1992 ANNUAL MEETING PROGRAM

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FOR THORACIC SURGERY
1992 Annual Meeting

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POSTGRADUATE COURSE

AATS
Postgraduate
COURSE
Congenital Heart *Disease*

Sunday, April 26, 1992
8:00 am - 3:00 p.m.
Century Plaza Hotel
Los Angeles, California

Objectives

The 1992 postgraduate course in congenital heart disease will cover three topics: pulmonary atresia with intact ventricular septum, deep hypothermia and circulatory arrest, and the Fontan procedure. This course will provide in depth coverage of these three challenging and evolving areas of congenital heart disease. There will be specific instruction in the morphology of various lesions, indications for the different surgical procedures, technical details (including cardiopulmonary bypass and myocardial protection) and long-term results. Lectures and discussions will focus on controversial areas within each topic, highlighting alternate approaches as applicable.

Registration

Enrollment in this course will be by pre-registration until March 27, 1992. Registration forms will be processed in the order in which they are received and must be accompanied by payment in full. After March 27, 1992, participants may register onsite. The registration fee is $25.00 and includes PC 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 education for physicians. The American Association for Thoracic Surgery designates this continuing medical education activity for 5 credit hours in category 1 of the Physicians Recognition Award of the American Medical Association.

SUNDAY, APRIL 26, 1992

I. Pulmonary Atresia/Intact Ventricular Septum

Moderators

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

Robert M. Sade, M.D., Charleston, SC

8:00 a.m. Morphology of pulmonary stenosis and atresia with intact ventricular septum
John W. Kirklin, M.D., Birmingham, AL

8:15 a.m. Early and midterm survival after treatment of the newborn with PS/PA with IVS
Robert M. Sade, M.D., Charleston, SC

8:30 a.m. Recommendations regarding transannular patching and shunting for PS/PA with IVS
Frank L. Hanley, M.D., Boston, MA

8:45 a.m. Right ventricular exclusion in the treatment of PS/PA with FVS
William G. Williams, M.D., Toronto, ON

9:00 a.m. Update on other CHSS data: Interrupted aortic arch and transposition
John W. Kirklin, M.D., Birmingham, AL

9:15 a.m. Discussion

9:45 a.m. Break

II. Deep Hypothermia/Circulatory Arrest

Moderators

David R. Clarke, M.D., Denver, CO

William G. Williams, M.D., Toronto, ON

10:30 a.m. Current research in cerebral metabolism and blood flow

Ross M. Ungerleider, M.D., Durham, NC

10:45 a.m. Neurologic sequelae following circulatory arrest and continuous low flow cardiopulmonary bypass: Early results of clinical trial
Gil Wernovsky, M.D., Boston, MA

11:00 a.m. Technical aspects of perfusion and cardioplegia in the newborn
John E. Mayer, JR., M.D., Boston, MA

11:15 a.m. Discussion

12:00 p.m. Lunch

III. Fontan Procedure/Staging

Moderators
John W. Brown, M.D., Indianapolis, IN

John L. Myers, M.D., Hershey, PA

1:30 p.m. Preliminary staging with the bidirectional Glenn procedure

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

1:45 p.m. Use of an adjustable atrial septal defect

Hillel Laks, M.D., Los Angeles, CA

2:00 p.m. Fenestrated patches with device closure

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

2:15 p.m. Management of complex venous drainage

Francisco J. Puga, M.D., Rochester, MN

2:30 p.m. Discussion

MONDAY MORNING, APRIL 27, 1992

American Association for Thoracic Surgery
72ND ANNUAL MEETING

Century Plaza Hotel, Los Angeles, CA
APRIL 26-29, 1992

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

8:45 a.m. SCIENTIFIC SESSION - Los Angeles Ballroom

1. Video Assisted Thoracic Surgical Resection of Malignant Lung Tumors

*RALPH J. LEWIS, M.D., ROBERT J. CACCAVALE, M.D.**

*and GLENN E. SISLER, M.D.**

New Brunswick, New Jersey

In a series of over 125 patients, who underwent Video Assisted Thoracic Surgery for a multitude of problems, 40 patients underwent resection of malignant parenchymal tumors. Lesions consisted of T1No primary tumors, metastatic tumors or parenchymal lymphoma. All resections of primary tumors were considered curative, and pathological specimens revealed squamous cell carcinoma, adenocarcinoma and broncho-alveolar carcinoma. Mediastinal, hilar and fissure nodes were biopsied, and frozen sections were obtained before resection. Utilizing various staplers, liga clips, and conventional thoracic instruments, these lesions are removed by lobectomy, partial lung (segmental) or wedge resection. A double lumen tube allows deflation of the operative lung. Three to four incisions, about 2 to 5 centimeters in length, are made in the intercostal spaces taking care not to bruise, spread or fracture any ribs. A thoracoscope with an attached microcamera projects the intra-thoracic contents on a screen allowing complex surgery to be performed. All patients had markedly reduced post operative pain and made an uneventful recovery. Most patients were discharged between 3-6 days and did not require post operative intensive care services. Video Assisted Thoracic Surgery seems to be another option for removing certain types of malignant parenchymal tumors.

*By Invitation

2. Subglottic Tracheal Resection and Synchronous Laryngeal Reconstruction

MICHAEL A. MADDAUS, M.D., JULIUS L.R. TOTH, M.D.*,*

PATRICK J. GULLANE, M.D. and*

F. GRIFFITH PEARSON, M.D.

Toronto, Ontario

Post intubation injury of the upper airway commonly results in stenotic lesions of larynx, subglottis and adjacent trachea. The traditional approach to surgical correction is

laryngofissure for the laryngeal component, and staged plastic reconstruction of the subglottic stenosis. Reported results are variable and unpredictable, and a significant number of patients do not achieve permanent extubation. We report experience with 17 patients with combined laryngeal, subglottic and tracheal stenosis, who were successfully managed by a one stage operation: circumferential resection of subglottis and trachea with primary thyrotracheal anastomosis, combined with laryngofissure and laryngeal reconstruction. These procedures required the collaboration of Otolaryngology and Thoracic Surgery.

