA was shown to divert more IVC flow to the right lung, thus distributing more caval flow to the bigger lung (QLPA/QRPA = 0.75).

2. BCPA: The wisdom of leaving forward flow from a stenosed PA while constructing a BCPA remains controversial. 98 such operations were performed between January 1988 and September 1994. Nine patients (9%) had postoperative SVC hypertension (18 mmHg or more), of whom five were reoperated. A computational model of the operation was developed in an attempt to predict postoperative haemodynamics. In tight PA stenosis (75% or more) the non-pulsatile forward QP is primarily directed to the LPA with little influence on the SVC pressure. A pulsatile forward flow through the PA corresponding to 15, 30, 50 and 75% of the systemic cardiac output increases the mean PA and SVC pressure respectively by 1.0, 1.7, 2.4 and 3.6 mmHg.

Inferences:

1. CFD has been applied clinically to refine the design of the TCPC. A small triangular patch is now placed on the right lateral aspect of the IVC pathway to reduce energy loss and achieve a more physiological distribution of flow between the two lungs.
2. In BCPA it is safe to maintain forward flow so as to have an overall systemic to pulmonary flow ratio of 1, thus improving the systemic arterial saturation without excessive SVC hypertension.

‡1973-74 Graham Fellow

*By invitation

35. ATRIAL FLUTTER INDUCED BY LATERAL TUNNEL CONSTRUCTION IN THE MODIFIED FONT AN OPERATION

Mark D. Rodefeld, M.D.*, Hurt I. Bromberg, M.D.*, Charles B. Huddleston, M.D.*, Richard B. Schuessler, Ph.D.*, John P. Boineau, M.D.* and James L. Cox, M.D.

St. Louis, Missouri

The cavopulmonary connection, or modified Fontan operation, utilizes a lateral tunnel through the right atrium. Intra-atrial reentry or atrial flutter (AFL) is a relatively common post-operative problem. We postulate that the lateral tunnel suture line establishes the anatomic substrate for AFL in these patients. The purposes of this study were 1) to determine if the lateral tunnel suture line alone is sufficient to permit initiation of AFL and 2) to map any resultant arrhythmias.

After induction of general anesthesia, 30-35 kg canines (n=6) underwent median sternotomy, cradling of the pericardium, and placement of a pacing electrode on the right atrial appendage. Normothermic cardiopulmonary bypass was initiated. Through a right atriotomy, a sham modified Fontan operation was performed. A suture line (no baffle) was placed beginning at the fossa ovalis, around the IVC, up the crista terminalis, around the SVC, and back to the fossa ovalis, simulating lateral tunnel construction. After closure of the atriotomy, bi-atrial 256 point form-fitting endocardia! electrodes were transannularly positioned in the atria via bilateral ventriculotomies. Atrial burst pacing and programmed extrastimulation were performed before and after placement of the lateral tunnel suture line.

AFL was non-inducible in 6/6 dogs prior to suture line placement. After suture line placement, sustained AFL was repeatedly inducible in all cases (100%), although in one case isoproterenol was required. The mean flutter cycle length was 196±77 msec. Activation sequence mapping revealed that the suture line was integral to the AFL pathway in all cases (100%). In each case, the AFL circuit revolved in the right atrium where slow conduction and unidirectional block occurred at the suture line. In no instance did the flutter pathway revolve around the atriotomy site alone.

We conclude that the baffle suture line used in the modified Fontan operation, without any alteration in circulatory physiology, is sufficient to create the substrate for AFL. A more complete understanding of the reentrant circuit may permit use of prophylactic measures such as incisional modification or placement of cryolesions that create bi-directional block, thereby inhibiting AFL in patients undergoing the modified Fontan operation.

*By invitation

36. STAGED OPERATION TO FONT AN INCREASES THE INCIDENCE OF SINO-ATRIAL NODE DYSFUNCTION

Peter B. Manning, M.D.*, John E. Mayer, Jr., M.D., Gil Wernovsky, M.D.*, Steven B. Fishberger, M.D.* and Edward P. Walsh, M.D.*

Kansas City, Missouri and Boston, Massachusetts

Morbidity and mortality of total cavo-pulmonary connection (modified Fontan procedure) is decreased in many high-risk single ventricle patients by performing the operations in a staged fashion. Some have advocated routine use of the bidirectional Glenn or "hemi-Fontan" procedure in all patients with single ventricle physiology. Each operative intervention, however, exposes the sino-atrial (SA) node region to risk of injury. A multi-stage approach may increase the risk of SA node dysfunction in patients that may not tolerate such a dysrhythmia or atrio-ventricular dysynchrony.

All patients completing a Fontan procedure between January 1988 and December 1992 were reviewed. Of 324 total patients, 227 completed the Fontan in a single operation, and 97 followed a two-stage approach. The mean age at Fontan completion was not different between groups (5.3 vs 4.6 yrs, p=.21). Overall survival was the same for both groups (91%).

The incidence of transient and fixed SA node dysfunction was similar for both groups following the first operative intervention despite a heterogeneous patient population (1 stage: 14%/7%, 2 stage: 12%/3%, p=28). Second operative intervention resulted in a higher incidence of dysrhythmia (transient: 29%, fixed: 23%), and more frequent SA node dysfunction on follow-up (1 stage: 11%, 2 stage: 46%, p<.002). In the two-stage group, 49% of patients without arrhythmia following first intervention experienced an arrhythmia after the second intervention, while of those with an arrhythmia following first operation, 67% experienced one at second intervention (p<.01). Despite these findings, most patients with SA node dysfunction tolerated it well clinically, with few patients requiring permanent pacing for atrial arrhythmias (1 stage: 3%, 2 stage: 7%, p=18).

In conclusion, a multi-staged operative pathway to Fontan reconstruction is associated with a higher early risk of SA node dysfunction. Longer follow-up is needed to assess the full impact of this finding. The possibility of this complication must be weighed carefully against factors increasing the risk of a single-staged approach.

2:45 pm INTERMISSION - VISIT EXHIBITS

*By invitation

3:15 pm SIMULTANEOUS SCIENTIFIC SESSION C CONGENITAL HEART DISEASE

Independence Ballroom, Sheraton Boston Hotel
Moderators: Richard A. Hopkins, M.D.

Richard A. Jonas, M.D.

37. CLINICAL EXPERIENCE WITH REPAIR OF

CONGENITAL HEART DEFECTS USING ADJUNCTIVE ENDOVASCULAR DEVICES

John G. Coles, M.D., Jeanne M. Lukanich, M.D.*, Jean Perron, M.D.*, Greg J. Wilson, M.D.*, Marlene Rabinovitch, M.D.*, David G. Nykanen, M.D.*, Lee N. Benson, M.D.*, Ivan M. Rebeyka, M.D.*, George A. Trusler, M.D., Robert M. Freedom, M.D.* and William G. Williams, M.D.

Toronto, Ontario, Canada

The use of intravascular devices as an adjunct in the repair of congenital heart anomalies represents a novel but unproven therapeutic approach. Intra-operative occlusion of an apical muscular ventricular septal defect (VSD) using a clamshell device from the right atrial approach was accomplished in 4 patients. One patient died following associated aortic arch reconstruction as a result of a hypoplastic left ventricle (LV). The results in the remaining 3 patients were favourable based on absence of late residual shunting, LV dysfunction or arrhythmia. Intraoperative implantation of pulmonary arterial stent (5-15 mm expanded diameter) was performed in 15 patients (unilateral 8; bilateral 7) during repair of neonatal pulmonary atresia VSD (n=4), obstructed pulmonary arterial confluence following truncus repair (n=3), bidirectional cavopulmonary shunt (n=2), Fontan procedure (n=2), and miscellaneous pulmonary arterial stenoses (n=5). The endovascular stents were effective at achieving immediate patency in all patients. There were 2 early deaths. Early reoperation was required in each of the 3 survivors of neonatal stent implantation due to bilateral, obstructive neointimal hyperplasia at 3, 10 and 11 months postoperatively. Intraoperative stent implantation was used at the site of obstructed pulmonary venous drainage in 4 patients. Lethal recurrent intraluminal obstruction occurred in all 4 patients, evident histologically as smooth muscle cell proliferation and extracellular matrix deposition. This initial experience supports continued application of intraoperative deployment of endovascular devices for closure of muscular VSD's otherwise inaccessible from the right atrial approach, and for cases of pulmonary arterial obstruction within larger calibre pulmonary arteries. Recurrent obstruction due to a proliferative healing response appears to be an eventual certainty in currently designed small diameter endovascular stents.
*By invitation

38. MANAGEMENT OF TETRALOGY OF FALLOT WITH PULMONARY ATRESIA AND DIMINUTIVE PULMONARY ARTERIES

Francis D. Pagani, M.D, Ph.D.*, John P. Cheatham, M.D.*, Robert H. Beekman, III, M.D.*, Thomas R. Lloyd, M.D.*, Ralph S. Mosca, M.D.* and Edward L. Bove, M.D.

Ann Arbor, Michigan and Omaha, Nebraska

Since September 1991, 14 consecutive patients with tetralogy of Fallot, pulmonary atresia, and diminutive pulmonary arteries (PA) have undergone staged repair. All patients had multiple aortopulmonary collateral arteries and the ductus arteriosus was absent in 12. Mean size of the right and left PA's was 2.3±1.0 mm and 2.0±0.8 mm, respectively (range, 1-3 mm). Six patients (42%) have gone on to complete repair (CR). Age at initial procedure (BT shunt [1], direct aorta-PA anastomosis or conduit [3], RV-PA conduit [2]) was 6.0±7.7 months. The number of operative procedures to achieve CR was 3.3±1.6 per patient (range, 2-6). Post bypass RV/LV was 0.5±0.1. Most patients required one or more interventional catheterizations for balloon dilatation of peripheral stenoses and/or stents in addition to collateral embolization. Mean follow-up in this group was 7.4±8.4 months (range, 0.5 to 20.3 months) and was 100% complete. There were 2 late deaths secondary to neurological complications in one and following reoperation for RV to PA conduit stenosis in the other. Eight patients are awaiting further surgery. Age at initial procedure was 22.2±57.2 months and follow-up was 8.5±8.0 months. Initial palliative procedures included unifocalization (1), direct ascending aorta-PA anastomosis (3), RV to PA conduit (3), and transannular RV outflow patch (1). One patient has had 2 additional procedures (RV to PA conduit and unifocalization). Operative mortality was 25% (2 of 8 patients). Six of 8 patients are alive and awaiting further intervention or repair.

Of the total of 4 deaths in this series, 3 occurred in patients undergoing shunts or unifocalization as the initial procedure (3/5, 60%). Among these 5 patients, only 1 is alive with a

CR. There was only 1 death among 9 patients (11%) receiving RV to PA conduits (5) or direct ascending aorto-PA anastomosis (4) as the intial operative procedure. Three of these 9 patients have gone on to complete repair with no deaths.

This experience suggests that CR is feasible even in patients with extremely diminutive PA's (<3 mm) and may be achieved by early (3-6 month) establishment of central PA flow by RV to PA conduit (PA's > 1.5 mm) or direct ascending aorta-PA anastomosis (PA's < 1.5 mm). Subsequent interventional catheterization and additional operative procedures as required for further PA stenoses and collateral embolization allow continued recruitment of central PA's and may obviate or minimize the need for unifocalization procedures.

*By invitation

39. PH-STAT COOLING IMPROVES CEREBRAL METABOLIC RECOVERY AFTER CIRCULATORY ARREST IN INFANTS WITH AORTOPULMONARY COLLATERALS

Paul M. Kirshbom, M.D.*, Lynne A. Skaryak, M.D.*, Louis R. DiBernardo, M.D.*, Frank H. Kern, M.D.*, William J. Greeley, M.D.*, J. William Gaynor, M.D.* and Ross M. Ungerleider, M.D.

Durham, North Carolina

The presence of aortopulmonary collateral arteries (APCA's) in infants with congenital heart disease has been associated with an increased incidence of neurologic injury following deep hypothermic circulatory arrest (DHCA). This may be due to a "steal" phenomenon resulting in inadequate cooling of the brain. This study was designed to examine the effects of different blood gas management strategies during cooling on cerebral blood flow (CBF) and metabolic recovery following DHCA in the presence of APCA's. PTFE shunts (4mm) were placed between the left subclavian and pulmonary arteries in twenty 1-month old piglets. Animals were randomized as follows:

	Alpha-State (Î± -S)	pH-Stat (pH-S)
Control (shunts ligated)	C- Î± (n=5)	C-pH (n=5)
Shunt (shunts open)	S- Î± (n=5)	S-pH (n=5)

All animals were placed on CPB, cooled to a nasopharyngeal temperature of 18°C with either Î±-S or pH-S blood gas strategy, arrested for 90 minutes, and then rewarmed to 37°C. Global CBF (radioactive microspheres) and cerebral metabolic rate of oxygen consumption (CMRC>2) were measured: I) Warm on CPB, II) Cold on CPB before DHCA, and III) Warm on CPB post-DHCA.

	CBF (ml/100gm/min±S.E.)				CMROj (ml O_2/100gm/min±S.E.)			
	C-Î±	C-pH	S-Î±	S-pH	C-Î±	C-pH	S-Î±	S-pH
I	68 ± 6	65 ± 3	46 ± 4†	47 ± 2†	3.2 ± 0.2	3.2 ± 0.5	2.5 ± 0.2†	2.8 ± 0.4
II	22 ± 4	45 ± 12	12 ± 2†	16 ± 1	0.5 ± 0.1	0.6 ± 0.2	0.4 ± 0.1	0.4 ± 0.01
III	50 ± 5	41 ± 6	23 ± 4†	37 ± 4*	2.3 ± 0.2	2.0 ± 0.3	1.2 ± 0.2†	2.1 ± 0.2*

†p<0.05 vs C-Î±, * p<0.05 vs S-Î±

Conclusions: In the absence of APCA's (C-Î± and C-pH), a-stat and pH-stat cooling strategies provide equivalent cerebral protection. In the presence of APCA's (S-Î± and S-pH), CBF and $CMRO_2$ recovery following DHCA are significantly decreased when compared to controls if a-stat

cooling is employed. This effect is eliminated through the use of pH-stat cooling. In infants with APCA's which are not amenable to pre-operative embolization or intra-operative ligation, pH-stat cooling may reduce the likelihood of neurologic injury.

*By invitation

40. BYPASS EFFECTS OF DELAYED REWARMING ON CEREBRAL BLOOD FLOW IN INFANTS FOLLOWING TOTAL CIRCULATORY ARREST

Erie H. Austin, III, M.D.*, Rosendo A. Rodriguez, M.D.* and Steve M. Audenaert, M.D.*

Louisville, Kentucky

Sponsored by: Laman A. Gray, Jr., M.D., Louisville, Kentucky

Purpose: A possible mechanism of brain injury after TCA may be cerebral hypoperfusion, which results in an unbalanced flow/metabolism ratio. A single report (Astudillo et al. 1993) that a delay in rewarming modified the flow pattern of recovery suggested that cold reperfusion could improve this unbalanced ratio. Our purpose was to detect any possible beneficial effect.

Methods:Transcranial Doppler sonography was used to measure the mean and end-diastolic CBF velocities (mCBFVs) prior to incision and at 20, 45, and 90 min postbypass in 2 pediatric TCA groups with TCA durations longer than 35 min. In group A (n=7), rewarming began immediately with reperfusion. In group B (n=5) rewarming was delayed by a 12 min period of cold reperfusion. Age and TCA duration were not significantly different ($p>0.20$) and CBFVs were analyzed relative to preincision baseline.

Results: At 90 min postbypass, mCBFVs in group A remained below baseline ($p<0.001$) but completely recovered for group B ($p>0.05$) (Table 1). At this time, a diastolic Doppler signal was present in 3 out of 7 cases for group A (43%) but in all cases of group B (100%). edCBFVs completely recovered in 4 cases of group B compared to none in the other group (Fisher's exact test: $p=0.01$).

Table I (mCBFVs % of baseline)

Group	TCA Duration	20 Min	45 Min	90 Min
A (n=7)	52 min ± 17	66% ±23	62% ± 18	68% ±15
B (n=5)	43 min ±3	105% ±38	110% ±29	128% ±52

Conclusions: A delay in rewarming resulted in improved edCBFV and recovery of MCBFVs after bypass for the group of cold reperfusion. This preliminary observation suggests that changes in rewarming strategy may attenuate noxious effects of TCA on brain perfusion.

4:35 pm EXECUTIVE SESSION (Limited to Members)

Republic Ballroom, Sheraton Boston Hotel

6:30 pm MEMBER RECEPTION

Museum of Fine Arts

*By invitation

WEDNESDAY MORNING, APRIL 26, 1995

7:00 am FORUM SESSION II

Grand Ballroom, Sheraton Boston Hotel

Moderators: Robert A. Guyton, M.D.

Shukri F. Khuri, M.D.

F9. THE EXPRESSION OF TNFÎ± AND CHEMOKINES (ENA-78 AND GROÎ±) DURING ISCHEMIA/ REPERFUSION INJURY ASSOCIATED WITH LUNG TRANSPLANTATION

K..E. Magliato, M.D.*, D.L. Swiderski, Ph.D.*,

S.L. Kunkel, Ph.D.*, C.A. Wilke, B.S.*, M.D. Burdick, B.S.*, G.M. Deeb, M.D. and R.M. Stricter, M.D.*

Akron, Ohio and Ann Arbor, Michigan

The reimplantation response or ischemia/reperfusion injury is potentially an important cause of morbidity during lung transplantation. While a number of cellular events are involved in this response, the neutrophil appears to play a pivotal role in mediating lung injury during reimplantation. The mechanisms that lead to neutrophil sequestration and activation are complex and have not been fully elucidated. We postulated that the generation of the early response cytokine, tumor necrosis factor alpha (TNFÎ±), and the chemokines, epithelial cell neutrophil activating protein (ENA-78), and growth related oncogene alpha (GROÎ±), would be significantly elevated during the reimplantation response and be associated with maximal neutrophil sequestration and subsequent lung injury. To test this hypothesis, we evaluated pulmonary microvascular permeability as index of injury, neutrophil influx , and expression of TNFÎ±, ENA-78, and GROÎ± during the reimplantation response using a rat orthotopic single-lung syngeneic transplant model. Cytokine expression in plasma, lung tissue homogenates, and bronchoalveolar lavage (BAL) at 4, 12, 24, 36, and 48 hours post-reimplantation was measured by specific ELISAs (n=30). Cytokine levels from the transplant recipient animals were similar to sham operated control animals until 24 hrs post-

reimplantation. At 24 hrs post-reimplantation, TNFa, ENA-78, and GROÎ± were all significantly elevated (2-fold) from the lung tissue homogenates of the transplant recipients, as compared to sham operated controls. The concentrations of all cytokines returned to levels similar to the sham operated animals by 48 hours. Pulmonary neutrophil sequestration, as evaluated by BAL cell counts, paralleled the changes in lung tissue cytokine levels. At 24 hours, neutrophil absolute and relative levels were higher in transplant recipients (6.5± 4.4 x 10^5, 69.7±5.8% of the total) than in sham operated controls (2.4±1.1 x 10^5, 12.3±5.7% of the total) (p=.002). The findings of BAL neutrophil levels were corroborated by elevated levels of myeloperoxidase activity from transplanted lung tissue homogenates (100.8±22.8) as compared with sham controls (35.1±8.1) (p=.02). Using Evan's blue dye as marker of pulmonary microvascular permeability, transplant recipients at 24 hours post-reimplantation demonstrated a 2.5-fold greater index of lung permeability, as compared to sham operated control animals (p=.01). In addition, transplanted animals pretreated with neutralizing TNF antisera showed a 50% decrease in lung injury by Evan's blue dye lung permeability assessment. These findings suggest that the temporal expression of TNFcc, ENA-78, GROa correlates with maximal neutrophil sequestration and lung injury associated with the reimplantation response and neutralizing TNF antisera has a protective role in ischemia/reperfusion injury.

*By invitation

F10. THERAPEUTIC ANGIOGENESIS: ACIDIC FIBROBLAST GROWTH FACTOR ACCELERATES COLLATERAL VESSEL FORMATION AND REVASCULARIZATION OF ISCHEMIC TISSUES

Todd K. Rosengart, M.D.*, Martin A. Duenas, B.S.*, Qing-Xue Zhang, Ph.D.*, Karl H. Krieger, M.D. and O. Wayne Isom, M.D.

New York, New York

Angiogenesis, the growth of new blood vessels, is thought to be an important biological mechanism allowing for the revascularization of ischemic tissues. Acidic fibroblast growth factor (aFGF) is a potent stimulant of angio-genesis that may prove useful in accelerating

collateral vessel development following vascular occlusion. We therefore assessed the ability of aFGF to enhance angiogenesis and improve perfusion in the setting of acute ischemia. Ligation of the left common femoral artery was performed in the rat followed by the daily transcutaneous injections into the ischemic hind limb region of saline (1ml) or recombinant human aFGF (1 mcgm in 1 ml saline) for 10 days following ligation (n=4). Following the treatment period, 2 x 10^6 color micro-spheres were injected via the infrarenal abdominal aorta and the relative perfusion of the ligated limb versus the unligated, contralateral control limb was determined from the microsphere counts per gram of tissue harvested from each limb. Relative perfusion of the ligated limb in the aFGF-treated animals was 62 ± 16% (mean ± SEM) of that to the unligated, contralateral control limb compared with 10 ± 3% relative perfusion in the saline-treated animals (p=0.004). These results were confirmed by histologic analysis of hind limb tissue specimens, which demonstrated 27 ± 2 vs 4 ± 1 vessels per high power field in the aFGF and saline-treated animals, respectively (p=0.001). Proliferating cell nuclear antigen (PCNA) analysis was utilized to assess mitogenic activity and demonstrated that the percentage of PCNA-labelled cells per total cell nuclei was 68 ± 4% vs 5 ± 1% in the two respective groups (p<0.001). The six-fold increase in perfusion noted in these studies, confirmed by analysis of vascularization and cell replication, suggest that exogenous aFGF administration may represent a useful means of enhancing collateral vessel growth and relieving acute ischemia. The administration of aFGF may potentially allow the revascularization of myocardial or other tissues not salvageable by bypass procedures or other conventional means.

*By invitation

F11. MYOBLAST TRANSPLANTATION IN THE PORCINE MODEL. A POTENTIAL TECHNIQUE FOR MYOCARDIAL REPAIR

Clifford H. Van Meter, Jr., M.D.*, Frank Smart, M.D.*, William Claycomb, Ph.D.*, Joseph DelCarpio, Ph.D.* and John L. Ochsner, M.D.

New Orleans, Louisiana

The increasing shortage of organ donors continues to fuel the search for other methods to manage heart failure.

The use of transgenic cells transplanted in syngeneic rodents has shown modest success, but allogeneic and xenogeneic transplants have not been uniformly successful. To assess the feasibility of xenogeneic and allogeneic myoblast transplantation, six adult swine underwent transplantation of murine atrial tumor cells (xenogeneic) and neonatal porcine (allogeneic) into the left ventricular wall. A subsequent seventh animal underwent transplantation of human neonatal myoblasts (16 weeks of age). Following general anesthesia, isolated cells were injected along the anterior and posterior wall of the porcine left ventricle (6 to 8 sites per animal). All the animals were immunosuppressed with cyclosporine and prednisone and were followed for one month post-injection at which time they were sacrificed.

In all injected sites, the transplanted cells proliferated within the host myocardium with no significant rejection. CPK MB isoenzymes did not increase after the procedure indicating that there was no damage to the host myocardium from the injection of cells. Moreover, transplant cells formed close associations with host myocytes that resembled intercalated discs on electron mycroscopy, and were composed of PAN cadherin on immuno-fluorescent staining. These cells also contained myofibrils and other cell architecture that resembled normal AT-1 or neonatal myocytes. Additionally, these cells produced angiogenic factors resulting in a proliferation of the surrounding microvasculature. In conclusion, these findings indicate successful xenogeneic and allogeneic myocyte cell transplantation in a large animal model. These experiments set the stage for future studies to test the ability of these cells to form a syncytium, to contract, and potentially repair failed myocardium.

*By invitation

F12. EFFICIENT CARDIAC GENE TRANSFER BY INTRA-CORONARY INFUSION OF ADENOVIRUS VECTOR MEDIATED REPORTER GENE IN TRANSPLANTED MOUSE HEART

Jeongryul Lee, M.D.*, Alistair I. Fyfe, M.D.*, Hillel Laks, M.D., Davis C. Drinkwater, M.D., Yuji Shiraishi, M.D.* and Paul Chang, B.S.*

Los Angeles, California

Gene transfer into the coronary arteries may offer an opportunity to modulate alloreactivity after cardiac transplantation. In the present study, we perfused the coronary arteries of donor mouse hearts with 10^7 pfu (200 |aL) of a recombinant adenovirus containing P-galactosidase encoding gene (AdCMV P gal) just prior to harvesting to demonstrate the feasibility, efficacy, and safety of adenovirus mediated in vivo transfer. The donor hearts were transplanted into the abdomen of the recipient mice of the same strain (BIO. BR). Both the grafts and recipient hearts were harvested at 3, 7, 15 and 30 days (n=6 in each group) after transplantation and P-galactosidase activity was assessed by immunohistochemistry. All grafts were functioning adequately at the time of harvest and P-galactosidase expression was seen throughout the myocardium, epicardium, and the surrounding coronary arteries. There were no significant differences in expression at least up to 30 days after transplantation. Staining was not seen in recipient hearts or sham control isografts (n=6) which were sacrificed 7 days after infusion of saline into the coronary arteries. This study revealed that intracoronary adenovirus mediated gene transfer is an efficient and feasible method for transfecting genes, with widespread expression, and there does not appear to be significant early cardiac graft dysfunction associated with the use of adenovirus. This approach may allow modification of graft immunogenicity in the future by allowing the production of therapeutic proteins that inhibit or delay the development of cardiac allograft vasculopathy.

*By invitation

F13. INTRATHYMIC STEM CELL TRANSPLANTS PROLONGSKIN GRAFT SURVIVAL IN UNRELATED PRIMATES

†Margaret D. Allen, M.D., Hiroji Akimoto, M.D.*, John T. Weyhrich, D.V.M.*, Karen A. Nelson, Ph.D.*, Robert Thomas, B.S.* and Robert G. Andrews, M.D.

Seattle, Washington

The intrathymic implantation of stem cells in transplant recipients may resolve several problems limiting the attainment of tolerance to transplanted organs. 1) Peripheral blood microchimerism has been associated with

reduced rejection in clinical liver and renal transplantation but peripheral infusion of donor marrow to induce chimerism carries a risk of graft-versus-host disease (GVHD). Recent bone marrow transplants in primates suggest that stem cell fractions may result in less GVHD. 2) Intrathymic implantation of lymphocytes, splenocytes, class II peptides, and even islets in rodents pre-treated with myeloablation or anti-lymphocyte globulin can extend subsequent donor organ or skin graft survival, but hematopoietic microchimerism has not been reported. Stem cells might provide a self-perpetuating source of donor antigens. 3) If suppressor mechanisms are operative, less, rather than more, immunosuppression may be beneficial. This study examines whether the intrathymic implantation of donor CD34+ enriched marrow (97% CD34+) might produce hematopoietic chimerism and induce tolerance to donor-derived grafts in unrelated primates with or without immunosuppression.

Four juvenile female baboons underwent intrathymic transplantation of CD34+ enriched marrow (1×10^6 cells) from unrelated male baboon donors through a left thoracotomy approach. Peripheral blood was sampled at biweekly intervals for the presence and persistence of donor cells using PCR for Y chromosome determinants. Two recipient animals were treated with standard triple drug immunosuppression consisting of cyclosporine, azathioprine, and steroids, in doses equivalent to the clinical cardiac transplant protocol. No myeloablative therapy or anti-lymphocyte serum induction was used. Two other recipients received no immunosuppression. All 4 recipients, both with and without immunosuppression, developed peripheral blood microchimerism within 2 weeks which is still persistent at 9-16 months. No clinical evidence of GVHD was observed.

To test whether the implantation of intrathymic stem cells resulted in tolerance, two further juvenile female baboons were implanted with male donor CD34+ marrow without recipient immunosuppression. Chimerism was detected in both animals. Three months following the intrathymic implants, 9 skin grafts were placed on each recipient: 3 from autologous skin, 3 from the stem cell donor, and 3 from a third party baboon. Autologous grafts were incorporated without rejection. Third party grafts sloughed at 24 days post-transplant and, in both recipients, the grafts from the stem cell donor persisted DNA. Repetition of the skin grafts again demonstrated

prolongation of the stem cell-donor over the third party-donor grafts.

These results demonstrate that: l)the intrathymic implantation of donor CD34+ enriched marrow can induce peripheral microchimerism in non-human primates with or without immunosuppression; 2) 1 x 10^6 CD34+ cells is sufficient to induce peripheral chimerism; 3) donor-specific skin graft, prolongation was achieved; and 4) graft versus host disease was not observed. These findings have current applications in the field of thoracic transplantation tolerance as well as future potential in the treatment of genetic deficiency diseases of the newborn.

†Nina S. Braunwald Career Development Award Recipient

*By invitation

F14. INHIBITION OF INDUCIBLE NITRIC OXIDE

AMELIORATES RAT LUNG ALLOGRAFT REJECTION

Takeshi Shiraishi, M.D.*, Neil K. Worrall, M.D.*, Steven R. DeMeester, M.D.*, Jon H. Ritter, M.D.*, Thomas P. Misko, Ph.D.*, T. Bruce Ferguson, M.D., Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.

St. Louis, Missouri

Inhibition of the production of nitric oxide (NO) by inhibition of inducible nitric oxide synthase (iNOS) has recently been demonstrated to attenuate the pathogenesis of acute cardiac allograft rejection. This study examined the effect of inhibition of NO production on rejection in a rat lung transplant model. Syngeneic (F344 to F344) (n=7) and allogeneic (Brown Norway to F344 recipient) (n=8) left lung transplants were performed. In this latter strain combination complete rejection occurs by postoperative day 8.

Using the same allogeneic combination, a third group of animals (n=9) underwent allotransplantation and received aminoguanidine (a specific iNOS inhibitor) 200 mg/kg IP q6h from transplantation until sacrifice on postoperative day 7 (POD-7). NO production was demonstrated by measuring serum nitrite/nitrate levels (stable end products of NO metabolism). Allograft serum nitrite/nitrate levels [16.5 ± 1.7μM, n=4 ($p<.01$ by ANOVA)] were elevated above normal control (8.0 ± 1.0 μM, n=3) and isograft levels (9.0 ± 1.4 μM, n=4) on postoperative day 4 (POD-4), indicating that NO is produced during lung allograft rejection. Aminoguanidine treatment prevented the increased serum nitrite/nitrate levels in the allografts on POD-4 (9.8 ± 1.0 μM; $p<0.01$ vs untreated allografts). The effect of iNOS inhibition on acute rejection was determined by using three scoring systems. Severity of rejection was evaluated using a radiographic score (Aeration Score; 0=opaque to 6=normal appearing lung). The histologic rejection grade was determined at sacrifice on POD-7 using the International Working Formulation (I.W.F. score: 0=normal to 4=diffuse infiltration of mononuclear cells) and Prop's scoring system (0=latent phase to 3=destruction). Aminoguanidine treatment significantly attenuated acute rejection as determined

radiographically [AS= 4.1 ± 1.4 vs 1.8 ± 0.9 for untreated allografts on POD-6 ($p<.05$) and 3.9 ± 1.5 vs 0.6 ± 0.5 on POD-7 ($p<.01$)]. Aminoguanidine treatment also significantly ameliorated the histologic degree of rejection as determined at sacrifice on POD-7 [2.6 ± 0.2 vs 3.8 ± 0.5 for untreated allografts by IWF score ($p<.005$) and 1.3 ± 0.4 vs 2.6 ± 0.7 by Prop's score ($p<.005$)]. These results demonstrate that NO is produced during acute lung allograft rejection and that selective inhibition of the inducible isoform of nitric oxide synthase significantly attenuated acute rejection. NO production may serve as a marker of early rejection and selective inhibition of iNOS may be a novel therapeutic modality in the management of acute lung allograft rejection.

*By invitation

F15. THE EFFECT OF N-co-NITRO-L-ARGININE ON PULMONARY VASCULAR TONE IN THE MATURING NEWBORN PIG

Patrick W. Domkowski, Ph.D.*, John T. Cockerham, M.D.*, Peter A. Kot, M.D.*, Jeffrey L. Myers, M.D.*, Yi-Ning Wang, M.D.*, Robert B. Wallace, M.D. and Richard A. Hopkins, M.D.

Washington, DC

Current therapeutic modalities for treatment of newborn pulmonary hypertensive crisis include the administration of nitric oxide (endothelium derived relaxing factor, EDRF). However, few data are available on the role of endogenously produced EDRF in the modulation of newborn pulmonary arterial tone. We investigated the acute effects of a 10-minute intravenous infusion of N-oo-

nitro-L-arginine (L-NA), a potent competitive inhibitor of EDRF synthase on the pulmonary vascular response, in 48-hour and 2-week-old anesthetized open-chest Yorkshire pigs. Fourier analysis of high fidelity main pulmonary artery pressure (PAP) and flow (PAF) waveforms was employed to distinguish between proximal and distal pulmonary arterial responses to L-NA. Input mean impedance (Z_m, reflective of distal vasocon-striction) and characteristic impedance (Z_0, reflecting proximal arterial disten-sibility) were calculated. Pulmonary vascular resistance (PVR) was also calculated. Data were collected at baseline and during intravenous infusion of L-NA.

	PAP	PAF	PVR†
48-hour-old pigs (n=8)	17.0 ± 1.1	4.0 ± 0.6	5171 ± 805
L-NA (100mg/kg/min) infusion	17.6 ± 1.2	4.0 ± 0.5	5189 ± 1058
2-week-old pigs (n=7)	13.7 ± 1.1	8.4 ± 1.0	810 ± 137
L-NA (100mg/kg/min) infusion	18.0 ± 1.4*	8.7 ± 0.7	1519 ± 239*

mean ± S.E.M. *p<0.05 v. corresponding baseline; PAP = mmHg, PAF = ml/sec, †uni

Baseline hemodynamic parameters were significantly diminished in 2-week-old pigs, reflecting the expected decrease in both proximal and distal pulmonary arterial vasoconstriction with maturation. L-NA produced no significant pulmonary hemodynamic changes in 48-hour-old pigs. However, in 2-week-old pigs, L-NA caused a significant pulmonary arteriolar vasoconstriction. L-NA infusion did not cause any significant alterations in ZQ in either group. These data indicate that endogenously produced EDRF modulates distal pulmonary arterial tone in 2-week-old pigs, but not in 48-hour-old pigs. Pulmonary hypertensive crisis of the newborn may reflect an inability of the pulmonary endothelium to synthesize and release nitric oxide in quantities sufficient enough to modulate pulmonary vascular tone.