Between 1972 and 1991, our Thoracic Surgical Division did 60 circumferential subglottic tracheal resections with primary thyrotracheal anastomosis. There was no operative mortality and all 60 patients were successfully extubated. In 17 of these patients, a concomitant laryngofissure for laryngeal reconstruction was required: excision of interarytenoid scar -10, in-terarytenoid mucosal graft - 4, mobilization of cricoarytenoid joint - 3. A temporary laryngotracheal stent (usually a Montgomery T-tube) was maintained post-operatively in all cases (duration 3 months to greater than 1 year). All patients are now permanently extubated and none have developed functionally significant re-stenosis. Vocal function is satisfactory to good in all patients.

The approach described for these combined laryngotracheal lesions provides superior results to those reported using traditional staged and plastic techniques of reconstruction. The collaboration of Otolaryngology and Thoracic Surgery was essential to achieve these results.

*By Invitation

3. A New Video Thoracoscopy Surgical Technique for Interruption of Patent Ductus Arteriosus in Infants and Children

FRANCOIS LABORDE, M.D., PHILIPPE NOIRHOMME, M.D.*,*

JOSEPH KARAM, M.D., ALAIN BATISSE, M.D.**

PATRICK BOUREL, M.D. and*

*OLIVIER SAINT MAURICE, M.D.**

Paris, France

Sponsored by: Francis M. Fontan, M.D., Bordeaux, France

Surgical endoscopy techniques are of growing interest in many fields. Endovascular closure of Patent Ductus Arteriosus (PDA) did not prove to be completely satisfactory. Classical surgical interruption of PDA can be advantageously replaced by Video Thoracoscopy Surgical Interruption (VTSI).

Under general anesthesia and intubation, two 5 mm holes were made through the left thoracic wall. A video camera and different adequate surgical tools were introduced (scissors, dissectors, electrocauthery, retractors, etc.). PDA was dissected and exposed. Two titanium clips were applied, completely interrupting the PDA.

Ten patients with isolated PDA were treated from April to October 1991. Mean age was 18 months (range: 3 months to 6 years). All had successful complete PDA interruption with only VTSI. Hospital stay was short: 2-3 days. The first 4 patients had a 24 hour thoracic drainage while the last 6 did not, thus decreasing post-operative pain. There was neither minor nor major complication and no death.

We conclude that VTSI is sure and safe: all ten PDAs were successfully and completely interrupted as was proved by echo doppler. VTSI is advantageous: decrease of post-operative discomfort and pain, no scar, no rib retraction, no painful thoracic drainage, very short hospital

stay. We now use VTSI routinely in infants and children. Availability of smaller-sized surgical tools should allow VTSI use in prematures and newborns.

9:45 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

10:30 a.m. SCIENTIFIC SESSION

4. Are Two Internal Thoracic Arteries Better Than One?

DELOS M. COSGROVE, M.D., ARTHUR HILL, M.D.,*

BRUCE W. LYTLE, M.D., PAUL C. TAYLOR, M.D.,*

ROBERT W. STEWART, M.D., ROBERTO NOVOA, M.D.*,*

PATRICK M. MCCARTHY, M.D.,*

LEONARD R. GOLDING, M.D.,*

MARLENE GOORMASTIC, MPH* and
FLOYD D. LOOP, M.D.

Cleveland, Ohio

It has been well-documented that survival, reoperation-free survival and freedom from cardiac events are positively influenced by the use of one internal thoracic artery (ITA) graft during myocardial revascularization. To test the hyposthesis that two ITA grafts incrementally improve surgical results, three groups of 327 patients receiving none, one or two ITA grafts were computer matched. Patients were matched according to the year of operation, age, gender, extent of disease, left ventricular function, completeness of revascularization, and history of congestive heart failure. No patients were lost to follow-up; the mean follow-up was 93 ± 37 months with 7,587 patient-years of follow-up available for analysis. There was a statistically significant trend towards improved survival, reoperation-free survival and freedom from cardiac events at eight years as the number of ITA grafts increased.

	Survival	**Reop-free Survival**	**Cardiac Event-free Survival**
Veins	73	67	39
1 ITA	84	79	49
2 ITAs	86	86	63
p value	.003	.0001	.0001

To evaluate the influence of ITA grafting for different age groups, patients were separated into groups <60 and those >60 years of age. In patients <60, there was a statistically significant trend for improving survival, reoperation-free survival and event-free survival at eight years as the number of ITA grafts increased.

	Survival	**Reop-free Survival**	**Cardiac Event-free Survival**
Veins	77	69	41
1 ITA	88	82	51

2 ITAs	93	92	71
p value	.0001	.0001	.0001

The difference between one and two ITA grafts was statistically significant ($p < 0.05$) for cardiac event-free survival for the entire group and patients <60. In patients >60 years of age, less beneficial influence can be seen as the number of ITA grafts increased from one to two.

	Survival	**Reop-free Survival**	**Cardiac Event-free Survival**
Veins	68	64	36
1 ITA	77	76	46
2 ITAs	75	74	51
p value	NS	NS	.04

We conclude 1) two ITA grafts provide an incremental improvement in survival, reoperation-free survival and freedom from cardiac events and 2) this incremental benefit is more pronounced in younger patients.