*By invitation

F16. ELUCIDATION OF A TRIPARTITE MECHANISM UNDERLYING THE IMPROVEMENT IN CARDIAC TOLERANCE TO ISCHEMIA BY COENZYME Q_{10} PRETREATMENT

Juan A. Crestanello, M.D.*, Joseph Kamelgard, M.D.*, David M. Lingle, M.D.*, Svend A.

Mortensen, M.D.* and Glenn J.R. Whitman, M.D.

Philadelphia, Pennsylvania and Copenhagen, Denmark

CoQ_{10}, involved with mitochondrial ATP production, is also a powerful antioxidant. We hypothesize that CoQ_{10} pretreatment protects myocardium from ischemia reperfusion injury by both its ability to increase aerobic energy production and by its ability to protect creatine kinase (CK) from oxidative inactivation during reperfusion.

Isolated hearts (n=6/group) from rats pretreated with either CoQ_{10} 20 mg/kg i.m. and 10 mg/kg i.p. (CoQ) or vehicle only (CTRL) 24 and 2 hours prior to the experiment were subjected to 15 minutes of equilibration (EQ), 25 minutes of ischemia, and 40 minutes of reperfusion (RP). Developed pressure (DP), ±dP/dt, myocardial oxygen consumption (MVO_2), and myocardial aerobic efficiency (DP/MVC^) were measured. In parallel groups of hearts, ^{31}P NMR spectroscopy was used to determine P-ATP and PCr concentrations. Hearts were assayed for myocardial CK activity at end RP. Values are expressed as mean ± SEM with t-test used for statistical evaluation.

	Reperfusion 40					PCr	
	DP (% EQ)	**+dP/dt** (% EQ)	**-dP/dt** (% EQ)	**CK** IU/g V	**DP/MVOZ** mmHg/(μ O_2/min/g V	**EQ** (%MDP)	(%
CTRL	37 ± 4	37 ± 5	36 ± 5	391 ± 10	0.621 ± 0.069	35 ± 3	
CoQ	62 ± 2**	64 ± 2**	61 ± 2**	435 ± 17*	0 835 ± 0 076*	49 ± 3**	45

(MDP: Methylenediphosphonic acid standard) *p<0.05 vs Control **p<0 005 vs Control

CoQ_{10} pretreatment improves myocardial function after ischemia reperfusion. This results from a tripartite effect: 1) higher ATP and PCr concentrations initially and during reperfusion, 2) improved myocardial aerobic efficiency during reperfusion, and 3) protection of CK from oxidative inactivation during reperfusion with an improved bioenergetic profile.

*By invitation

F17. INDUCED IMMUNOLOGIC UNRESPONSIVENESS INHIBITS CARDIAC GRAFT ARTERIOSCLEROSIS

Yong T. Shin, M.D.*, Mohamed H. Sayegh, M.D.*, Lauri R. Wyner, B.A.*, Morris J.

Karnovsky, M.B., B.Ch.* and †David H. Adams, M.D.*

Boston, Massachusetts

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

Chronic rejection, manifested by the development of graft arteriosclerosis, remains the leading cause of late death in cardiac transplant recipients. Immunologic mechanisms have been implicated in both experimental and clinical studies, yet the pathogenesis and therapy of chronic allograft rejection remains poorly defined. Recent studies have shown that intrathymic (i.t.) injection of donor cells or processed allo-MHC antigens with or without transient immunosuppression induces specific systemic T cell tolerance and prevents acute allograft rejection in several experimental trasnplantation models. In this study we examined the effects of i.t injection of donor cells with or without systemic anti-lymphocyte serum (ALS) on the development of chronic rejection in the Lewis-to-F344 rat cardiac allograft model. Recipients were divided into 4 groups: control group A was pre-treated with saline i.t., group B received donor (Lewis) splenocytes i.t. ($2x10^6$), group C was pre-treated with a one-time dose of ALS (1 ml) via intraperitoneal injection (i.p.), and group D received both the splenocytes i.t. and a one-time dose of ALS i.p. two weeks prior to transplantation. Allografts were followed by daily palpation and graded from 0-4 (grade 0=non-palpable, grade 4=normal heartbeat). Graft survival on post-op day 90 for all three treated groups was 100% vs. 33% for the control (n=6/group), and mean heartbeat grade for the three treated groups (B=2.0±0.41, C=2.83±0.17, D=2.67±0.21) was significantly higher ($p<0.01$) vs. the control group (A=0.4±0.24). Histologic and immunocytochemical analysis of 90-day old grafts revealed the following results (*($p<0.02$, **$p<0.003$vs Control):

Groups	Inflammation	% Vessels Diseased	% Luminal Occlusion	Intima:Media Ratio
Control (A)	severe	89 ± 1	64 ± 5	0.97 ± 0.33
Splenocyte only (B)	moderate-severe	79 ± 8	57 ± 7	0.68 ± 0.16
ALS only (C)	minimal	25 ± 3**	8 ± 3**	0.09 ± 0.04*
Splenocyte+ALS (D)	minimal	27 ± 3**	8 ± 3**	0.07 ± 0.03*

Conclusions: (1) Treatment with donor splenocytes i.t. alone partially suppresses the immune response, prolonging graft survival and preserving function without inhibiting graft inflammation or arteriosclerosis. (2) The induction of systemic unresponsiveness with ALS i.p. or splenocytes i.t. plus ALS i.p. markedly reduces inflammation and inhibits graft arteriosclerosis, suggesting the development of therapies resulting in better control of the immune response remains the key to ameliorating this disease process.

†Alton Ochsner Research Scholar .

*By invitation

F18. MOLECULAR GENETIC DIFFERENTIATION BETWEEN PRIMARY LUNG CANCER AND LUNG METASTASES OF OTHER TUMORS

Daniela Kandioler, M.D.*, Gerhard Dekan, M.D.*, Adelheid End, M.D.*, Michael Mttller, M.D.*, Michael Gnant, M.D.*, Eva Pasching*, Christine Mannhalter, Ph.D.*, Franz Eckersberger, M.D.*, and Ernst Wolner, M.D.

Vienna, Austria

When single pulmonary tumors are observed in patients with history of other cancer, differentiation between metastasis and second primary lung cancer is crucial for appropriate therapy. Particularly in low differentiated tumors this determination by means of histology alone may be difficult or impossible even with the help of immunochemistry. Implicating that p53 mutations are conserved in metastases, we hypothesized that a genetic fingerprinting using the p53 gene could be a valuable tool in differentiating between metastases and second primary carcinomas since the p53 gene is

frequently affected by various mutations in many different types of human carcinomas.

Out of 267 resected lung tumors we analysed 9 cases of solid lung tumors whose origin could not exactly be defined histologically. As a control we used tumor material of a patient with adenocarcinoma of the lung and histologically defined brain and lymphnode metastases.

Cases	*History of*	*Lung Tumor*
5	colon carcinoma	adenocarcinoma
1	breast cancer	adenocarcinoma
1	soft tissue sarcoma	undifferentiated blastoma
1	renal & thyroid cancer	clear cell carcinoma
1	head and neck	squamous cell carcinoma

We extracted DNA from the shock frozen tissue of the unclassified lung tumors, from the paraffin embedded tissue of the "past primary cancers" and from peripheral blood of the patients. Exons 2 to 11 of the p53 gene were amplified separately by polymerase chain reaction (PCR) and directly sequenced. In all cases the presence of germline mutations could be excluded by analysis of the peripheral blood. The p53 mutation detected in the control tumor proved to be conserved in the lymph node as well as in the brain metastasis.

In two cases the lung tumor was identified as (second) primary lung cancer since the genetically compared tumors exhibited different mutations. In five cases the lung tumors proved to be metastases exhibiting the identical p53 mutation as their "primary." In one case the lung tumor could be identified as metastases from renal cancer whereas a

thyroid cancer metastasis could be excluded. One case remained inconclusive since no p53 mutation could be found in any tissue. Thus, due to genetic analysis using the p53 gene, correct surgical treatment could be approved in six cases regarded as metastatic disease of the lung, whereas further pulmonary surgery was mandatory in two cases identified as second primary lung cancer.

We conclude that analysis of p53 mutations is a valuable tool in the exact diagnosis of metastases and recurrent tumors which are difficult to classify by standard methods. Since the information about tumor origin is essential in assessing an individual patient's treatment strategy, these results are clinically relevant.

*By invitation

9:30 am SIMULTANEOUS SCIENTIFIC SESSION D ADULT CARDIAC SURGERY

Grand Ballroom, Sheraton Boston Hotel

Moderators: D. Glenn Pennington, M.D.

Robert L. Hardesty, M.D.

41. REFERRAL SOURCE AS A RISK FACTOR FOR CORONARY BYPASS SURGERY: HMO VS. PRIVATE PRACTICE

Albert Starr, M.D., Aftab Ahmad, M.D.* Anthony Furnary, M.D.*, Guo Wei He, M.D.*, Jeffrey Swanson, M.D.* and Gary Grunkemeier, Ph.D.*

Portland, Oregon

We began performing coronary bypass surgery (CBS) for a large HMO in 1974, as the sole provider of their cardiac surgery. The HMO system entails pre-intervention, multi-disciplinary screening conferences and is absent of self-referral and personal financial incentives. Since 1985, the operative mortality for HMO patients has consistently

been lower than for private patients. There were 8, 483 surgeries during this study period: 3, 168 (37%) HMO, with an overall operative mortality of 2.7%, and 5, 315 (63%) non-HMO, with an operative mortality of 4.6% (P=.00002). All patients were operated on in the same hospital by the same surgical group.

We investigated this difference with univariate and multivariable analyses. 19 risk factors were found to univariately affect the risk of operative mortality ($p<.05$). For 11 of these risk factors there was a significant maldistribution between HMO and non-HMO patients; HMO patients had worse average values for 3 factors and better values for 8 (the latter included age, previous CBS, abnormal wall motion, chronic angina, and emergency surgery).

We used logistic regression to explore the influence of this imbalance in risk factors. The model found 7 factors to significantly affect operative mortality, after which the variable indicating HMO membership was no longer significant ($p=.006$). Because of missing data, only 1, 279 (40%) HMO patients and 2, 407 (45%) non-HMO patients, with operative mortalities of 2.6% and 4.1%, respectively, could be used.

A reasonable conclusion is that the difference in operative mortality between a coherent and chaotic system of cardiologic management results in a distribution of risk factors that enhance the operative results in the coherent group. The question of whether this selection process would be beneficial to a large population at risk has not been addressed.

*By invitation

42. REDUCING THE RISK OF INFECTIVE ENDOCARDITIS AFTER AORTIC VALVE REPLACEMENT

Arvind K. Agnihotri, M.D.*, David C. McGiffin, M.D.*, Andrew J. Galbraith, M.D.* and Mark F. O'Brien, M.D.

Birmingham, Alabama and Brisbane, Australia

Replacement valve endocarditis (RVE) occurred on 3.9% of 2, 686 aortic valve replacement devices inserted between December 31, 1969 and January 1, 1992, based on a cross-sectional follow-up in 1992 which was 98.8%

complete. As shown by others, the risk (hazard function) for RVE was highest early after valve replacement, peaking 3 months after operation, then decreasing to reach a constant hazard by about 6 months after operation. By multivariable analysis, patients with active preoperative endocarditis ($p<.0001$) or those receiving a synthetic aortic root replacement ($p=.0006$) were at an increased early risk for RVE, but this increased risk was neutralized when an allograft valve was used ($p<.0001$; figure A [figures A and B are nomograms of the multivariable equation]). Late risk (later than 6 months after operation) was increased in younger patients ($p<.0001$), those with renal failure ($p=.01$), and in patients with active or remote preoperation endocarditis ($p=.04$). Late risk was particularly high in patients with preoperative *Staphylococcus aureus* endocarditis ($p=.05$), which tended to recur as another staphylococcal endocarditis. Use of some mechanical valves resulted in a reduction in late risk ($p=.05$; figure B, while use of some xenograft valves increased late risk ($p=.02$).

In patients without risk factors, valve replacement with any modern device results in a similar infectious risk (figure C). In the presence of early phase risk factors use of an allograft valve reduces the risk of RVE. In patients with only late phase risk factors the use of some mechanical valves may be preferable.

*By invitation

43. SURGICAL TREATMENT OF PROSTHETIC VALVE ENDOCARDITIS

Bruce W. Lytle, M.D., Brian P. Priest, M.D.*, Paul C. Taylor, M.D.*, Floyd D. Loop, M.D., Robert W. Stewart, M.D.*, Patrick M. McCarthy, M.D.*, Derek Muehrcke, M.D.* and Delos M. Cosgrove, M.D.

Cleveland, Ohio

From 1975 through 1992, we reoperated on 146 patients for the treatment of prosthetic valve endocarditis (PVE). Inclusion in the study stopped in 1992 to allow follow-up of all patients. PVE was considered early (<1 year after operation) in 46 cases, active in 103 cases, and the extent of the infection was prosthesis only, 66, annulus, 46, and cardiac invasion, 34. Surgical techniques evolved in the direction of increasingly radical debridement and reconstruction with biological materials. All patients were treated with prolonged postoperative antibiotic therapy.

There were 19 (13%) in-hospital deaths. Univariate analyses demonstrated trends toward increasing risk for patients with active PVE and extension of infection beyond the prosthesis; however, the only variables with a significant ($P<0.05$) association with increased in-hospital mortality confirmed with multivariate testing were impaired LV function, heart block, major organ system failure and identification of organisms in the pathology specimen. During the study period, mortality decreased from 20% (1975-1984) to 10% (1984-1992). For hospital survivors the mean length of stay was 25 days.

Follow-up (mean interval 62 months) documented late survival of 82% at 5 and 60% at 10 postoperative years. Increasing age was the only factor associated ($P=0.006$) with late mortality. Nineteen patients needed at least one further operation; reoperation-free survival was 75% at 5 and 50% at 10 postoperative years. Fever in the immediate preoperative period was the only factor associated with decreased late reoperation-free survival ($P=0.049$).

PVE remains a serious complication of valve replacement but the in-hospital mortality of reoperations for PVE has declined. With extensive debridement of infected tissue and postoperative antibiotic therapy the extent and activity of PVE does not appear to have a major impact on late outcome and the majority of patients with PVE survive for 10 years after surgery.

*By invitation

44. THE ADDITIONAL HOSPITAL COSTS GENERATED IN THE MANAGEMENT OF COMPLICATIONS OF PACEMAKER AND DEFIBRILLATOR IMPLANTATIONS

T. Bruce Ferguson, Jr., M.D., Candice Lilley Ferguson, M.S.P.H.* and Patricia Crimmins-Reda, R.N., M.S.N.*

St. Louis, Missouri

The rapid approach of capitated reimbursement mandates that providers examine their practice patterns associated with all surgical procedures. As part of this effort, documentation of 1) the complications associated with these procedures; and 2) the **additional hospital costs** associated with the management of these complications, is critical for comprehensive fiscal accountability. This study analyzed the fully-loaded hospital costs generated in the management of the most common surgical complications - infection, lead-related problems and hematoma formation requiring evacuation - associated with pacemaker and non-thoracotomy implantable defibrillator therapies.

Between July 1989 and September 1994 a total of 1, 031 pacemaker (PM) and 331 implantable defibrillator (ICD) procedures were performed by a cardiac surgeon in a tertiary-level teaching hospital setting. Over the past two years, 105 of these ICD procedures involved non-thoracotomy (NT-ICD) systems. Because of the commonly presumed similarities between PM and NT-ICD procedures, these two groups will be examined in this analysis.

There were no deaths associated with the 1, 136 procedures. The overall in-house PM infection rate was 0.58% (6/1, 031). An additional nine PM patients were transferred from outside institutions for definitive therapy of infected systems. The in-house PM lead reposition/replacement rate was 4.1% (37/902 procedures involving lead placement), performed from 1-78 days post-initial implant. Four PM patients developed pocket hematomas requiring evacuation; all were on systemic anticoagulation for medical problems. The overall in-house infection rate for NT-ICD was 0.95% (1/105). Two patients, both on systemic anticoagulation, developed pocket

hematomas requiring reexplora-tion; both required lead configuration modifications due to elevated defibrilla-tion thresholds (DFTs) at the second procedure. Two patients had ICD system malfunctions, and four other patients (3.8%) had lead fractures (N=2) or dis-lodgements that necessitated surgical reexploration.

The additional fully-loaded hospital costs were documented as additional length of stay (LOS) in-hospital, ICU requirement, OR procedure costs, device costs and additional pharmacy, administrative and miscellaneous costs **directly** associated with the complication (data are expressed as mean ± SD):

Procedure	Complication	#Pts	Mean Age (Yrs)	Mean (Da
PM	Infection	15	63.4 ± 17.4	17.3 ±
	Lead-Related	37	65.1 ± 15.3	2.4 ±
	Hematoma	4	63.5 ± 16.5	5.0 ±
NT-ICD	Infection	1	52	16
	Lead-Related	4	47.3 ± 17.6	3.5 ±
	Hematoma/High DFTs	2	46.5 ± 24.7	11.0 ±
	System Malfunction	2	69.5 ± 10.6	2.5 ±

Thus management of these common complications of PM and NT-ICD therapies is expensive. For all Medicare patients with complications in this study, costs exceeded reimbursement, suggesting that similar shortfalls would occur under a capitation scheme. The study demonstrates that accurate identification of the costs associated with complications of interventional procedures is feasible. This information is critical to a complete understanding of the financial impact of surgical procedures in a capitated reimbursement environment.

10:50 am INTERMISSION

*By invitation

11:10 am SIMULTANEOUS SCIENTIFIC SESSION D

ADULT CARDIAC SURGERY
Grand Ballroom, Sheraton Boston Hotel
Moderators: D. Glenn Pennington, M.D.

Robert L. Hardesty, M.D.

45. A PROSPECTIVE RANDOMIZED TRIAL OF EARLY EXTUBATION AFTER CORONARY ARTERY BYPASS

Charles M. Peniston, M.D., C.M.*, Davy Cheng, M.D.*, Jacek Karski, M.D.*, Zhao Sun, M.A.*, Susan Armstrong, M.Sc.* and

Tirone E. David, M.D.

Toronto, Ontario, Canada

Early extubation within the first few hours after cardiac surgery has been proposed as a safe method of cost containment by reducing ICU-and hospital stay. Early extubation has been possible in most cardiac patients by modifying the anaesthetic. All patients who had this modified anaesthetic were retrospectively reviewed to determine the predictors of failure of early extubation. Stepwise logistic regression analysis revealed that age > 65 years, female gender, moderate or severe left ventricular function and urgent operations were associated with a lower probability of early extubation. We then undertook a prospective study to determine if early extubation is safe and reduces the length of ICU and hospital stay after coronary artery bypass (CAB).

Methods: After institutional approval, 89 patients undergoing elective CAB were randomized to either conventional anaesthesia (CONVENTIONAL) (induction with fentanyl 50 meg/kg; maintenance with isoflurane and midazo-lam 0.1 mg/kg/hr) and assisted ventilation until next morning after surgery, or modified anaesthesia (EARLY EXTUBATION) (induction with fentanyl 15 meg/kg, isoflurane and propafol 2-4 mg/kg/hr) and early extubation (1 to 6 hours post-op). Continuous ST analysis was performed with pre-op baseline to 48 hrs postop. CK and CK-MB were measured at 6, 12, 24, 36, and 48 hrs after X-clamp release. Arterial and venous blood gases were measured at 240 mins after extubation. Respiratory patterns and shunt fraction (Qs/Qt) were monitored for 24 hours after extubation. Unless indicated data are expressed as mean ± S.D. and were compared by Student's t-test.

RESULTS	CONVENTIONAL	EARLY EXTUBATION
Number of patients	45	44
Age	61 ± 11	58 ± 9

Number of grafts	3.2 ± 1.0	3.5 ± 1.0
Incidence of ST changes	39%	50%
Maximum CK-MB (IU/L)	25	18
Pulmonary shunt (Qs/Qt)	23 ± 9%	13 ± 6%*
Assisted ventilation (hrs)	18.9 ± 1.9	3.8 ± 1.1*
Apnea incidence	30%	22%
ICU discharge criteria (hrs)	25.6 ± 10.1	7.0 ± 1.8*
Hospital stay (days)	9.1 ± 3.8	6.9 ± 1.4*

*indicates $p<0.05$ between groups

Data from 784 patients undergoing CAB or valve procedures were also examined. ICU length of stay was reduced from 2.5±4.7 days to 1.43±0.9 days ($p<0.0001$) and hospital stay was reduced from 10.6±7.5 days to 8.3±4.2 days ($p<0.0001$).

Conclusion: These results indicate that early extubation after elective CAB is safe and reduces the length of ICU and hospital stay.

*By invitation

46. ANTI-ISCHEMIC AND ANTI-ARRHYTHMIC PROTECTION BY PERIOPERATIVE INFUSION OF NIFEDIPINE AND ETOPROLOL IN PATIENTS UNDERGOING ELECTIVE AORTOCORONARY BYPASS SURGERY

Bruno K. Podesser, M.D.*, Severin S. Schwarzacher, M.D.*, Werner Zwolfer, M.D.*, Friedrich Peschl, M.D.*, Thomas Binder, M.D.*, Ernst Wolner, M.D. and Rainald Seitelberger, M.D.*

Vienna, Austria

A randomised study was performed on 70 patients undergoing elective coronary bypass procedure to examine whether the combined

perioperative 24-hour infusion of nifedipine (10 ug/kg/h) and metopropol (12 ug/kg/h) reduces the incidence of perioperative myocardial ischemia and arrhythmias (group NM, n=34). The control group (group N, n=36) received nifedipine only. Repeated assessments of serum enzyme levels (CK, CK-MB) and 12-lead-ECG together with a 3-channel Holier monitoring over 48h were used to classify perioperative myocardial ischemia (transient ischemic event, myocardial infarction) and supraventricular and ventricular arrhythmias.

The two groups did not differ with respect to their demographic data, extracorporeal circulation, aortic cross-clamping time, or number of distal anastomosis. No perioperative myocardial infarction in either group was detected. However, a significantly lower incidence of transient ischemic events was observed in the NM group as compared to the N group (3% versus 11%; $p<0.05$). In addition, there was a tendency towards lower CK-MB-levels and peak-values of CK- and CK-MB-enzymes in the NM group. With regard to perioperative dysrhythmias, there was a significantly lower incidence of sinus tachycardia (9%) and atrial flutter/fibrillation (6%) in the NM group as compared to the N group (33% and 27%, $p<0.05$). In addition, postoperative heart rate was lower in the NM group starting from the 6th hour after opening the aortic cross-clamp.

In conclusion, the combined perioperative infusion of nifedipine and metoprolol is superior in preventing perioperative myocardial ischemia and decreasing the incidence of supraventricular arrhythmias as compared to a single-drug regimen with nifedipine.

*By invitation

47. TRANSPLANT CANDIDATES' CLINICAL STATUS RATHER THAN RIGHT VENTRICULAR FUNCTION DEFINES

NEED FOR UNIVENTRICULAR VS. BIVENTRICULAR SUPPORT

Robert L. Kormos, M.D, Si M. Pham, M.D.*, Thomas A. Gasior, M.D.*, Srinivas Murali, M.D.*, Brack G. Rattier, M.D., Ph.D.* and Hartley P. Griffith, M.D.

Pittsburgh, Pennsylvania

We have studied our experience since 1988 in 31 patients who required a mechanical circulatory bridge to transplantation who also had biventricular failure (mean RV ejection fraction [RVEF] = 12%) to better define the need for biventricular or total artificial heart support vs univentricular support (LVAS). Clinical factors including preoperative inotropic need, fever without detectable infection, radiographic diffuse pulmonary edema, postoperative blood transfusion and RV thickness were compared with hemodynamic parameters including cardiac index, RVEF, CVP, mean PAP, and total pulmonary resistance for their ability to predict need for mechanical or high dose inotropic support for the RV. Patients were grouped as to the need for RV support following LVAS implantation: None (I)=14, inotropic drugs (II)=7, and RV mechanical support (III)=10. There were no differences in preimplant hemodynamic variables. Groups II and III had a significantly lower mixed venous oxygen saturation (39.2% vs 52.4% in Group I; $p<0.001$), a greater level of inotropic need ($p<0.02$), greater

impairment of mental status, and a lower ratio of RVEF to inotropic need (0.37 vs 0.56 for Group I; $p<0.02$) before LVAS implantation. A significant discriminator between Groups II and III was the presence of a fever without infection within 10 days of LVAS implant (14% in Group II vs 80% in Group III). Group III had more patients with preimplant pulmonary edema on chest radiograph and a greater requirement for postoperative blood transfusion (6 units cells in Group II vs 38 in Group III). RV thickness at LVAS explant was 0.87 cm in Group III vs. 44 cm in Group II ($p<0.05$). Transplantation rates after bridging were 100% in Group I, 71 % in Group II and 50% in Group III. Clinical factors which reflect preimplant degree of illness and perioperative factors which result in impairment of pulmonary blood flow or reduced perfusion of the right ventricle after LVAS implantation are now felt to be more predictive of the need for additional RV support than preimplant measures of RV function or hemodynamics.

12:10pm ADJOURN

*By invitation

9:30 am SIMULTANEOUS SCIENTIFIC SESSION E

GENERAL THORACIC SURGERY

Republic Ballroom, Sheraton Boston Hotel

Moderators: Victor F. Trastek, M.D.

Martin F. McKneally, M.D.

48. RESULTS AND PROGNOSTIC FACTORS IN

RESECTIONS OF PRIMARY TRACHEAL TUMORS

J.F. Regnard, M.D.*, P. Fourquier, M.D.* and Ph.Levasseur, M.D.*

Paris, France

Sponsored by: Peter C. Pairolero, M.D., Rochester Minnesota

From 1970 to 1993, 208 patients with primary tracheal tumors were evaluated in a multicentric retrospective study including 26 centers. Ninety-four patients had a squamous cell carcinoma of the trachea, 4 had adeno-carcinoma, 65 had adenoid cystic carcinoma and 45 patients, miscellaneous tumors. The following resections were performed: tracheal resection with primary anastomosis, 146; partial tracheal resection, 19; carinal resection, 24; and tracheo-laryngeal resection, 19. In average, the length of the resected specimen was 38.5 mm (10-60). Postoperative complications occurred in 41% and postoperative mortality was 10.5%. Postoperative morbidity and mortality were statistically correlated (PO.05) to the length of the resection, to the need of a laryngeal release, to the type of resection and to the histologic type. Follow-up was complete for all but 10 patients with a mean follow-up of 5 years. Fifty-nine percent of the patients with tracheal cancers and 43% of the patients with adenoid cystic carcinomas had postoperative radiotherapy.

In complete resection, the 5 and 10-year actuarial survival were respectively 55% and 30% in tracheal cancers, 82% and 25% in adenoid cystic carcinomas. Long-term survival was altered by the occurrence of second primary cancer (ORL, lung) in the group of patients with tracheal cancers and by late metastases in adenoid cystic carcinomas. Postoperative radiotherapy did not improve survival in the group of patients with tracheal cancer when the resection was complete, even if nodes were involved. However, in case of incomplete resection, postoperative radiotherapy did not improve survival in the group of patients with tracheal cancer when the resection was complete, even if nodes were involved. However, in case of incomplete resection, postoperative radiotherapy

improved survival (P<0.02). For adenoid cystic carcinomas, the actuarial survival seemed better in complete resection than in incomplete resection but the difference was not statistically significant (PO.20). Postoperative radiotherapy did not improve survival in adenoid cystic carcinoma, even in incomplete resection.

Surgery is the treatment of choice of primary tracheal tumors.

*By invitation

49. THE IMPORTANCE OF SURGICAL STAGING IN THE TREATMENT OF MALIGNANT PLEURAL MESOTHELIOMA

Valerie W. Rusch, M.D. and Ennapadam Venkatraman, Ph.D.*

New York, New York

Progress in the therapy of malignant pleural mesothelioma (MPM) is hindered by the lack of an adequate staging system and controversy about prognostic factors. This personal surgical series was analyzed to determine the value of a new TNM staging system proposed by the International Mesothelioma Interest Group (IMIG), and to examine factors that could affect the design of future clinical trials.

Methods: Thoracotomy was done in previously untreated patients (pts) if CT scans showed technically resectable tumor confined to one hemitnorax. Pleurectomy/decortication (P/D) was used in pts with minimal visceral pleural tumor, extrapleural pneumonectomy (EPP) in pts with more locally advanced disease. Complete resection was defined as no gross residual tumor. Adjuvant therapy was given as required by serial clinical trials. Pts were followed by chest and abdomen CT scans every 3 months after death. Prognostic factors were examined by log rank and Cox regression analyses.

Results: From 10/83 to 7/94 there were 131 consecutive thoracotomies, 101 resections, 72 complete. **EPP:P/D=50:51.** Men:women=108:23. Median age=63 years (range=32-80). Operative mortality 5/131 pts (3.8%), 3/50 EPP (6%). Most frequent morbidity: atrial arrhythmias, 14/131 pts (11%), 11/50 EPP (22%). 95/131

tumors were epithelial. 51/89 pts (57%) having node dissections had (+) nodes, 45 (50%) N2.

Survival	Operation		T status				N status		S	
	EPP	P/D	Tl	T2	T3	T4	NO	Nl-3	I	
Median (mos)	9.9	18.3	27	12	13	6.5	18.3	9.4	35	

By univariate analysis, type of resection (EPP vs P/D), T and N status, stage, histology and adjuvant therapy but not gender or age, significantly affected survival. Type of resection, stage and histology were significant in a multivariate analysis. 99 known sites of relapse occurred in 60 pts. In EPP pts these were local in 5/30 (16%) and distant in 32/48 (66%) instances.

Conclusions: (1) N2 nodal disease is more frequent than previously reported; (2) The prognostic importance of histology is confirmed; (3) Both T and N status significantly influence outcome, and the IMIG staging system successfully identifies poor prognosis pts; (4) despite more locally advanced disease in most EPP pts, it provided better local control than P/D but failed to improve survival because of distant metastatic disease. Contrary to past practice, future clinical trials should stratify for histology, must control for TNM stage and must consider the impact of type of surgical resection on the pattern of relapse.

*By invitation

50. ASSESSMENT OF PREOPERATIVE ACCELERATED RADIOTHERAPY AND CHEMOTHERAPY IN STAGE IIIA NON-SMALL CELL LUNG CANCER (NSCLC)

Douglas J. Mathisen, M.D., Noah Choi, M.D.*, Robert Carey, M.D.*, John C. Wain, M.D.*, Alan Hilgenberg, M.D., William Daly, M.D.*, Ashby Moncure, M.D.*, Cameron Wright, M.D.*, Thomas Lynch, M.D.* and Hermes Grillo, M.D.

Boston, Massachusetts

Mediastinoscopy proven N2 disease in non-small cell lung cancer carries a poor prognosis (less than 10%) with surgery and radiation therapy. Neoadjuvant therapy offers the promise of improved results. The optimum regimen has yet to be found. We have utilized preoperative accelerated radiotherapy (RT) (42Gy in 2 sessions - 21Gy in 1.5Gy bid, 5 days per week in 2 5-day-intervals) cisplatin 100mg/m^2 days 1 and 29, Velban 3mg/m^2 days 1 and 3 and 29 and 31, and 5-FU 30mg/kg/day by continuous infusion

days 1 to 3 and 29 to 31. Surgery was performed on day 56. Postoperative therapy consisted of 1 cycle of chemotherapy and 15 to 18Gy of accelerated RT.

The patient age ranged from 38 to 77 years, and male to female ratio was 2.3:1. The tumor stages included T1N2MO 5, T2N2MO 28, T3N2MO 3, and T4N2MO 2 pts. Histologic types consisted of squamous cell carcinoma 14, adenocarcinoma 13, and large cell carcinoma 8.

Major clinical response (> 50% reduction in tumor volume) was obtained in 92% (35/38) of pts. Curative resection was performed in 92% (35/38) of pts with their resection margins being negative in 82% (31/38) and microscopically positive in 11% (4/38). Tumor down staging was achieved in 70% of pts. Two patients were unable to undergo surgery because of distant metastasis. Moderate dysphagia and leukopenia were noted in 50% of patients. The mortality of this regimen was 8% (3/38): pulmonary embolism during induction CT+RT, 1 and postoperative death, 2. The median survival time was 24M and their survival rates by Kaplan-Meier method were 50%, 43% and 43% at 2, 3, and 5 years, respectively.

Control of local-regional tumor was obtained in 89% (34/38) of patients. This regimen offers superior resectability rates and improved survival at 2, 3, and 5 years.

*By invitation

51. MANAGEMENT OF UNILATERAL VOCAL CORD PARALYSIS IN INTRATHORACIC MALIGNANCY

Muhammed K. Ali, M.D.*, Dennis H. Kraus, M.D.*, Robert J. Ginsberg, M.D., Christopher J. Hughes, M.D.*, Valerie W. Rusch, M.D, Michael E. Hurt, M.D. and Patricia M. McCormack, M.D.

New York, New York

Patients with unilateral vocal cord paralysis (UVCP) from intrathoracic malignancies can suffer from significant dysfunction of speech, swallowing, ventilation, and the ability to cough effectively unless the non-paralysed cord can accommodate. In patients with already compromised pulmonary function, aspiration can be a life threatening event. Acute post-operative UVCP can also be life threatening, the paralyzed vocal cord interfering with

effective cough, endogenous PEEP, and the ability of the patients to protect the airway from acute aspiration events. In symptomatic patients medialization of the affected vocal cord allows effective glottal closure and immediately improves dysfunction.

Materials and Methods: Fifty-seven patients with intrathoracic malignancies required surgical correction of UVCP between 1991 and 1994. Primary pathology included lung cancer (46), esophageal cancer (8), and miscellaneous tumors (3). Paralysis was due to invasion of the vagus/recurrent laryngeal nerve in 32 and as result of the surgical procedure in 25. There were 46 left-sided paralyses and 11 right-sided. Presenting symptoms included hoarseness (56), dyspnea (18), aspiration (24), weight loss (16), and pneumonia (12). Surgical procedure consisted of silastic medialization (44), temporary Gelfoam injection (5), and Teflon injection (8) to medialize the affected cord. In 9 patients, the surgery was performed in the acute post-operative setting, the others being done electively (48).

Results: Of the presenting symptoms, the following table summarizes the results:

	# Pts	# Improved	%. Improved
Hoarseness	56	49	88%
Dyspnea	18	14	78%
Dysphagia	14	13	93%
Aspiration	24	20	83%
Pneumonia	12	11	92%
Wt Loss	16	7	44%

Overall success of the intervention was 52 of 57 patients (91%). All 9 patients treated in the acute setting were immediately improved. Minor complications including transient airway compromise (3), tracheostomy (1), tooth fracture (1), hematoma (1), foreign body sensation (2), and transient laryngeal edema (6), occurred in 23% of patients. Currently, 33

patients are alive, only one of whom had an unsuccessful long-term result.