*By Invitation

5. Superiority of Surgical Reperfusion vs. **PTCS in Acute Coronary Occlusion**

BRADLEY S. ALLEN, M.D., GERALD D. BUCKBERG, M.D.,*

FRANCIS M. FONTAN, M.D.,

MARVINM, KIRSH, M.D., GEORGE POPOFF, M.D.,*

FRIEDHELM BEYERSDORF, M.D.,*

JEAN-NOEL FABIANI, M.D. and*

*CHRISTOPHER ACAR, M.D.**

Los Angeles, California

Although PTCA is successful in > 90%of pts after acute coronary occlusion, overall mortality remains approximately 10% with higher subgroup mortality (i.e. LAD occlusion, multivessel disease, age > 70 yrs, cardiogenic shock) and early recovery of regional wall motion is marginal. This multi-center survey shows that controlled surgical reperfusion in acute coronary occlusion reduces overall and subgroup mortality and restores substantial early contractility.

In a survey from 6 institutions, 156 consecutive pts with acute coronary occlusion documented by angiography underwent surgical revascularization with controlled reperfusion using amino acid enriched blood cardioplegia on total vented bypass. Ventricular wall motion was studied by ECHO or MUG A at post-operative day 5-7, and scored by an independent radiologist (0 = normal, 1 = mild hypokinesia, 2 = severe hypokinesia, 3 = akinesia, 4 = dyskinesia). Results are compared to 1,203 patients with acute coronary occlusion treated by PTCA in 5 reported medical series.

Surgical patients were revascularized at longer ischemic intervals 6.3 vs 3.6 hrs*, had a greater incidence of LAD occlusion 61% vs 43%*, multivessel disease 42% vs 10% and

cardiogenic shock 41% vs 9%* with 12 pts undergoing CPR en route to the operating room. Surgical results were superior in all categories with overall mortality reduced from 8.8% (after PTCA) to 3.9%* after CABG. All surgical deaths occurred in patients with preoperative cardiogenic shock. Regional wall motion recovered significantly (score < 2) in 140/156 (90%) of surgical patients with an average score of 0.9 ± 0.8 (normal to mild hypokinesia) despite longer ischemic times. Subgroup mortality is shown below.

Reperfusion	PTCA (uncontrolled n = 1203)	CABG (controlled n = 156)
Ischemic Time:(hr)	3.6 ± 1.8	6.3 ± 3.6 (1.5-36 hrs)*
Mortality: Overall	105/1203 (8.8%)	6/156* (3.9%)*
LAD occlusion	39/331 (11%)	5/95 (5%)
3 vessel Dis.	27/158 (17%)	0/66 (0%)*
Age ‰¥ 70 yrs	49/209 (23%)	1/22 (5%)*
Preop Shock	49/114 (43%)	6/64 (9%)*

This multi-center study demonstrates that controlled surgical reperfusion lowers mortality in all groups compared to PTCA, despite a longer ischemic time and more pts in cardiogenic shock. Early and substantial return of segmental wall motion in sugical pts suggests superior muscle salvage, supports an aggressive approach to treating acute coronary occlusion, and implies that the mode of reperfusion is more important than the rapidity of reperfusion after acute ischemia.

*$p < 0.05$

11:15 a.m. PRESIDENTIAL ADDRESS

The Association at 75; The Challenge of the Future
(Do we need a stress test?)

John A. Waldhausen, M.D., Hershey, Pennsylvania

12:00 noon ADJOURN FOR LUNCH - VISIT EXHIBITS

*By Invitation

MONDAY AFTERNOON, APRIL 27, 1992

1:30 p.m. SCIENTIFIC SESSION - Los Angeles Ballroom

6. Preoperative Prediction for Cardiopulmonary By-Pass in Lung Transplantation

*ALBERTO de HOYOS, M.D. *, WILFRED DEMAJO, M.D.*,*
TIMOTHY WINTON, M.D., GREGORY SNELL, M.D.*,*
JOHN MILLER, M.D., JANET MA URER, M.D.**
and G. ALEC PATTERSON, M.D.

Toronto, Ontario

Recent innovations in the surgical technique of isolated lung transplantation have made feasible sequential bilateral transplants without car-diopulmonary by-pass (CPB). We analyzed preoperative parameters of car-diopulmonary function that may predict the need for CPB in single (SLT n = 51) and double lung transplant recipients (DLT n = 37). 25/51 SLT

and 14/37 DLT required CPB. Of the 14 DLT, 10 required CPB for implantation of both lungs. The other 4 required CPB for the second implant only. The Table depicts preoperative parameters of cardiopulmonary function analyzed, (*p<0.05 CPB vs NCPB). FP = first pass; RV = right ventricular ejection fraction; LV = left ventricular ejection fraction; SMW = six minute walk; PAP = pulmonary artery pressure; PVR = pulmonary vascular resistance. Results are expressed as mean values ± SD.

SLT		RV		LV		SMW
	FP	**Rest**	**EX**	**Rest**	**Ex**	**m**
NCPB	34*	34*	34*	59	63	382*
SD	(6)	(11)	(12)	(10)	(11)	(100)
CPB	25	24	24	57	58	266
SD	(7)	(12)	(13)	(10)	(12)	(146)
BLT						
NCPB	30	37	38*	57	60	441
SD	(9)	(9)	(8)	(8)	(9)	(161)
CPB	25	33	29	53	58	489
SD	(12)	(7)	(9)	(7)	(9)	(143)
SLT	**$Pa0_2$**	**Oz**	**0_2 Sat**	**Exer**	**PAP**	**PVR**
	mmHg	**1/m**	**%**	**min**	**mmHg**	**d.s.m**
NCPB	56*	2.3*	87*	4.6*	35*	235
SD	(9)	(2)	(5)	(2)	(7)	(54)
CPB	39	5	81	3.4	57	743
SD	(12)	(3)	(6)	(2)	(22)	(490)
BLT						
NCPB	NA	1.8*	89	5.5	28	286
SD		(1.5)	(3)	(3)	(7)	(90)
CPB	NA	3.3	86	6	28	298
SD		(3)	(5)	(4)	(7)	(106)