Conclusions: Surgical management of UVCP in patients with intra-thoracic malignancies prevents life threatening pulmonary complications in the acute post-operative setting. In chronic situations it provides patients with improved speech, swallowing, and pulmonary function, resulting in improved quality of life, including patients not cured of disease.

10:50 am INTERMISSION

*By invitation

11:10 am SIMULTANEOUS SCIENTIFIC SESSION E

GENERAL THORACIC SURGERY

Republic Ballroom, Sheraton Boston Hotel

Moderators: Victor F. Trastek, M.D.

Martin F. McKneally, M.D.

52. IMPROVED AIRWAY HEALING AFTER LUNG TRANSPLANTATION - AN ANALYSIS OF 348 BRONCHIAL ANASTOMOSES

Hiroshi Date, M.D.*, Elbert P. Trulock, M.D.*, Joseph Arcidi, M.D.*, Sudhir Sundaresan, M.D., Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.

St. Louis, Missouri

Although less commonly encountered than in the early days of lung transplantation, airway complications following lung transplantation remain a significant cause of morbidity. We evaluated various clinical factors to identify predictors of airway complication following lung transplantation. Two

hundred and twenty-nine consecutive single (SLT, n=110) and bilateral (BLT, n=119) lung transplants were performed in our center between September 1988 and August 1994. These 348 bronchial anastomoses were retrospectively analyzed. Airway complication requiring clinical intervention occurred in 33 anastomoses (9.5%), 29 patients (12.8%). Conservative therapy such as dilation, stent and/or laser resulted in excellent improvement in 22 patients. There were five deaths (2.2%) attributable to airway complications.

Recipient factors (age, sex, diagnosis, preoperative steroid use), donor factors (age, sex, Pa02, sputum gram stain, ischemic time), operation factors (side of transplant, use of cardiopulmonary bypass, type of anastomosis [end-to-end vs. telescope], type of wrapping [omentum vs. peribronchial tissue vs. pericardial fat], suture material) and postoperative factors (Pa02, mean PAP, method of steroid administration, biopsy proved rejection, CMV status, CMV pneumonitis) were not predictors of airway complication. Mattress suture technique (21/153=13.7%) was associated with more frequent complications than was simple interrrupted (8/122=6.6%) or figure of eight suture (4/73=5.5%), (p=0.02). Complication occurred more often in SLT (16/110=14.4%) than in BLT (17/238=7.1%, p=0.02) Patients who subsequently developed airway complications required longer mechnical ventilation than those without airway complications (12.9 ± 18.4 days vs. 5.5 ± 20.5 days, $p<0.01$).

The chronologic incidence of airway complications was evaluated by separating the 229 transplants into three groups: Phase I, the first 77 transplants; Phase II, the next 76 transplants; and Phase III, the last 76 transplants. The airway complication rate per anastomosis was significantly lower in Phase III (5/126=4.0%) than in Phase I (12/110=10.9%) and Phase II (16/112=14.3%) (pO.Ol). All parameters noted above were similar for the three phases. However, immunosuppression and rejection monitoring was different in Phase III. Early low dose corticosteroid

administration, induction cytolytic therapy with antithymocyte globulin, and rejection therapy based on histologic (transbronchial lung biopsy) rather than clinical diagnosis were routinely employed in Phase III.

Mattress suture technique, SLT and prolonged mechanical ventilation are predictors of airway complication. The majority of airway complications are successfully treated and are rarely fatal. The recent reduction in incidence of airway complications is likely due to better maintenance immunsuppression and rejection surveillance.

*By invitation

53. UNILATERAL THORACOSCOPIC SURGICAL APPROACH FOR DIFFUSE BULLOUS EMPHYSEMA

Robert J. Keenan, M.D.*, Rodney J. Landreneau, M.D., Frank C. Sciurba, M.D.*, Peter F. Person, M.D.*, Lynda S. Fetterman, M.D.*, Claudia M. Bowers, B.S.N.* and Hartley P. Griffith, M.D.

Pittsburgh, Pennsylvania

We are currently evaluating the use of a lateral thoracoscopic approach to improve lung function in patients with diffuse bullous emphysema. Patients entered into the study include those with end-stage emphysema recalcitrant to medical therapy and with disabling dyspnea based on a Baseline Dyspnea Index. Preoperative testing included pulmonary function testing with exercise oxygen consumption studies, chest x-ray, CT scan, quantitative ventilation-perfusion scan, transthoracic echocardiography and six-minute walk. Forty-two consecutive patients (29 males, 13 females) with a mean age of 63.4 years (range 36-78

years) underwent surgery for type III or IV bullous emphysema with 81% requiring preoperative supplemental oxygen. Operative side was determined by pre-operative imaging (right 56%, left 44%). Operative procedure was laser ablation in 10 pts, stapler resection in 11 pts and combined laser/stapler in 21 pts. Chest tube duration averaged 10 days (range 3-50 days) with median length of hospital stay being 13 days (range 6-99 days). Four pts (9.5%) required conversion to open thoracotomy to control air leaks and 5 pts (12%) were treated for pneumonia; 1 pt suffered a significant postoperative stroke. Three pts (7%) died from cardiopulmonary failure. Among the 39 survivors, 31 were discharged within 30 days and 28 returned for evaluation at 3 weeks. Seven pts experienced prolonged hospitalization due to cardiopulmonary problems and have not undergone postoperative testing. Significant ($p<0.005$) improvements were seen in FVC (2.7L post vs. 2.31L pre) and FEVj (1.05L post vs. 0.79L pre) with 18/28 pts (64%) showing a >20% improvement and only 1 pt experiencing a decline of >10%. At the time of this analysis, 9 pts had undergone their procedures greater than 3 months previously. Significant ($p<0.05$) increases were seen in postoperative FEV] (average improvement of 74.8%) and DLCO (average increase of

84%); pOj and pCO2 did not change. The significance of hypercarbia and/or reduced DLCO (Burrows predicted equation corrected for Hgb) as predictors of poor outcome was examined. Six/ten pts (60%) with poor outcome (death or hospitalization>30 days) demonstrated hypercarbia (pCO2 ^ 50 torr) or reduced DLCO (< 25%) compared to only 5/28 (17.9%) of the remaining pts (p=0.0193). The combination of hypercarbia and reduced DLCO was significantly more frequent (p=0.0026) in pts with a poor outcome and was 86% specific (5/6 pts) in identifying serious postoperative risk. We conclude that the lateral thoracoscopic surgical approach to diffuse bullous emphysema offers significant improvement in pulmonary function for many pts but that pts with a combination of hypercarbia and reduced DLCO should not be offered this procedure because of significant perioperative risk.

*By invitation

54. SURGICAL TREATMENT OF DIFFUSE EMPHYSEMA. A RANDOMIZED, PROSPECTIVE TRIAL OF LASER BULLECTOMY VERSUS LUNG REDUCTION SURGERY WITH STAPLES AND BOVINE PERICARDIUM

Robert J. McKenna, Jr., M.D.*, Matthew Brenner, M.D.*, Michael J. Mullin, M.D.*, Narindar Singh, M.D.* and Michael Berns, Ph.D.*

Los Angeles, Orange, Tustin and Irvine, California

Sponsored by: Quentin R. Stiles, M.D., Los Angeles, California

Both the efficacy and the optimal method for surgical treatment of diffuse emphysema are controversial. This is an interim analysis of a randomized, prospective study of laser bullectomy versus lung reduction surgery with staples and the bovine pericardial patch. All patients had diffuse emphysema with no bulla greater than 2 cm in size, and underwent the surgical treatment of one lung by thoracoscopy. 21 patients underwent laser bullectomy with a 10-watt contact tip YAG laser (average 24, 200 Joules, range 16.5K-29.1K) and 23 patients underwent lung reduction surgery (average 8 firings of the endoscopic 60 mm stapler). The average length of stay was 11.4 days for laser and 12.4 days for staples. There was no mortality for the laser and 1 mortality (2%) for the staple group due to contralateral tension pneumothrorax postoperatively. Complications included prolonged air leak at least 10 days in 7 laser cases (33%) and 8 staple cases (35%). The average hospital charge was $44, 482 for laser and $57, 858 for staples.

Multivariate analysis showed prolonged length of stay was related to adhesions found at surgery and the use of the staples. Major clinical improvement (defined as significantly increased activity, decreased pulmonary medicines, termination of prednisone, and the termination or significant reduction of oxygen therapy) was seen in 11 laser cases (52%) and 13 staple cases (57%). Some clinical improvement (increased activity with decreased oxygen) was seen in 6 laser cases (29%) and 4 staple cases (17%), and no improvement in 4 laser cases (19%) and 5 staple cases (22%). Poor outcome was related to poor diffusion capacity (<10%) and the presence of emphysema worse in the lower lobe than in the upper lobe.

12:10 pm ADJOURN

*By invitation

9:30 am SIMULTANEOUS SCIENTIFIC SESSION F

CONGENITAL HEART DISEASE

Independence Ballroom, Sheraton Boston Hotel

Moderators: Edward L. Bove, M.D.

John L. Myers, M.D.

55. SURGICAL APPROACHES FOR DOUBLE OUTLET RIGHT VENTRICLE OR TRANSPOSITION OF THE GREAT ARTERIES ASSOCIATED TO STRADDLING ATRIOVENTRICULAR VALVES.

‡Alain Serraf, M.D.*, Tomohiro Nakamura, M.D.*, Francois Lacour-Gayet, M.D.*, Dominique Piot, M.D.*, Jacqueline Bruniaux, M.D.*, Anita Touclot, M.D.*, Miguel SousaUva, M.D.*, Lucile Houyel, M.D.* and Claude Planche, M.D.

Le Plessis Robinson, France

The surgical management of pts with DORV or TGA and straddling AV valves remains controversial. Biventricular repair (BVR) has theoretical advantages because it establishes normal anatomy and physiology; however, in some instances it seems to carry a too high operative risk and a univentricular heart repair (UVH) is preferred. Since 1984, 34 patients with DORV (15) or TGA (19) with isolated straddling tricuspid valve (10), isolated straddling mitral valve (8), both mitral and tricuspid valves straddling (2) or abnormal insertion of tricuspid (10) or mitral (2) chordae in the left ventricular outlet precluding an adequate tunnel construction were operated on. Straddling were categorized according to the location of the papillary muscle (PM) insertion in the opposite ventricular chamber: type A, on the edge of the VSD

(10); type B, on the opposite side of the ventricular septum away from the edge of the VSD (7); type C, on the free wall of the opposite ventricular chamber (3). Abnormal chordal inertion were classified according to the location of their attachments around the edges of the VSD. There were 3 types of chordal distribution: on the aortic conus, on the pulmonary conus crossing the VSD, or around the VSD closing it like a curtain. All but 1 patient had 2 adequately sized ventricles. Sixteen pts underwent palliation. Median age at definitive surgery was 6.5 months (range: 1-130). Thirty pts underwent a BVR, whereas 4 had UVH repair. BVR was carried out by means of arterial switch operation in 18 and tunnel construction from the left ventricle to the aorta in 12. In isolated straddlings type A and B, closure of the VSD was performed by adjusting the septal patch on the ventricular side above the straddled PM. In type C, the patch was oversewn over the PM by applying it on the septum. In double straddling, the ventricular septum was incised between the 2 PM and an ellipsoid patch was used to reconstruct the septal defect rejecting each subvalvar apparatus in its own ventricular chamber. When insertion of abnormal chordae in the left outflow tract was on the aortic conus or on the pulmonary conus, they were tailored in order to make a flap leaving an unobstructed left ventricular outflow

tract. In 2 pts, the subvalvar apparatus was resected and reattached to the patch. Curtain-like chordae were a contraindication to BVR in DORV but not in TGA. There were 4 early and 1 late deaths, all occurring in the BVR group. Death was due to myocardial ischemia (1), to RV hypoplasia (1), to pulmanary hypertension (1) and to residual subaortic stenosis (1). Two pts demonstrated moderate to severe postoperative AV valve incompetence, once due to a cleft in the mitral valve. Three pts were reoperated on for SAS (1), PS (1) and MR (1). Mean follow-up of 27.4 ± 19.3 months was achieved in all survivors. All but one (UVH repair) were in NYHA class 1, without AV valves incompetence. Five years actuarial survival was 85.3 ± 3%. It is concluded that straddling or abnormal distribution of chordae tendinae of the AV valves do not preclude BVR in DORV or TGA provided that ventricular sizes are adequate. Curtain like abnormal tricuspid chordae remain a contra indication to BVR in DORV.

‡1993-94 Graham Fellow

*By invitation

56. AORTICOVENTRICULOPLASTY USING PULMONARY AUTOGRAFT: THE "ROSS-KONNO" PROCEDURE

V. Mohan Reddy, M.D.*, J.A.M. vanSon, M.D.*, Hiranya A. Rajasinghe, M.D.* and Frank L. Hanley, M.D.*

San Francisco, California

Sponsored by: Benson B. Roe, San Francisco, California

Background: For patients with hypoplastic aortic annulus and severe diffuse subaortic stenosis various aorticoventriculoplasty procedures (e.g., Konno) have been associated with less than optimal results. Younger patients often require reoperation for valve replacement. Pulmonary autograft is being increasingly used for aortic valve replacement. Long term follow up shows that the pulmonary autograft functions well as the systemic arterial (neo-aortic) valve.

Methods and results: We have extended the application of pulmonary autograft to patients with complex left ventricular outflow tract (LVOT) obstruction. Since July 1992 through October 1994, 7 patients (age: 3 weeks to 19 years) have undergone plumonary autograft replacement in association with aorticoventriculplasty (Ross-Konno procedure). The diagnoses were aortic stenosis (AS) and insufficiency with subaortic stenosis (n=4), Shone's complex (n=2), and interrupted aortic arch with subAS (n=1). On average each patient underwent 2.2 previous operations including a previous Konno procedure in one patient. The aortic valve was replaced with a pulmonary autograft conduit. The subannular left ventricular outflow tract was enlarged with a dacron patch in 4 patients and with right ventricular infundibular muscular patch in 3 patients. At the time of harvesting the pulmonary autograft, a portion of the right ventricular infundibular wall was included. This infundibular muscular patch was used for enlarging the LVOT. With this procedure the use of prosthetic valve as well as prosthetic patch is avoided. Intraoperative transesophageal echocardiographic assessment revealed no AI. One patient had a residual LVOT gradient of 15 mmHg. Significant complications were cardiac tamponade from bleeding (1 pt) and complete heart block requiring a permanent pacemaker (1 pt). Follow up ranged from 2 months to 12 months. There have been no early or late deaths. Aortic valve function and LVOT was evaluted by echocardiography. Three patients had trivial AI and none of the patients had LVOT obstruction.

Conclusions: Initial experience suggests that aorticoventriculoplasty using pulmonary autograft is an excellent alternative for patients with complex LVOT obstruction. Since pulmonary autograft has been shown to

grow, future reoperations may not be required in young patients.

*By invitation

57. RESULTS OF AN AUTOLOGOUS TISSUE RECONSTRUCTION FOR REPLACEMENT OF OBSTRUCTED EXTRACARDIAC CONDUITS

Robert J. Cerfolio, M.D.*, Gordon K. Danielson, M.D., Francisco J. Puga, M.D, Hartzell V. Schaff, M.D. and Carole A. Warnes, M.D.*

Rochester, Minnesota

Between May 1983 and August 1, 1994, 42 patients having replacement of an obstructed pulmonary ventricle-to-pulmonary artery conduit underwent an autologous tissue reconstruction in which a prosthetic roof was placed over the fibrous tissue bed of the explanted conduit. The roof was constructed with xenograft pericardium (most recently) (n=35), homograft dura mater (n=5), or dacron (n=2). The explanted conduits were: Hancock (n=28), Tascon (n=6), dacron tube (n=3), homograft (n=3), and others (n=2). There were 26 males and 16 females, and the ages ranged from 5 to 34 years (median=16). Diagnoses included transposition of the great arteries (n=15), pulmonary atresia (n=11), truncus arteriosus (n=8), tetralogy of Fallot (n=4), double outlet right ventricle (n=3), and corrected transposition (n=1). The original conduit sizes ranged from 8 to 25 mm (median=22). Preoperative peak

systolic gradients by cardiac catheterization or echocardiography ranged from 40 to 140 mmHg (median=79). Valves were placed in 13 reconstructions (2 were mechanical); no valve was inserted in the others. It was possible to place large valves in the autologous tissue reconstructions, ranging from 23 to 29 mm (median=25). Thirty-two concomitant procedures were performed in 25 patients which included pulmonary angioplasty (n=11), closure of residual VSD (n=9), tricuspid annuloplasty (n=5), closure of residual ASD (n=2), aortic valve replacement (n=2), and others (n=3). Cardiopulmonary bypass time ranged from 34 to 223 minutes (median=84) and the aortic cross-clamp time ranged from 0 (in 28 patients) to 109 minutes (median=0). Five patients required partial sternal resection to prevent conduit compression. There was no early mortality. The total hospital stay ranged from 6 to 23 days (median=9). Post repair peak systolic gradients from pulmonary ventricle to pulmonary artery ranged from 0 to 36 mmHg (median=13). Follow-up was complete in all patients and ranged from 5 months to 10.6 years (median-6.1 years). There was no late mortality. Three patients with non-valved conduits required reoperation for aortic (truncal) valve insufficiency, tricuspid valve insufficiency, and defibrillator

implantation, respectively. None of the autologous tissue conduits developed an obstructive peel or valve obstruction. At 8 years, the acturial freedom from reoperation for conduit obstruction was 100%, and freedom from reoperation for any cause was 97%. This technique simplified conduit replacement, allows for a generous-sized outflow tract, has a low risk, and yields late results which appear superior to those of cryopreserved homografts or other types of extracardiac conduits.

*By invitation

58. UNIVENTRICULAR REPAIR - EARLY AND MIDTERM RESULTS

Rajeesh Sharma, M.Ch.*, Krishna S. Iyer, M.Ch.*, Balram Airan, M.Ch.*, Kamales Saha, M.S.*, Bhabha Das, M.Ch.*, Anil Bhan, M.Ch.*, I. M. Rao, M.Ch.* and P. Venugopal, M.Ch.*

New Delhi, India

Sponsored by: Richard A. Jones, M.D., Boston, Massachusetts

Two hundred and two (62 tricuspid atresia and 140 non-tricuspid atresia) patients underwent univentricular repair at our unit, from January 1990 to September 1994. 182 of these patients had nonfenestrated while 20 had fenes-trated interatrial baffles. Early mortality was 15.9% (29/182) in the nonfenestrated and 5% (1/20) in the fenestrated group. Follow-up period ranged from 2 months to 58 months. There were 7 late deaths and 5 patients were lost to follow-up. Out of 160 patients, who have been evaluated in the out-patient department in the last 3 months, 142 (88.75%) were off all medications and were in functional class-I.

Risk factors analyzed for early mortality and significant effusion were: age, preoperative diagnosis, type of Fontan

modification, duration of cardiopulmonary bypass (CPB) and aortic cross clamp, pulmonary artery size, associated pulmonary arterioplasty, takedown of systemic to pulmonary artery shunt and pulmonary artery debanding along with Fontan operation. CPB time exceeding 120 minutes was associated with higher early mortality (12/47 vs 18/155 p value = 0.0187). CPB time exceeding 120 minutes (p value = 0.0456) and aortic cross clamp time exceeding 60 minutes (p value = 0.0278) were associated with significant effusion postoperatively. Other factors were not associated with any significantly increased risk for early mortality or postoperative effusions.

Fenestration of interatrial baffle appeared to decrease early mortality although the numbers are too small to be statistically significant. There was no difference in the incidence of effusions in the fenestrated and non-fenestrated group.

10:50 am INTERMISSION

*By invitation

11:10 am SIMULTANEOUS SCIENTIFIC SESSION F

CONGENITAL HEART DISEASE

Independence Ballroom, Sheraton Boston Hotel

Moderators: Edward L. Bove, M.D.

John L. Myers, M.D.

59. SUBAORTIC STENOSIS IN THE SPECTRUM OF ATRIOVENTRICULAR SEPTAL DEFECTS: SOLUTIONS MAY BE COMPLEX AND PALLIATIVE

Glen S. Van Arsdell, M.D.*, William G. Williams, M.D, George A. Trusler, M.D, John G. Coles, M.D, Ivan M. Rebeyka, M.D.* and Robert M. Freedom, M.D.*

Toronto, Ontario, Canada

From November 1982 through September 1994 seventeen children with atrioventricular spetal defects (AVSD's) underwent 24 operations for subaortic stenosis. Age at initial subaortic resection was 17 months to 13 years (6.2±3.7). Specific diagnosis included 9 transitional AVSD's, *5* septum primum defects, 1 complete AVSD, 1

inlet VSD with malattached chordae, and 1 complete AVSD with tetralogy of Fallot. Eight patients had Down's syndrome. All but 2 operations were performed in this institution. Indications for a subaortic procedure were subaortic stenosis at initial operation (2), severe subaortic stenosis (12), or progressively worsening subaortic stenosis (3). Two patients had resection at initial repair treated by fibromyectomy (1) and fibrous membrane resection with minor papillary muscle excision (1). Fifteen patients had sub-aortic resection following AVSD repair. Average time to resection was 4.7y ± 3.4. Pathology included isolated fibromuscular stenosis (8), combined fibromuscular stenosis and mitral valve abnormalities (5), fibromuscular stenosis and papillary muscle obstruction (1), and tunnel stenosis (1). Fourteen of these patients had fibromyectomy and 1 an apicoaortic conduit. Additionally, there was papillary muscle thinning, mitral valve replacement, pericardial patch augmentation of the aortic mitral continuity, and resection of accessory chordae and tissue of the left AV valve.

Six patients (35%) required 8 reoperations for previously resected sub-aortic stenosis: 2 fibromyectomies, 2 radical fibromyectomies with VSD creation and infundibular patch, 1 fibromyectomy with papillary muscle thinning, 1 conduit replacement, and 9 years following the initial conduit, successful conversion to antegrade flow by radical subaortic resection, removal of accessory chordae and tissue of the left AV valve and conduit excision.

Follow-up ranges from 10 days to 9 years (median 3.2y). There were no major cardiac morbidities and no mortalities. Gradients are not significant in 10 patients (0-10 mmHg), mild in 5 patients (11-20 mmHg), and moderate in 2 patients (21-50 mmHg). The treatment of subaortic stenosis associated with AVC defects can be complex and only palliative. Aggressive resections at the initial presentation should be considered.

*By invitation

60. SURGICAL MANAGEMENT OF THE LEFT ATRIOVENTRICULAR VALVE IN COMPLETE ATRIOVENTRICULAR SEPTAL DEFECTS: A TWENTY-YEAR EXPERIENCE

‡Ko Bando, M.D.*, Mark W. Turrentine, M.D.*, Kyung Sun, M.D.*, Thomas G. Sharp, M.D.*, Andrew P. Miller, B.S.*, Robert S. Binford, M.D.*, Kenneth A. Kesler, M.D.*, Gregory J. Ensing, M.D.*, Timothy M. Cordes, M.D.*, Donald A. Girod, M.D.*, Roger A. Hurwitz, M.D.*, Randall L. Caldwell, M.D.*, Joyce Hubbard, M.D.*, Harold King, M.D. and John W. Brown, M.D.

Indianapolis, Indiana

Background and Purpose: Creation of a competent left atrioventricular (AV) valve is a cornerstone in surgical repair of complete AV septal defects. The purpose of this study was to identify risk factors for mortality and failure of left AV valve repair and to determine the impact of "cleft" closure on postoperative AV valve function.

Method: Two hundred patients operated between January 1974 and October 1994 were included in this study. Using univariate and multiple regression analysis, risk of early and late mortality and need for reoperation for postoperative left AV valve regurgitation was estimated.

Results: Overall early mortality was 8% (16/200 patients). Operative mortality decreased significantly over the period of the study from 19% (4/21) before 1980 to 3% (2/64) after 1990 (p=0.03). By multiple logistic regression, risk factors for early death included double orifice left AV valve (p<0.001) and post-operative pulmonary hypertensive crisis (/><0.001). At a mean follow-up of 94 months, late survival was 99% (182/184 operative survivors); all are NYHA class I or II. Preoperative AV valve regurgitation was assessed in 198 patients by angiography or echocardiography and was absent or mild in 103 (52%), moderate in 81 (41%), and severe in 16 (8%). Left AV valve "cleft" was closed in 93% (186/200), but left alone when valve leaflet tissue was inadequate and closure of "cleft" might cause significant stenosis. More complex valvuloplasty was required in 4 patients. Reoperation for severe postoperative left AV valve regurgitation was necessary in 8 patients (4%); 6 patients initially did not have closure of "cleft" and 2 patients had "cleft" closure. Six patients had reoperation with annuloplasty and 2 patients required left AV valve replacement. Five patients survived reoperation and are currently NYHA class I or II. On most recent evaluation assessed by angiography or echocardiography (a mean of 59 months after repair), left AV valve regurgitation was absent or mild in 129 of the 138

survivors (93%) examined; none had moderate or severe left AV valve stenosis. Preoperative severe AV valve regurgitation ($p<0.001$) and double orifice AV valve ($p<0.001$) predicted reoperation for residual left AV valve regurgitation.

Conclusion: These results indicate that complete AV septal defect can be repaired with low mortality and good mid- to long-term results. Routine approximation of the cleft is safe and has a low incidence of reoperation for left AV valve insufficiency.

‡1991-92 Graham Fellow

*By invitation

61. RESULTS OF NORWOOD OPERATION FOR LESIONS OTHER THAN HYPOPLASTIC LEFT HEART SYNDROME

Marshall L. Jacobs, M.D.*, Jack Rychik, M.D.*, John D. Murphy, M.D.*, Susan C. Nicolson, M.D.*, James M. Steven, M.D.* and William I. Norwood, M.D.

Philadelphia, Pennsylvania

Norwood's operation provides satisfactory palliation for neonates with hypoplastic left heart syndrome. The dominant physiologic features of hypo-plastic left heart syndrome, ductal dependency of the systemic circulation and parallel pulmonary and systemic circulations, are shared by a multitude of other less common congenital heart malformations. Theoretically, these should be equally amenable to palliation by Norwood's operation. From January 1990 through June 1994, 60 neonates with malformations other than hypoplastic left heart syndrome have undergone initial surgical palliation by Norwood's procedure. Diagnoses include single left ventricle with L-transposition of the great arteries (12), critical aortic stenosis (8), complex double outlet right ventricle (8), interrupted aortic arch with ventricular septal defect and subaortic stenosis (7), ventricular septal defect, subaortic stenosis, and coarctation of the aortia (7), aortic atresia with large ventricular septal defect (6), tricuspid atresia with transposition of the great arteries (6), heterotaxy syndrome with subaortic obstruction (3), and other (3). There were 10 hospital deaths and 50 survivors (83% survival). After the introduction of inspired carbon dioxide

into the postoperative management protocol (1991), 42 of 47 patients survived (89% survival). Mortality is independent of diagnosis, and essentially the same as for hypoplastic left heart syndrome. With minor technical modifications, Norwood's operation provides satisfactory initial palliation for the wide variety of malformations characterized by ductal dependency of the systemic circulation, in anticipation of either a Fontan procedure or a biventricular repair.

12:10 pm ADJOURN

*By invitation

GEOGRAPHICAL ROSTER

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NECROLOGY
Vladimir I. Burakovsky, M.D. Moscow, Russia
Preston R. Cutler, M.D. Salt Lake City, Utah
Norman Delarue, M.D. Toronto, Ontario
William B. Ford, M.D. Pittsburgh, Pennsylvania
Cranston W. Holman, M.D. New York, New York
Alfred Hurwitz, M.D. Liberty, Maine
Adrian Lambert, M.D. New York, New York
Harvey J. Mendelsohn, M.D. Pepper Pike, Ohio
Dennis M. Rosenberg, M.D. New Orleans, Louisiana
William H. Sewell, M.D. Sayre, Pennsylvania
Horace C. Stansel, M.D. Woodbridge, Connecticut
E.J. Zerbini, M.D. Sao Paulo, Brazil

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(Listed by Countries, States, Provinces and Cities)
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El Cajon
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Plum, James R
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Read, C Thomas
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Copeland, Jack G, III
Sanderson, Richard O
Sethi, Gulshan K
ARKANSAS
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Campbell, Gilbert S
Read, Raymond C

Hutchin, Peter
Lafayette
May, Ivan A
Loma Linda
Bailey, Leonard L
Gundry, Steven R
Long Beach
Bloomer, William E
Stemmer, Edward A
Los Angeles
Buckberg, Gerald D
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DeMeester, Tom R
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Holmes, E Carmack
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Khonsari, Siavosh
Laks, Hillel
Lindesmith, George G
Longmire, William, Jr
Maloney, James V, Jr
Mandal, Ashis K
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Peters, Richard M
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Lui, Alfred H F
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Palm Springs
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Baisch, Bruce F
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Miller, George E, Jr
Ramsay, Beatty H
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Benfield, John R
Berkoff, Herbert A
Follette, David M
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Misbach, Gregory A
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Baronofsky, Ivan D
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Lamberti, John J
Moreno-Cabral, Ricardo J
Trummer, Max J
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Ellis, Robert J
Gardner, Richard E
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Hill, J Donald
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Rankin, J Scott
Richards, Victor
Roe, Benson B
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Cukingnan, Ramon A
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Burrington, John D
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Condon, William B
Eiseman, Ben
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Grow, John B, Sr
Harken, Alden H
Hopeman, Alan R
Paton, Bruce C
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Rainer, W Gerald
Wright, George W
Englewood
Kovarik, Joseph L
Lakewood
Swan, Henry
Littleton
Pappas, George
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Vail
Fuller, Josiah
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Rose, Daniel M
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Kemler, R Leonard
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Glenn, William W. L
Hammond, Graeme L
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New Milford
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Norwich
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Randolph, Judson G
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Reis, Robert L
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Spear, Harold C
Naples
Battersby, James S

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Stranahan, Allan
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Lasley, Charles H
Boca Raton
Seley, Gabriel P
Clearwater
Wheat, Myron W, Jr
Coconut Grove
Center, Sol
Coral Gables
Cooke, Francis N
Delray Beach
Shumacker, Harris B, Jr
Gainesville
Alexander, James A
Jacksonville
Edwards, Fred H
Koster, J Kenneth, Jr
Stephenson, Sam, Jr
Jupiter
Gerbasi, Francis S
Lakeland
Brown, Ivan W, Jr
Marathon
Mangiardi, Joseph L
Miami
Bolooki, Hooshang
Kanter, Kirk R
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Lee, Arthur B, Jr
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Miller, Joseph I
Rivkin, Laurence M
Symbas, Panagiotis
Williams, Willis H
Augusta
Ellison, Robert G
Rubin, Joseph W
Chickamauga
Hall, David P
Macon
Dalton, Martin L, Jr
Sealy, Will C
Van De Water, Joseph M
Savannah
Yeh, Thomas J
HAWAII
Honolulu
Ching, Nathaniel P
Gebauer, Paul W
McNamara, J. Judson
IDAHO
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
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Smyth, Nicholas P D
Orlando
Scott, Meredith L
Sherman, Paul H
Ponte Vedra Beach
Gilbert, Joseph, Jr
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DeMatteis, Albert
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Kraeft, Nelson H
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Angell, William W
Robinson, Lary A
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Winter Haven
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Winter Park
Bloodwell, Robert D
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Atlanta
Graver, Joseph M
Guyton, Robert A
Hatcher, Charles, Jr
Hopkins, William A
Jones Ellis L
Raffensperger, John
Replogle, Robert L
Sanders, John H, Jr
Shields, Thomas W
Tatooles, C. J
Thomas, Paul A, Jr
Vanecko, Robert M
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Downers Grove
Leininger, Bernard J
Evanston
Fry, Willard A
Glencoe
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Norman, John C
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Blakeman, Bradford P
DeLeon, Serafin Y
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Pifarre, Roque
Sullivan, Henry J
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Hudson, Theodore R
Ilbawi, Michel N
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Jensik, Robert J
Mason, G. Robert
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Park Ridge
Baffes, Thomas G
Levett, James M
Weinberg, Milton, Jr
Peoria
DeBord, Robert A
Springfield
Wellons, Harry A, Jr

Hartz, Renee S
Head, Louis R
Hunter, James A
Karp, Robert B
Kittle, C Frederick
Mavroudis, Constantine
Michaelis, Lawrence
Najafi, Hassan

O'Neill, Martin J, Jr
Siderys, Harry

IOWA

Cedar Rapids
Lawrence, Montague S

Council Bluffs
Sellers, Robert D

Des Moines
Dorner, Ralph A
Phillips, Steven J
Zeff, Robert H

Iowa City
Behrendt, Douglas M
Ehrenhaft, Johann L
Rossi, Nicholas P
Stanford, William

KANSAS

Cunningham
Allbritten, Frank F, Jr

Lawrence
Miller, Don R

Prairie Village
Holder, Thomas M

Shawnee Mission
Adelman, Arthur

Wichita
Tocker, Alfred M

KENTUCKY

Lexington
Crutcher, Richard R
Dillon, Marcus L, Jr
Todd, Edward P

Louisville
Austin, Erie H, III
Gray, Laman A, Jr
Mahaffey, Daniel E
Ransdell, Herbert, Jr

LOUISIANA

Alexandria
Knoepp, Louis F

Baton Rouge
Berry, B Eugene
Beskin, Charles A

Winnetka
Mackler, S Allen

INDIANA

Indianapolis
Brown, John W
King, Harold
King, Robert D
Mandelbaum, Isidore

Metairie
Ochsner, Alton, Jr

New Orleans
Blalock, John B
DeCamp, Paul T
Hewitt, Robert L
Lindsey, Edward S
McFadden, P Michael
Mills, Noel L
Moulder, Peter V
Ochsner, John L
Pearce, Charles W
Schramel, Robert J
Webb, Watts R

MAINE

Portland
Bredenberg, Carl E
Drake, Emerson H

Rockport
Swenson, Orvar

Windham
Hiebert, Clement

MARYLAND

Baltimore
Attar, Safuh
Baker, R. Robinson
Baumgartner, William A
Blair, Emil
Dodrill, Forest D
Gott, Vincent L
Haller, J Alex, Jr
McLaughlin, Joseph S
Michelson, Elliott
Salomon, Neal W
Turney, Stephen Z
Watkins, Levi, Jr
Wylie, Robert H