SLT's required CPB for increased PAP (n = 12), desaturation (n = 8) and systemic hypotension (n = 5). There was no difference in RV and LV function, SMW, and 0_2requirements among these groups. 0_2 saturation < 79% during exercise (N = 6) predicted patients requiring CPB for desaturation. Indications for CPB for both lungs in DLT's were prophylactic (n = 3), desaturation (n = 3), increased PAP (n = 3) and technical (n = 1). Indications for CPB for the second implant were ventricular arrhythmias secondary to hyperkalemia and acidosis (n = 3), and systemic hypotension, desaturation and pulmonary hypertension (n = 1). No difference in postoperative complications and operative mortality were seen between CPB and NCPB groups. We conclude that preoperative parameters of RV function, exercise capacity, $Pa0_2$, oxygen requirements and pulmonary hemodynamics can be utilized to predict SLT who require CPB. In DLT recipients

however, the decision to utilize CPB was made intraoperatively based on the level of PAP, desaturation and hemodynamic instability secondary to ventricular arrhythmias.

*By Invitation

7. Heart Transplantation: Changing Patterns in Patient Selection and Costs

KEITH REEMTSMA, M.D., GRETCHEN BERLAND, B.A.,*

JEFFREY MERRILL, DR. P.H.*
RAYMOND R. ARONS, CRAIG EVANS, ESQ., J.D., MBA,*

RONALD E. DRUSIN, M.D., CRAIG SMITH M.D.,*

and ERIC ROSE, M.D.

New York, New York

Our heart transplant program, now in its fifteenth year, involves experience with 498 recipients, all of whom have been followed with at least annual visits. This report is focused on disturbing trends in our patient selection and costs.

We have been startled to discover that in the last four years the mean hospital cost per transplant in our program has almost tripled. We have looked at possible explanations and have concluded that as our waiting list has increased, we are operating on an increased proportion of the most sick patients.

Between 1988 and 1991, the percentage of ICU-bound patients rose from 34% to 62%. During the same time, the length of stay (LOS) rose from 29.5 days to 44.9 days, and the preoperative length of stay increased from 8.9 to 17.5 days.

We found no significant difference in survival of the ICU-bound versus the other patients at 30 days (90.7 vs 88%), and at one year through 1990 (74.5 vs 75%).

Year	N	%ICU-Bound	LOS-Total	LOS Pretransplant	Cost
1991 (thru 10/14/91)	73	62	44.9	17.5	$129,319
1990	104	47	29.4	4.8	$106,692
1989	79	28	31.3	6.0	$83,703
1988	59	34	29.5	8.9	$50,290

This study documents the trend of sharply rising costs associated with operating on an increasing proportion of the most seriously ill patients. Although survival rates are equivalent in the ICU-bound and the less sick patients, we raise the question of how best to select recipients of these donor organs in limited supply.

*By Invitation

8. Anterior Trans-Cervical Approach for Radical Resection of Lung Tumors Invading the Thoracic Inlet

PHILIPPE G. DARTEVELLE, M.D.,*

ALAIN R. CHAPELIER, M.D., GEORGES S. TABET, M.D.*,*

*BERNARD LENOT, M.D. *, FRANCOIS LE ROY*

*LADURIE, M.D. * and JACQUES CERRINA, M.D. **

Le Plessis Robinson, France

Sponsored by: Jean DesLauriers, M.D., Quebec

Pulmonary tumors invading the thoracic inlet present a surgical challenge. Through the classical posterior thoracic approach, as described by Paulson, one cannot perform complete resection of the cervical extension of the tumor above the thoracic inlet (Subclavian vessels, Brachial plexus, Scalenus muscles, and Phrenic nerve). Therefore, we describe herein an original anterior transcervical approach which is required for a safe exposure and radical resection of cervical structures.

From 1980 to 1991, 29 patients underwent radical resection of such tumors through this approach (Squamous cell carcinoma n = 11, Adenocar-cinoma n = 9, Large cell carcinoma n = 7, and Mixed cell carcinoma n = 2).

A large L shaped cervical incision with removal of the internal half of the clavicle was performed and the following steps were carried out: 1- Dissection free or resection of the subclavian vein when involved in the tumor (n = 7); 2- Section of the scalenus muscles in free margin, and resection of the cervical portion of the phrenic nerve when invaded (n = 6); 3- Exposure of the subclavian artery which was resected in 9 patients (prosthetic replacement n = 7, end to end anastomosis n = 2), and resection of the vertebral artery (n = 5); 4- Dissection free of the brachial plexus up to the spinal foramen, with Tl resection (n = 15); 5- Section of invaded upper ribs in free margins (n = 29); 6- En-bloc removal of chest wall and lung tumor was possible through this single approach in 9 patients (wedge resection n = 7, lobectomy n = 2) with extension of the cutaneous incision into the delto-pectoral groove.

An additional posterior thoracotomy was necessary in the remaining patients for a larger chest wall resection below the second rib. This additional step is less and less required while experience with cervical approach is increasing.

There were no operative deaths. Post operative radio-therapy was given to 25 patients, and chemo-therapy to 11 patients.

Median survival rate was 20,8 months, and cumulative survival rate was 77, 45 and 30% at 1, 2 and 5 years respectively. No significant difference was found among patients with vascular invasion (n = 12). Five with subclavian artery resection are still alive at 4, 18, 19, 28, and 71 months.

Conclusion: - Radical surgery of apical lung tumors invading structures above the thoracic inlet can provide an acceptable survival rate.

- Such a goal can only be achieved through an anterior transcervical approach.

2:30 p.m. BASIC SCIENCE LECTURE

Twinning
Kurt Benirschke, M.D., San Diego, California

3:15 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

4:00 p.m. SCIENTIFIC SESSION - Los Angeles Ballroom

9. Prognosis of Patients With Hypertrophic Obstructive Cardiomyopathy After Transaortic Myectomy. Late Results up to 25 Years.

HAGEN D. SCHULTE, M.D., WOLFGANG H. BIRCKS, M.D.*

*and BENNO LOESSE, M.D.**

Duesseldorf, Germany

Long-term results after surgery for HOCM are less well documented than data concerning the early outcome.

Out of a total series of 353 patients (pts.) (210 males, 143 females, mean age 41.7 years) operated upon since 1963 up to June 1991,262 pts. had trans-aortic myectomy only (mortality rate 3.1%, n = 8), whereas 91 pts. needed additional cardiac procedures (mortality 8.8%, n = 8). Since 1984 the early mortality rate could be reduced to 1.5% (2 of 137 pts.) and 1.8% (1 of 56 pts.), respectively. There were no differences for typical (subvalvular) and for atypical (midventricular) HOCM.

A complete follow-up study could be performed for 309 pts. who survived surgery in the years 1963 to 1989. The longest follow-up time was 25.2 years, the shortest 1 year (mean 6.6 years). During the observation period 36 pts. (11.6%) died. The death of 17 pts. was closely related to HOCM (sudden death n = 8, LV-failure n = 4, embolic events due to atrial fibrillation n = 4, reoperation for residual symptomatic HOCM n = 1). Other causes - non related to HOCM - were responsible for the death of 19 pts. (extracardiac disease n = 15, coronary artery disease n =4, double valve replacement after acute native valve endocarditis n= 1). In consideration of these data there was a total yearly death rate of 2.2%, in close relation to HOCM it was 1.1%.

Most of the survivors (n = 257) belong to clinical class I and II (NYHA) at the end of the observation period (80.9%, n =208). Reoperations because of primarily insufficient myectomy had to be performed in 3 pts. with 1 death. Additional valve replacement was necessary in 4 pts. with 1 death, and 2 pts. had coronary revascularisation.

In conclusion, our long-term follow-up data indicate a reduced late mortality rate after surgery compared to pts. after medical treatment. In symptomatic pts. and failing medical therapy the prognostic benefit of surgery appears to become more and more evident.

*By Invitation

10. Transhiatal Esophagectomy for Benign and Malignant Disease of the Intrathoracic Esophagus

MARK B. ORRINGER, M.D. and
*MACK C. STIRLING, M.D. **

Ann Arbor, Michigan

Since 1977, of 614 patients undergoing a transhiatal esophagectomy (THE), the operation has been performed in 559 with diseases of the in-trathoracic esophagus: 164 (29%) benign and 395 (71%) malignant (6% upper, 30% middle, and 64% lower). The benign esophageal diseases included strictures of varying etiology (41%); neuromotor dysfunction-achalasia (31%), esophageal spasm (7%); acute perforation (10%); acute caustic injury (9%); and others (2%). Among the patients with benign disease, 56% had undergone at least one prior esophageal operation and 29% had a history of between two and six esophageal operations.

THE was possible in 99% of patients in whom it was attempted. Esophageal resection and reconstruction were performed in a single operation in all but 10 patients. The esophageal substitute was positioned in the posterior mediastinum in the orginal esophageal bed in 96%. Stomach was used to replace the esophagus in 544 patients (97%) and colon in 15 (3%) who had undergone prior gastric resections.

Hospital mortality was 2% in patients with benign disease and 5% in patients with carcinoma. There was 1 intraoperative death. Complications included intraoperative entry into a pleural cavity requiring a chest tube (73%), anastomotic leak (8%), recurrent laryngeal nerve injury (6%), and chylothorax (< 1%). Three patients required re-operation for mediastinal bleeding. Average intraoperative blood loss was 928 ml (1136 ml for benign disease and 833 ml for carcinoma). 513 patients (91%) were discharged able to swallow within three weeks of operation. The actuarial survival of the patients with carcinoma is similar to that reported after more traditional transthoracic esophagectomy. Among patients with benign disease, excellent functional results have been achieved in more than 84% after a cervical esophagogastric anastomosis. While approximately 50% have required one or more anastomotic dilations within 1-3 months of operation, true anastomotic strictures have developed in <7%. Clinically significant gastroesophageal reflux has occurred postoperatively in <1%. THE is feasible in most patients requiring esophageal resection for either benign or malignant disease and is a safe, well-tolerated operation if performed with care and for the proper indications.

*By Invitation

11. Long-Term Function of Cryopreserved Aortic-Valve Homografts: A 10 Year Study

JAMES K. KIRKLIN, M.D., DAVID C. NAFTEL, Ph.D.,*

WILLIAM NOVICK, M.D., DENNIS C. SMITH, M.D.*,*

ALBERTO. PACIFICO, M.D., JOHN W. KIRKLIN, M.D.,

ROBERT C. SOURCE, M.D., SANDRA J. PHILLIPS, B.S.*,*

*and NAVIN C. NANDA, M.D. **

Birmingham, Alabama

Cryopreserved aortic valve (AV) homografts have become an accepted AV substitute, but long-term studies with echocardiographic (echo) assessment of valve function are largely unavailable. Therefore, the following study of our 10-year experience with AV homografts was undertaken. Between 1981 (the introduction of Cryopreserved homografts at our institution) and 4/1/90, 163 Cryopreserved AV homografts were implanted in 163 patients (pts) (ages 9 mo to 80 yrs, median 46 yrs) of which 148 were implanted in the infra-coronary position and 15 as aortic root replacements. Serial 2-Dimensional (2-D) echo studies (n = 322) were obtained in 136 pts 1 day to 108 months after operation, with 35 pts receiving 2-D echo studies more than 5 yrs post-op.