Bethesda
Pass, Harvey I

Chevy Chase
Iovine, Vincent M

Ellicott City
MacManus, Joseph E

Fort Detrick
Zajtchuk, Rostik

Towson
Brawley, Robert K

Worton
Walkup, Harry E

MASSACHUSETTS

Boston
Akins, Gary W
Austen, W. Gerald
Barsamian, Ernest M
Berger, Robert L
Bougas, James A
Buckley, Mortimer J

Cambridge
Malcolm, John A

Chestnut Hill
Laforet, Eugene G

Concord
Soutter, Lamar

Dover
Black, Harrison

Falmouth
McElvein, Richard B

Framingham
Bernhard, William F

Burke, John F
Cohn, Lawrence H
Collins, John J
Daggett, Willard M
Daly, Benedict D T
Ellis, F Henry
Frank, Howard A
Gaensler, Edward A
Grillo, 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
Neptune, 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

Schuster, Samuel R

Medford

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Methuen

Wilson, Norman J

North Andover

Cook, William A

Shrewsbury

Moran, John M

Springfield

Engelman, Richard M
Rousou, John A

Vineyard Haven

Malm, James R

Wellesley

Cleveland, Richard J

West Roxbury

Khuri, Shukri F

Westport Harbor

Findlay, Charles W

Williamstown

Wilkins, Earle W

Worcester

Vander Salm, Thomas J

MICHIGAN

Ann Arbor

Bartlett, Robert H
Boiling, Steven F
Bove, Edward L
Deeb, G. Michael
Gago, Otto
Greenfield, Lazar J
Kirsh, Marvin M
Morris, Joe D
Neerken, A John
Orringer, Mark B
Prager, Richard L
Sloan, Herbert E

Bloomfield Township

Timmis, Hilary H

Detroit

Arbulu, Agustin
Silverman, Norman A
Steiger, Zwi
Stephenson, Larry W
Wilson, Robert F

Grand Rapids

Harrison, Robert W
Rasmussen, Richard A
Tomatis, Luis A

Grosse Pointe

Benson, Clifford D

West Bloomfield

Arciniegas, Eduardo

MINNESOTA

Coon Rapids

Gannon, Paul G

Mendota Heights

Dennis, Clarence

Minneapolis

Arom, Kit V
Bolman, R. Morton, III
Emery, Robert W
Foker, John E
Garamella, Joseph J
Olsen, Arthur M
Orszulak, Thomas A
Pairolero, Peter C
Payne, W Spencer
Puga, Francisco J
Schaff, Hartzell V
Trastek, Victor F

St Paul

Lillehei, C Walton
Miller, Fletcher A

Waubun

DeNiord, Richard N

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Carthage

Logan, William D, Jr

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Hardy, James D

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Bridgeton

Codd, John E

Chesterfield

Bergmann, Martin

Columbia

Bryant, Lester R
Curtis, Jack J
Silver, Donald
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Kansas City

Ashcraft, Keith W
Benoit, Hector W, Jr
Borkon, A Michael

Helseth, Hovald K
Kaye, Michael P
Kiser, Joseph C
Molina, J. Ernesto
Nicoloff, Demetre M
Shumway, Sara J

Rochester

Bernatz, Philip E
Danielson, Gordon K
McGoon, Dwight C
McGregor, Christopher G A
Killen, Duncan A
Mayer, John H, Jr
Padula, Richard T
Piehler, Jeffrey M
Reed, William A
VanWay, Charles W, III

Mount Vernon

Campbell, Daniel C, Jr

St Louis

Barner, Hendrick B

Baue, Arthur E
Connors, John P
Cooper, Joel D
Cox, James L
Ferguson, T Bruce, Jr
Ferguson, Thomas B
Flye, M. Wayne
Johnson, Frank E
Kaiser, George C
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Lewis, J Eugene, Jr
McBride, Lawrence R
Naunheim, Keith S
Pasque, Michael K
Patterson, G Alec
Pennington, D Glenn
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Willman, Vallee L

MONTANA

Missoula

Oury, James H

Twin Bridges

Lower, Richard R

NEBRASKA

Omaha

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Schultz, Richard D

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Swain, Julie A

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Jaffrey

Woods, Francis M

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Holswade, George R

Bellville

Gerard, Franklyn P

Browns Mills

Fernandez, Javier
McGrath, Lynn B

Camden

Camishion, Rudolph C
DelRossi, Anthony J

East Orange

Auerbach, Oscar

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Hutchinson, John E, III

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Millburn

Personnel, Victor

Moorestown

Morse, Dryden P

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Lewis, Ralph J
MacKenzie, James W
Scholz, Peter M

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Gielchinsky, Isaac
Swan, Kenneth G

Pittstown

Garzon, Antonio A

Tenally

Gerst, Paul H

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Edwards, W Sterling

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Santa Fe

Davila, Julio C

Silver City

Waddell, William R

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Bay Shore

Ryan, Bernard J

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Attai, Lari A
Brodman, Richard F
Fell, Stanley C
Ford, Joseph M
Frater, Robert W M
Gay, William A, Jr
Hirose, Teruo
Boyd, Arthur D
Bregman, David
Burt, Michael E
Cahan, William G
Clauss, Roy H
Conklin, Edward F
Culliford, Alfred T
Ergin, M Arisan
Friedlander, Ralph
Galloway, Aubrey C, Jr

Veith, Frank J
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Sawyer, Philip N
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Andersen, Murray N
Bhayana, Joginder N
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Blumenstock, David A
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New Rochelle
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Adams, Peter X
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Ginsberg, Robert J
Gold, Jeffrey P
Green, George E
Griepp, Randall B
Isom, O Wayne
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King, Thomas C
Kirschner, Paul A
Krieger, Karl H
Litwak, Robert S
Martini, Nael
McCord, Colin W
McCormack, Patricia M
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Redo, S Frank
Reemtsma, Keith
Rose, Eric A
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Spencer, Frank C
Spotnitz, Henry M
Subramanian, Valavanur
A Tice, David A
Tyras, Denis H
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Decker, Alfred M, Jr
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NORTH CAROLINA
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Hudspeth, Allen S
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Starek, Peter J
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Robicsek, Francis
Selle, Jay G
Taylor, Frederick H

Durham

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Lowe, James E
Oldham, H Newland, Jr
Sabiston, David C
Van Heeckeren, Daniel W

Columbus

Davis, J Terrance
Kakos, Gerard S
Meckstroth, Charles
Myerowitz, P. David
Williams, Thomas E, Jr

Dayton

DeWall, Richard A

Delaware

Clatworthy, H Williams, Jr

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Kilman, James W

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Jenks

LeBeck, Martin B

Lawton

Barnhorst, Donald A

Oklahoma City

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Felton, Warren L, II
Fisher, R Danyl
Greer, Allen E
Munnell, Edward R
Williams, G Rainey
Zuhdi, M Nazih

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Miller, Arthur C

Portland

Cobanoglu, Adnan
Krause, Albert H
Lemmer, John H, Jr
Okies, J Edward
Poppe, J Karl
Starr, Albert

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Frobese, Alfred S

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Snyder, John M

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Dunn, Jeffrey M
Hiratzka, Loren F
Ivey, Tom D
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Yee, Edward S

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Ankeney, Jay L
Cosgrove, Delos M
Geha, Alexander S
Groves, Laurence K
Kay, Earle B
Loop, Floyd D
Lytle, Bruce W
Rice, Thomas W
Snow, Norman J

Bryn Mawr

Haupt, George J

Camp Hill

Pennock, John L

Carlisle

DeMuth, William, Jr

Darby

McKeown, John J, Jr

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, V. Paul
Bowles, L Thompson
Brockman, Stanley K
Diehl, James T
DiSesa, Verdi J
Edie, Richard N
Edmunds, L. Henry, Jr
Fineberg, Charles
Gardner, Timothy J
Goldberg, Melvyn
Kaiser, Larry R
Levine, Frederick H
MacVaugh, Horace
Nemir, Paul, Jr
Shochat, Stephen J
Spray, Thomas L
Whitman, Glenn J R

Pittsburgh

Armitage, John M
Bahnson, Henry T
Clark, Richard E
Griffith, Bartley P
Hardesty, Robert L
Kormos, Robert L
Landreneau, Rodney J
Magovern, George J
Pontius, Robert G
Rams, James J
Siewers, Ralph D
Brott, Walter H
Domm, Sheldon E

Memphis

Cole, Francis H

Rosemont
Sink, James D
Templeton, John, III
Villanova
Mundth, Eldred D
Wayne
Hargrove, W Clark, III
Lemmon, William M
Wyncote
Mendelssohn, Edwin
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
Kratz, John M
Parker, Edward F
Sade, Robert M
Columbia
Almond, Carl H
Hilton Head Island
Humphrey, Edward W
Isle of Palms
Mullen, Donald C
Landrum
Stayman, Joseph W
Spartanburg
Utley, Joe R
TENNESSEE
Knoxville
Blake, Hu Al
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
TEXAS
Amarillo
Sutherland, R Duncan
Austin
Hood, R Maurice
Tyson, Kenneth R T
Burnet
Ross, Raleigh R
Coppell
McPhail, Jasper L
Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Lambert, Cary J
Mills, Lawrence J
Paulson, Donald L
Platt, Melvin R
Razzuk, Maruf A
Ring, W Steves
Seybold, William D
Urschel, Harold, Jr
Dilley
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El Paso
Glass, Bertram A
Galveston
Conti, Vincent R
Derrick, John R
Zwischenberger, Joseph B
Houston
Baldwin, John C
Beall, Arthur C, Jr
Burdette, Walter J
Cooley, Denton A
Coselli, Joseph S
DeBakey, Michael E
Frazier, O. Howard
Hallman, Grady L
Henly, Walter S
Jones, James W
Lawrie, Gerald M
Mattox, Kenneth L
Mountain, Clifton F
Ott, David A
Overstreet, John W
Salt Lake City
Doty, Donald B
Liddle, Harold V
Mortensen, J D
Nelson, Russell M
VERMONT
Burlington
Miller, Donald B
Richford
Grondin, Claude M
West Dover
Humphreys, George H, II
VIRGINIA
Altavista
Pierucci, Louis, Jr
Annandale
Akl, Bechara F
Burton, Nelson A
Lefrak, Edward A
Arlington
Klepser, Roy G
Aylett
Gwathmey, Owen
Charlottesville

Reul, George J, Jr
Roth, Jack A
Walker, William E
Wukasch, Don C

Kemp
Davis, Milton V

Lubbock
Bricker, Donald L
Feola, Mario
Wallsh, Eugene

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

Shepherd
Morris, George C, Jr

Temple
Brindley, G. Valter, Jr

Woodville
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UTAH

Bellingham
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Friday Harbor
Lawrence, G Hugh

Issaquah
Jarvis, Fred J

Kirkland
Mills, Waldo O

Poulsbo
Malette, William G

Seattle
Allen, Margaret D
Anderson, Richard P
Dillard, David H
Hill, Lucius D
Jones, Thomas W
Li, Wei-I
Manhas, Dev R
Mansfield, Peter B
Miller, Donald W, Jr
Rittenhouse, Edward
Sauvage, Lester
Thomas, George I
Verrier, Edward D

Spokane
Berg, Ralph, Jr

WEST VIRGINIA

Charlestown
Walker, James H

Huntington
Gonzalez-Lavin, Lorenzo

Dammann, John F
Daniel, Thomas M
Kron, Irving L
Minor, George R
Muller, William, Jr
Nolan, Stanton P
Spotnitz, William D

Lynchburg
Moore, Richmond L

McLean
Conrad, Peter W
Mills, Mitchell

Reston
Boyd, Thomas F

Richmond
Bosher, Lewis H, Jr
Brooks, James W
Cole, Dean B
Damiano, Ralph J, Jr
Wechsler, Andrew S

WASHINGTON

Morgantown
Graeber, Geoffrey M
Gustafson, Robert A
Hill, Ronald C
Murray, Gordon F
Warden, Herbert E

Parkersburg
Tarnay, Thomas J

WISCONSIN

Eau Claire
McEnany, M Terry

Madison
Chopra, Paramjeet S
Mentzer, Robert M, Jr
Young, William P

Marshfield
Myers, William O
Ray, Jefferson F, III
Sautter, Richard D

Mequon
Narodick, Benjamin

Milwaukee
Johnson, W Dudley
Litwin, S Bert
Olinger, Gordon N
Tector, Alfred J

West Bend
Gardner, Robert J

WYOMING

Teton Village
Kaunitz, Victor H

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Edmonton
Callaghan, John C
Gelfand, Elliot T
Sterns, Laurence P

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Walker, George R

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Christakis, George T
Coles, John G
David, Tirone E
Delarue, Norman C

Allen, Peter
Ashmore, Phillip G
Jamieson, W R Eric
Tyers, G Frank O
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Stenstrom, John D
West Vancouver
Robertson, Ross
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Winnipeg
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Cohen, Morley
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Murphy, David A
Mabou
Thomas, Gordon W
ONTARIO
Collingwood
Heimbecker, Raymond
London
McKenzie, F Neil
Novick, Richard J
North York
Goldman, Bernard S
Nottawa
Key, James A
Ottawa
Keon, Wilbert J
Fremes, Stephen E
McKneally, Martin F
Mickleborough, Lynda L
Pearson, F Griffith
Salerno, Tomas A
Scully, Hugh E
Todd, Thomas R J
Trimble, Alan S
Trusler, George A
Weisel, Richard D
Williams, William G
Westbrook
Lynn, R Beverley
QUEBEC
Montreal
Blundell, Peter E
Chartrand, Claude C. C
Chiu, Ray Chu-Jeng
Cossette, Robert
Dobell, Anthony R C
Duranceau, Andre C H
MacLean, Lloyd D
Morin, Jean E
Mulder, David S
Pelletier, Conrad L
Scott, Henry J
Shennib, Hani
Outremont
Lepage, Gilles
Sainte-Foy
DesLauriers, Jean
Sillery
Grondin, Pierre

OTHER COUNTRIES

AFGHANISTAN
Kabul
Hankins, John R
ARGENTINA
Buenos Aires
Favaloro, Rene G
AUSTRALIA
QUEENSLAND
Brisbane
O'Brien, Mark F, FRCS
SOUTH AUSTRALIA
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Sutherland, H D'Arcy, FRCS
VICTORIA
Melbourne
Nossal, Gustav J V
AUSTRIA
Leonding
Bruecke, Peter E
Salzburg
Unger, Felix H
Vienna
Wolner, Ernst
BELGIUM
Bertem
Sergeant, Paul T
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Lerut, Antoon E M R
BRAZIL
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Sao Paulo
Jatene, Adib D
Herefordshire
Smith, Roger A
London
Braimbridge, Mark V
de Leval, Marc R
Lennox, Stuart C
Lincoln, Christopher R
Ross, Donald N
Stark, Jaroslav F
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Thompson, Vemon C
Yacoub, Magdi
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Helsinki
Mattila, Severi P
FRANCE
Bordeaux
Couraud, Louis
Fontan, Francis M
Marseille
Metras, Dominique R
Montpellier
Thevenet, Andre A
Paris
Binet, Jean-Paul
Blondeau, Philip
Cabrol, Christian E A
Carpentier, Alain F
Menasche, Philippe
Piwnica, Armand H
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Weldon, Clarence S
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Kennedy, John H

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Messmer, Bruno J
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Munchen
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GUATEMALA
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VanAllen, Chester M
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O'Malley, Eoin
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Parenzan, Lucio
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Cotrufo, Maurizio
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Bortolotti, Uberto
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Marcelletti, Carlo
JAPAN
Kanazawa
Iwa, Takashi
Kitakyushushi
Miyamoto, Alfonso T
Osaka
Kawashima, Yasunaru
Sendai
Mohri, Hitoshi
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Imai, Yasuharu
Wada, Juro J
KOREA
Seoul
Cho, Bum-Koo
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Dor, Vincent
UNITED ARAB EMERIT
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Brom, A Gerard

NEW ZEALAND
Waiwera HBC
Barratt-Boyes, Brian G
P.R. OF CHINA
Beijing
Ying-Kai, Wu
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Lisbon
Macedo, Manuel E M
ROMANIA
Targu-Mures
Deac, Radu C
RUSSIA
Moscow
Bockeria, Leo A
SAUDI ARABIA
Riyadh
Duran, Carlos Gomez
Merendino, K Alvin
SCOTLAND
Edinburgh
Logan, Andrew
Glasgow
Wheatley, David J
SPAIN
Madrid
Rivera, Ramiro
SWEDEN
Sollentuna
Bjork, Viking
SWITZERLAND
Arzier
Hahn, Charles J
Genolier
Castaneda, Aldo R
Norwood, William I
Pully
Naef, Andreas P
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ARTICLE I. NAME

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

ARTICLE II. PURPOSE

The purposes of the Association shall be:

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

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

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

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

ARTICLE III. MEMBERSHIP

Section 1. There shall be three classes of members: Honorary, Senior, and Active. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on

the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws. Honorary members shall have the privilege of voting but shall not be eligible to hold office.

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

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

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

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

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

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

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

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

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

ARTICLE IV. Board of Directors ("Council")

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

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

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

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

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

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

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

Section 3. The President, Vice-President, Secretary and Treasurer shall be elected at the annual meeting of the Association and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for a one-year term of office and neither may be re-elected to succeed himself in the same office, unless such officer is filling the unexpired term of an officer previously elected to such office. The Secretary and the

Treasurer shall be elected for a one-year term of office and may be re-elected for not more than four additional terms.

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

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

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

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve as Treasurer of the Association 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 year. The Chairman shall be the member-at-large serving his fourth year. The duties of the Committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and Fellows, past, present, and future.