Overall survival was 93% at 1 yr and 90% at 5 yrs. Survival of pts undergoing isolated infra-coronary free-hand homograft AV replacement was 95% at 1 yr, 93% at 5 yrs, and 92% at 8 yrs. Eleven homograft valves were ex-planted, 2 at the original operation due to obstruction, 6 at 0.5 to 41 mos due to aortic regurgitation (AR) (n = 4) or left ventricular outflow obstruction (n = 2), and 3 at 6, 35, and 92 mos for probable degeneration with progressive incompetence (n = 2) or calcific stenosis (n = 1). Freedom from explantation for any reason was 83% at 8 yrs. Surviving patients had a mean NYHA Class of 1.2 at 5 yrs and 1.4 at 8 yrs post-implantation. Mean echo AR (grade 0, no AR to grade 4, severe AR) was 0.5 at 1 mo, 1.0 at 1 yr, 1.3 at 5 yrs and 1.4 at 8 yrs post-

implantation. *"Probable valve degeneration,"* as evidenced by valve degeneration at explant, severe AR (4/4) by post-op echo without reoperation, and/or death related to valve failure was identified in 5 pts at 6, 35, 36, 60, and 92 mos after implantation. The overall freedom from probable valve degeneration was 97% at 5 yrs and 85% at 8 yrs. By multivariable analysis, no specific risk factors for degeneration were identified.

Inferences:

* Early and late survival is excellent following homograft AVR with an 8 yr survival of 92% after isolated homograft AVR.

* The quality of life is excellent with little progression of AR in most pts for at least 9 yrs.

* Valve degeneration is unusual during the first 8-10 yrs, but may rarely occur after about the first 6 mos. Eighty-five % of patients are expected to be free of valve degeneration at 8 yrs.

*By Invitation

12. Experience in 112 Pulmonary Thromboendarterectomy Operations Over a Two Year Period

STUART W. JAMIESON, M.D., WILLIAM R. AUGER, M.D.,*

*PETER F. FEDULLO, M.D. *,*

*RICHARD N. CHANNICK, M.D. * and*

*KENNETH M. MOSER, M.D.**

San Diego, California

Three hundred pulmonary thromboendarterectomy operations have been performed at this institution since 1970. Of these, 112 were done by one surgeon over the last 24 months. The operation involves a median ster-notomy incision, the institution of cardiopulmonary bypass, and cooling with circulatory arrest. Incisions are made in both pulmonary arteries into the lower lobe branches. Pulmonary thromboendarterectomy is always bilateral, with removal of both thrombus and an endarterectomy plane from all involved lobes. The right atrium is routinely explored for atrial septal defects. Changes in the technique have been made to allow more thorough revascularization, and shorter circulatory arrest times. This has produced improved results.

Comparison of the last 100 cases (February 1990 to October 1991) to 100 cases done prior to the institution of new methods showed a mean total circulatory arrest time of 34.35 ± 14 vs 58.14 ± 23.5 ($p < 0.0001$). The incidence of transient post-operative delirium decreased to 10% from 26%, post-operative arrhythmias 15% from 26% and mortality 9% from 16%. The majority of cases have exhibited normal post-operative hemodynamics, and late functional results have been excellent.

Chronic pulmonary thromboembolism that is surgically correctable is. likely an under-diagnosed entity. Pulmonary thromboendarterectomy can be performed with an acceptable risk and good symptomatic results.

*By Invitation

TUESDAY MORNING, APRIL 28, 1992

7:30 a.m. FORUM SESSION I - Cardiac Surgery - Los Angeles Ballroom

F1. Pathogenesis of Ischemic Mitral Insufficiency

MARIO R. LLANERAS, M.D.,*

STEPHEN W. DOWNING, M.D., JOAO A. C. LIMA, M.D.*,*

MICHAEL L. NANCE, M.D., RADU C. DEAC, M.D.,*

PHILIP L. LINDEN, B.A. and*

L. HENRY EDMUNDS, JR., M.D.

Philadelphia, Pennsylvania

We hypothesized that the combination of a moderate sized posterior in-farct and infarction of the posteromedial papillary muscle (PPM) were required to cause ischemic mitral regurgitation (MR); neither condition alone would cause MR. In 72 sheep (19 slaughterhosue hearts, 25 acute experiments and 28 chronic experiments) the coronary artery anatomy was detailed by gross inspection, dye injections in cadaveric hearts, vital staining and coronary angiography. Ligation of the first and second marginal (OM1 & OM2) branches of the left circumflex artery consistently infarcted 23 ± 3.3% (by planimetry) of the left ventricular mass. Color flow doppler performed in-traoperatively in 12 sheep with the OM1/OM2 infarct, revealed no appreciable MR up to eight weeks postinfarct despite dilatation on the left ventricle (LV).

Ligation of the second and third (OM2 & OM3) marginal branches, in 16 sheep, infarcted 21.4 ± 4.0% of the LV mass and completely infarcted the PPM in all but two animals. Wall thickness at the level of the PPM is 1.6 ± 0.2 cm before infarction and decreases to 0.4 ± 0.1 cm 8 weeks after infarction. Left ventriculography and intraoperative color flow echocardiography demonstrated MR which progressed in severity following infarction. Eight sheep have been followed serially for 8 weeks; two had incompletely infarcted PPM and did not develop IMR. Three other sheep developed severe mitral regurgitation immediately after infarction and died early. Five sheep with MR have not reached 8 weeks (cf. graph). The consistency of coronary arterial anatomy in sheep and lack of collaterals result in anatomically uniform infarcts between sheep and predictable ischemic MR. The data show that a 23% posterior infarction sparing the PPM or an infarction of the PPM alone does not produce MR. Both conditions must be present. This preparation provides a reproducible model of ischemic MR and offers a means to devise repairs based on the pathophysiology of the disease.

*By Invitation

F2. Very Small Diameter Polyurethane Vascular Prostheses With Rapid Endothelialization for Coronary Artery Bypass Grafting

TAKAFUMI OKOSHI, M.D., GIORGIO SOLDANI, Sc.D.**

MOSES GODDARD, M.D. and*

*PIERRE M. GALLETTI, M.D.**

Providence, Rhode Island

Sponsored by: Karl E. Karlson, M.D.,

Providence, Rhode Island

The growing incidence of "redo" coronary artery bypass grafting (CABG) calls for the development of satisfactory prosthetic substitutes for the internal mammary artery or the saphenous vein. Porous non-woven tubular fabrics provide one approach to that challenge. Two types of spongy polyurethane-polydimethylsiloxane (Cardiothane 51™, Kontron Instruments, Inc.) vascular grafts with an internal diameter (ID) of 1.5 mm were fabricated by a spray, phase-inversion technique. Low porosity grafts (LPG) with hydraulic permeability (HP) of 2.7 ± 0.4 ml/min.cm^2, and medium porosity grafts (MPG) with HP of 39 ± 8 ml/min/cm^2, displayed good handling properties and suturability. Twelve straight LPG, seventeen straight MPG (1.5 - 2.0 cm in length) and one loop MPG (10 cm in length) were implanted by the same surgeon end-to-end in the infrarenal aorta of 30 male Sprague-Dawley rats. No antithrombogenic agents were administered pre- or postoperatively. At 3 months postimplantation, patency was 8% for LPG (1/12) and 76% for the straight MPG (13/17). The loop MPG was also patent. The sole patent LPG showed neointimal hyperplasia and incomplete endothelialization. All but one of the patent straight MPG showed a glistening and transparent neoin-tima with complete endothelialization. The loop MPG displayed endothelialization from each anastomosis and in many islands in the middle portion of the graft, totalling 47% of the luminal surface by morphometric analysis. Thick mural thrombus, anastomotic hyperplasia, or aneurysm formation were not observed in patent MPG. These data indicate that in the rat aortic replacement model it is possible to achieve patency and a high degree of endothelialization in very small diameter prostheses of appropriate porosity. This may open an avenue to the use of synthetic grafts in aorto-coronary bypass surgery.

*By Invitation

F3. Cardiopulmonary Dysfunction Produced by Initiating Reoxygenation on Cardiopulmonary Bypass in Immature Hypoxemic Piglets: Prevention by Intravenous Metabolic Treatment

GEORG MATHEIS, M.D. GERALD D. BUCKBERG, M.D.,*

DENIS B. TIXIER, M.D., HELEN H. YOUNG, Ph.D.**

*and MICHAEL P. SHERMAN, M.D.**

Los Angeles, California

This study tests the hypothesis that reoxygenation injury is produced when Cardiopulmonary bypass (CPB) is initiated in immature hypoxemic piglets, and causes cardiopulmonary dysfunction that can be avoided by intravenous metabolic treatment before and during CPB.

Of eighteen immature Yorkshire-Duroc piglets (<3 weeks old) six were anesthetized, instrumented and observed over 5 hours (control). Twelve piglets underwent up to 2 hours of hypoxemia (paO_2 = 20-30 mmHg) before initiation of reoxygenation on CPB. Six received an intravenous metabolic infusate (mercaptopropionyl glycine, catalase, aspartate, glutamate, glucose/insulin) which was started before and continued during CPB.

Hypoxia produced an initial hyperdynamic response (39% increased cardiac index) followed by progressive hemodynamic deterioration. This required premature initiation of bypass in 8 out of 12 hypoxemic piglets (67%).

	Control	**NOR_X**	**Intravenous R_x**
Stroke Work Index @ LAP 8 mmHg	1.08 ± 0.16	0.66 ± 0.06*	1.07 ±0.09
Conjugated Dienes (A_{233} nm/mg lipid)	0.73 ± 0.10	1.31 ± 0.14*	1.01 ± 0.09
Pulmonary Vascular Resistance Index	83 ± 12	294 ± 34*	190 ± 27
Static Lung Compliance (% Control)	100 ± 8%	78 ± 4%*	105 ± 21%
a/A pO_2 Ratio #	0.66 ± 0.04	0.33 ± 0.06*	0.62 ± 0.07

(values are mean ± SEM,* = p<0.05 vs. Control, ANOVA, ^arterial/alveolar pO, Ratio)

Reoxygenation induced injury (assessed 30 minutes after CPB) was characterized by reduced stroke work index, increased myocardial lipid peroxidation (conjugated dienes), increased pulmonary vascular resistance index, impaired statis lung compliance, and decreased a/A pd ratio. These reoxygenation changes were avoided by intravenous metabolic treatment.

We conclude that the reoxygenation of immature hypoxemic piglets by initiating CPB results in cardiopulmonary dysfunction which may increase vulnerability to subsequent ischemia (i.e. aortic crossclamping) and is preventable by intravenous metabolic treatment before and during CPB needed for cardiac repair.