Section 8. The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, *ex officio,* the chairman of this board and shall be privileged to appoint and indefinitely reappoint such members of the Association, regardless of class of membership, and such non-members of the Association as in his opinion may be best calculated to meet the editorial requirements of the Association.

Section 9. The Ethics Committee shall consist of five members appointed by the Council. No member shall serve more than four years. The Ethics Committee shall advise the Council concerning alleged breaches of ethics. Complaints regarding alleged breaches of ethics shall be

received in writing by the Ethics Committee and shall be investigated by it. In addition, the Ethics Committee may investigate on its own initiative.

Section 10. The Thoracic Surgical Workforce Committee shall be a Joint Committee of this Association and The Society of Thoracic Surgeons. The Committee shall consist of two members of this Association, two members of The Society of Thoracic Surgeons, and a Chairman who shall be a member of this Association and The Society of Thoracic Surgeons. The duties of this Committee, and the manner of appointment and term of its members and chairman, shall be determined jointly by the Council of this Association and the Council of The Society of Thoracic Surgeons.

Section 11. The Committee on Graduate Education in Thoracic Surgery shall consist of five members appointed by the Council. One member shall be appointed each year for a five-year term. The committee shall review graduate medical education in thoracic surgery and makes its recommendations to the Council to assist in meeting the educational mission of the Association.

ARTICLE VII. Finances

Section 1. The fiscal year of the Association shall begin on the first day of January and end on the last day of December each year.

Section 2. Members shall contribute to the financial maintenance of the Association through initiation fees, annual dues, and special assessments. The amount of the annual dues and the initiation fees shall be determined by these By-Laws. If, at the end of any fiscal year, there is a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Active members to contribute the necessary amount so that no deficit is carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment for any purpose consistent with the purposes of the Association, and such special assessment shall become an obligatory charge against the classes of members affected thereby.

Section 3. To meet the current expenses of the Association, there shall be available all revenue derived by the Association subject to the provisions of Section 4, following.

Section 4. Funds derived from the payment of initiation fees shall not be available to current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact, 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 the By-Laws.

Section 2. Notice of any meeting of the Association shall be given to each member of the Association not less than five nor more than forty days prior to any annual meeting and not less than thirty nor more than forty days prior to any special meeting by written or printed notice delivered personally or by mail, by or at the direction of the Council, the President or the Secretary. Such notice shall state the place, day and hour of the meeting and in the case of a special meeting shall also state the purpose or purposes for which the meeting is called.

Section 3. A special meeting of the Association may be called by the Council or on the written request of fifteen members delivered to the Council, the President or the Secretary. The specific purposes of the meeting must be stated in the request.

Section 4. Attendance at annual meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members, but it shall be expected from all Active Members.

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

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

1. Appointment of necessary committees.

2. Miscelleneous business of an urgent nature.

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

1. Reading or waiver of reading of the minutes of the preceeding meetings of the Association and the Council.

2. Report of the Treasurer of the last fiscal year.

3. Audit Report.

4. Report of the Necrology Committee.

5. Report of the Program Committee.

6. Action on amendments to the Articles of Incorporation and By-Laws, if any.

7. Action on recommendations emanating from the Council.

8. Unfinished Business.

9. New Business.

10. Report of the Membership Committee.

11. Election of new members.

12. Report of the Nominating Committee.

13. Election of officers.

Section 8. Except where otherwise required by law of these By-Laws, all questions at a meeting of the members shall be decided by a majority vote of the members present in person and voting. Voting by proxy is not permitted.

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

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

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

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

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

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

ARTICLE IX. Indemnification and Directors and Officers

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

Section 2. Upon specific authorization by the Council, the Association may purchase and maintain insurance on behalf of any and all of its directors or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against any liability or settlement based on asserted liability, incurred by them by reason of being or having been directors or officers as a director or officer of the Association or of such other corporation or association, whether or not the Association would have the power to indemnify them against such liability or settlement under the provisions of Section 1.

ARTICLE X. Papers

Section 1. All papers read before the Association shall become the property of the Association. Authors shall leave original copies of their manuscripts with the Editor or reporter, at the time of presentation, for consideration for publication in the official Journal.

Section 2. When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions.

ARTICLE XI. Initiation Fees, Dues and Assessments

Section 1. Honorary Members of the Association are exempt from all initiation fees, dues, and assessments.

Section 2. Annual dues for Active Members shall be $150.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Senior Members are exempt from dues.

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

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

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

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

ARTICLE XII. Parliamentary Procedure

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

ARTICLE XIII. Amendments

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

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

As amended, Tuesday, April 27, 1993

ANNUAL MEETING DATES

Back to Annual Meeting Program

Meetings of the American Association for Thoracic Surgery

1918-Chicago..... President, Samuel J. Meltzer
1919-Atlantic City..... President, Willy Meyer
1920-New Orleans..... President, Willy Meyer
1921-Boston..... President, Rudolph Matas
1922-Washington..... President, Samuel Robinson
1923-Chicago..... President, Howard Lilienthal

1924-Rochester, Minn..... President, Carl A. Hedblom
1925-Washington..... President, Nathan W. Green
1926-Montreal..... President, Edward W. Archibald
1927-New York..... President, Franz Torek
1928-Washington..... President, Evarts A. Graham
1929-St. Louis..... President, John L. Yates
1930-Philadelphia..... President, Wyman Whittemore
1931-San Francisco..... President, Ethan Flagg Butler
1932-Ann Arbor..... President, Frederick T. Lord
1933-Washington..... President, George P. Muller
1934-Boston..... President, George J. Heuer
1935-New York..... President, John Alexander
1936-Rochester, Minn..... President, Carl Eggers
1937-Saranac Lake..... President, Leo Eloesser
1938-Atlanta..... President, Stuart W. Harrington
1939-Los Angeles..... President, Harold Brunn
1940-Cleveland..... President, Adrian V. S. Lambert
1941-Toronto..... President, Fraser B. Gurd
1944-Chicago..... President, Frank S. Dolley
1946-Detroit..... President, Claude S. Beck
1947-St. Louis..... President, I. A. Bigger
1948-Quebec..... President, Alton Ochsner
1949-New Orleans..... President, Edward D. Churchill
1950-Denver..... President, Edward J. O'Brien
1951-Atlantic City..... President, Alfred Blalock
1952-Dallas..... President, Frank B. Berry
1953-San Francisco..... President, Robert M. Janes
1954-Montreal..... President, Emile Holman
1955-Atlantic City..... President, Edward S. Welles
1956-Miami Beach..... President, Richard H. Meade
1957-Chicago..... President, Cameron Haight
1958-Boston..... President, Brian Blades
1959-Los Angeles..... President, Michael E. De Bakey
1960-Miami Beach..... President, William E. Adams
1961-Philadelphia..... President, John H. Gibbon, Jr.
1962-St. Louis..... President, Richard H. Sweet (Deceased 1-11-62)
..... President, O. Theron Clagett
1963-Houston..... President, Julian Johnson
1964-Montreal..... President, Robert E. Gross
1965-New Orleans..... President, John C. Jones
1966-Vancouver, B. C..... President, Herbert C. Maier
1967-New York..... President, Frederick G. Kergin
1968-Pittsburgh..... President, Paul C. Samson
1969-San Francisco..... President, Edward M. Kent
1970-Washington, D. C..... President, Hiram T. Langston
1971-Atlanta..... President, Thomas H. Burford
1974-Las Vegas..... President, Lyman A. Brewer, III
1975-New York..... President, Wilfred G. Bigelow
1976-Los Angeles..... President, David J. Dugan
1977-Toronto..... President, Henry T. Bahnson
1978-New Orleans..... President, J. Gordon Scannell
1979-Boston..... President, John W. Kirklin
1980-San Francisco..... President, Herbert Sloan
1981-Washington, D.C..... President, Donald L. Paulson
1982-Phoenix, Arizona..... President, Thomas B. Ferguson
1983-Atlanta..... President, Frank C. Spencer

1984-New York..... President, Dwight C. McGoon
1985-New Orleans..... President, David C. Sabiston
1986-New York..... President, James, R. Malm
1987-Chicago..... President, Norman E. Shumway
1988-Los Angeles..... President, Paul A. Ebert
1989-Boston..... President, W. Gerald Austen
1990-Toronto..... President, F. Griffith Pearson
1991-Washington, D.C..... President, Keith Reemtsma
1992-Los Angeles..... President, John A. Waldhausen
1993-Chicago..... President, John L. Ochsner
1994-New York..... President, Aldo R. Castaneda

AWARDS

Back to Annual Meeting Program

GRAHAM EDUCATION AND RESEARCH FOUNDATION

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

President James L. Cox, M.D., St. Louis, Missouri
Vice President Andrew S. Wechsler, M.D., Richmond, Virginia
Secretary-Treasurer William T. Maloney, Manchester, Massachusetts
Director Henry M. Spotnitz, M.D., New York, New York

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWSHIP

The Evarts A. Graham Memorial Traveling Fellowship was established in 1951 by The American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young surgeons from abroad who have completed their formal training in general, thoracic, and cardiovascular surgery. The award allows the recipient to study a year to intensify his training in a program of special interest and to travel to several sites to broaden his overall training and increase his contacts with thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 43 young surgeons from 23 countries have completed their training at thoracic surgical centers.

1st	1951-52	L.L. Whytehead, M.D., F.R.C.S 790 Sherbrooke St., Winnepeg, M CANADA
2nd	1953-54	W.B. Ferguson, M.B., F.R.C.S. Royal Victoria Infirmary, Newca ENGLAND
3rd	1954-55	Lance L. Bromley, M.Chir., F.R. St. Mary's Hospital, London, W.2
4th	1955-56	Raymond L. Hurt, F.R.C.S. The White House, 8 Room Lane, ENGLAND

5th	1956-57	Mathias Paneth, F.R.C.S. Brompton Hospital, London, S.W
6th	1957-58	Peter L. Brunnen, F.R.C.S. Department of Thoracic Surgery, Hospital, Aberdeen, SCOTLANI
7th	1958-59	N.G. Meyne, M.D. University of Amsterdam, Wilhe Amsterdam, HOLLAND
8th	1960-61	Godrej S. Karai, M.D. Calcutta, INDIA
9th	1961-62	Fritz Helmer, M.D. Second Surgical Clinic, Universi AUSTRIA
10th	1962-63	Theodor M. Scheinin, M.D. Tammisalonite 20, Helsinki, 008
11th	1963-64	Masahiro Saigusa, M.D. National Nakano Chest Hospital, Ku, Tokyo 165, JAP AN
12th	1963-64	Adar J. Hallen, M.D. Department of Thoracic Surgery, Uppsala, SWEDEN
13th	1964-65	Stuart C. Lennox, M.D. 18 Alexander Sq., 5W3 2AX, Lo
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S University Hospital Surgical Clir Thessoliniki, Thessaloniki, GRE
15th	1965-66	Gerhard Friehs, M.D. Chirugische University Klinik, G
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 Par AUSTRALIA
18th	1966-67	G.B. Parulkar, M.D. Wookhardt Heart Institute, Poona Road, Bombay 400 018, INDIA
19th	1967-68	Claus Jessen, M.D. Surg. Dept. D, Rigshospitalet, Bl Copenhagen, DENMARK
20th	1969-70	Peter Brueke, M.D. AM Steinbruch, 29 Linz-Puchena
21st	1970-71	Michel S. Slim, M.D. New York Medical College, Div New York, New York 10595 US
22nd	1971-72	Severi Pellervo, Mattila, M.D. Forsellesintie 5.7.D, Kaunianen,
23rd	1972-73	Yasuyuki Fujiwara, M.D. Department of Cardiovascular Su College Hospital, Shinjuko, Tokyo, JAPA
24th	1973-74	Marc Roger de Leval, M.D. Hospital for Sick Children, Great WC1N 3JH, ENGLAND
25th	1974-75	J. J. DeWet Lubbe, M.D. 1406 City Park Medical Center, Town 8001, REPUBLIC OF SOU
26th	1975-76	Mieczyslaw Trenkner, M.D. Institute of Surgery, 80-211 Ul, I POLAND

27th	1976-77	Bum Koo Cho, M.D. Yonsei University, P.O. Box 71 S Seoul, KOREA
28th	1977-78	Alan William Gale, M.D., FRAC 171 Sutherland, Paddington 2021
29th	1978-79	Eduardo Otero Goto, M.D. Servicio de Cirugia Cariovascula Fe", Valencia, SPAIN
30th	1980-81	Richard K. Firmin, M.D. "Moss Grove," 5 Knighton Oran Leicester LE2 2LF, ENGLAND
31st	1981-82	Claudio A. Salles, M.D. Av Celco Porfirio Machado, 370 Belo Horizonte MG, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki, M.D. First Dept. of Surgery, Osaka Un Fukushima-ku, Osaka, 533, JAP/
33rd	1983-84	Georg S. Kobinia, M.D. LKH Klagenfurt St., Veider Stra Surgery, Klagenfurt, A-9026, AU
34th	1984-85	Aram Smolinsky, M.D. Department of Cardiac Surgery, Center Tel Hashomer, 52621, ISREAL
35th	1985-86	Florentino J. Vargas, M.D. San Martin 1353, Buenos Aries,
36th	1986-87	Ari L. J. Harjula, M.D. Helsinki University Hosp. Surge Helsinki, 00290, FINLAND
37th	1987-88	Byung-Chul Chang, M.D. Dept. of Thoracic and Cardiovas University College of Medicine, KOREA
38th	1988-89	Wang Cheng, M.D. Department of Cardiac Surgery, Blood Vessel Medical Center an Andingmenwai, Beijing, PEOPL CHINA
39th	1989-90	Christopher John Knott-Craig, M University of Oklahoma, Thorac Surgery, P.O. Box 26901, Oklah USA
40th	1991-92	Ko Bando, M.D., Ph.D. University of Pittsburgh, 1084 S 15261, USA
41st	1992-93	Timothy E. Oaks, M.D. Department of Surgery, The Milt Center, Room #6314, Box 850, H
42nd	1993-94	Alain E. Serraf, M.D. Hospital Marie-Lannelongue, Un avenue de la Resistance, 92350 L FRANCE
43rd	1994-95	Cornelius McKown Dyke, M.D. Medical College of Virginia, Bo Richmond, VA 23298-0645, US/

TSFRE AWARDS

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THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION

ORGANIZATION

The Thoracic Surgery Foundation for Research and Education was established in December of 1989 to identify, encourage and provide for the education and research needs in thoracic surgery. The Foundation is entirely supported through the generosity of philanthropic supporters. All gifts are tax deductible as provided by law.

The American Association for Thoracic Surgery, The Society of Thoracic Surgeons, The Southern Thoracic Surgical Association and The Western Thoracic Surgical Association fully endorse and encourage the work of the Foundation. The sixteen-member Board of Directors was established from nominations made by these societies.

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GRANTS AND AWARDS

Evaluation of grant and award applications to the Foundation is based on meticulous peer review procedures. Both the merit of the proposal and the leadership promise of the applicant are rated by recognized authorities committed to impartiality.

Funding programs of the foundation are:

I. Individual Research Investigator Grants
II. Research Fellowship Awards
III. Career Development Awards
IV. Alley-Sheridan Scholarships

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TSFRE AWARDS

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THORACIC SURGERY FOUNDATION 1994 AWARDS

Nina S. Braunwald Research Fellowship

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

The Role of Nitric Oxide in Mediating Neurologic Injury in a Canine Model of Hypothermic Circulatory Arrest

Nina S. Braunwald Career Development Award

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

To Expand the Scope of Research on Adhesion Molecules

The Thoracic Surgery Foundation Research Fellowship Award

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

Pathogenesis and Repair of Ischemic Mitral Regurgitation

Hiranya Rajasinghe, M.D., University of California, San Francisco

Fetal Induction of Immune Tolerance for Neonatal Cardiac Xenotransplantation

The Thoracic Surgery Foundation Research Investigator Grant

Richard P. Embrey, M.D., The University of Iowa College of Medicine

Mechanisms of Coronary Microvascular Abnormalities Due to Cardiopulmonary Bypass and Cardioplegia

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

Cardiac Allograft Vasculopathy in Miniature Swine

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

Basic Fibroblast Growth Factor Protects Ischemic Skeletal Muscle During Long-Term Electrical Stimulation

RESEARCH SCHOLARSHIP

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY RESEARCH SCHOLARSHIP

The American Association for Thoracic Surgery Research Scholarship was established by the Association in 1985. Funded by the Association and individual contributions, the Research Scholarship provides opportunity for research, training and experience for North American surgeons committed to pursuing an academic career in cardiothoracic surgery. Administered by the Graham Education and Research Foundation, the program is undertaken within the first three years after completion of an approved cardiothoracic residency and is about two years in duration.

EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

1986-1988 Mark K. Ferguson, M.D.

University of Chicago,
Department of Surgery

ALFRED BLALOCK RESEARCH SCHOLARSHIP

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

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

Massachusetts General Hospital and Harvard Medical School

JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

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

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

Duke University Medical Center,

ALTON OCHSNER RESEARCH SCHOLARSHIP

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

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

Brigham and Women's Hospital

ROBERT E. GROSS RESEARCH SCHOLARSHIP

"Role of Intra-Cellular Messenger cAMP and cGMP in Organ Preservation"

1994-1996 Mehmet C. Oz., M.D.

"Development of a Model of Chronic Pulmonary Rejection in Miniature Swinie"
1994-1996 Toralf Mauritz Sundt, III, M.D.

Washington University School of Medicine