*By Invitation

F4. Normocalcemic Blood or Crystalloid Cardioplegia Provides Superior Neonatal Myocardial Protection Over Low Calcium Cardioplegia

JEFFREY M. PEARL, M.D., HILLEL LAKS, M.D.,*

DAVIS C. DRINKWATER, M.D.,*

AVEDIS MENESHIAN, B.S. and PAULA. CHANG**

Los Angeles, California

Although standard blood cardioplegia provides good myocardial protection for adult cardiac surgery, protection of the cyanotic, immature myocardium remains suboptimal. Calcium, which has been implicated in reperfusion injury and the development of "stone heart" in mature myocardium, is routinely removed from standard cardioplegia solutions. Immature, neonatal myocardium has lower intracellular calcium stores and is more reliant on extracellular calcium for contraction, and for maintenance of the glycocalyx membrane. In order to determine if normocalcemic cardioplegia would result in improved cardiac function in the neonatal heart, we conducted a series of experiments using an isolated, blood perfused working heart model. Neonatal piglet hearts (24-48 hours) were excised without intervening ischemia and placed directly on a blood perfused circuit. Baseline stroke-work index (SWI) was assessed. Hearts were then arrested with cold cardioplegia delivered at 45 mmHg for 2 minutes; Group I = low-Ca + 2 Blood CP (Ca = 0.6 mmol/1); Group II = normal-Ca + 2 blood cardioplegia (Ca=1.1 mmol/1); Group III = University of Wisconsin (UW) solution; and Group IV = UW with added calcium (Ca= 1.0 mmol/1). Each group consisted of 8 hearts. Cardioplegia was administered every 20 minutes for 2 hours and topical hypothermia was employed. Hearts were then reperfused with warm whole blood. Functional recovery, expressed as percent of control SWI, was determined 60 minutes following reperfusion.

Hearts preserved with normocalcemic CP (Groups II and IV) had complete (100%) functional recovery at 60 minutes, whereas hearts preserved with low-calcium CP (Groups I and III) had only 80% and 58% recovery at LAP of 9 mmHg, respectively. Percent recovery decreased further at higher left atrial pressures in the low calcium groups, indicating diastolic dysfunction. Electronmicrographs taken 1 hour after reperfusion showed minimal edema and only mild myofibrillar changes, and were identical in both the low calcium and normal calcium groups. **Conclusion:** 100% functional recovery is possible in immature myocardium when calcium is added to either blood or crystalloid cardioplegia. The addition of calcium does not result in ultrastructural damage ("calcium paradox") and results in superior functional recovery.

By Invitation

F5. Effect of Calcium and Preischemic Hypothermia on Recovery of Myocardial Function After Cardioplegic Ischemia in Neonatal Lambs

MITSURU AOKI, M.D., JOHN E. MAYER, JR., M.D.,*

FUMIKAZU NOMURA, M.D.* and
*HIROAKI KAWATA, M.D.**

Boston, Massachusetts

Most neonatal cardiac operations are peformed at deep hypothermia, but controversy exists over the danger of hypothermia prior to cardioplegic arrest. Hypothermia can cause myocyte Ca accumulation experimentally and has been noted to result in reduced postischemic recovery in some

experimental and clinical reports. Preischemic hypothermia is routinely used in other centers with good results. To explore this discrepancy we evaluated the effects of preischemic ionized (i) Ca concentration and temperature on outcome after 2 hrs and 15°C cardioplegia-protected ischemia in 38 isolated, blood perfused neonatal lamb hearts. LV maximal developed pressure (DP), dP/dt, -dP/dt, coronary blood flow (CBF), and oxygen consumption (MVO_2) were measured before and 30 and 60 minutes after 2 hours of ischemia. After baseline measurements, hearts were perfusion-cooled (groups B, C, and D) for 10 minutes to 17 °C and then arrested with cold (4°C) St. Thomas cardioplegia. Group A had 10 minutes of *normothermic* perfusion after baseline measurements, then were arrested with the cold cardioplegia. Group B had cooling normal iCa. Group C had citrate (Cit) added as cooling was started to low iCa and iCa was not normalized until 15 minutes into reperfusion. Group D received citrate plus Ca to give normal iCa during cooling. Results are given as % recovery of baseline except iCa (nM/1).* =$p<0.05$ vs groups B and D.

		Pre	Cooling	30 minutes reperfusion				60 minutes reperfusion		
Group	**n**	**iCa**	**iCa**	**iCa**	**DP**	**dP/dt**	**-dP/dt**	**DP**	**dP/dt**	**-dP/dt**
A (No Cooling)	8	0.99	-	1.00	92.8*	86.0*	76.2*	85.7*	72.1*	64.0
B (Cooling)	8	1.06	1.06	1.06	79.0	69.9	66.8	72.0	60.0	57.2
C (Cooling + Cit)	8	1.06	0.26*	1.10	91.6*	84.7*	77.8*	86.5*	74.8*	71.3*
D (Cooling + Cit + Ca	8	1.02	1.03	1.07	67.6	62.2	54.7	59.7	51.9	46.6

MVO_2 per beat significantly increased while the coronary blood flow decreased during preischemic cooling ($p<0.05$) in all groups. CBF was higher in Group C than in other groups during both pre and postischemia as long as iCa was low ($p<0.05$).

These data suggest that preischemic hypothermia results in reduced postischemic recovery of function than *simultaneous* induction of cardioplegia and hypothermia. Low iCa during preischemic hypothermia and early reperfusion offsets these effects. Therefore, careful attention to both temperature and iCa prior to ischemia are important in the outcome after ischemia in neonatal hearts.

*By Invitation

F6. Temporary Leukocyte Depletion Reduces Ventricular Dysfunction During Prolonged Postischemic Reperfusion

IAN C. WILSON, MB, ChB, TIMOTHY J. GARDNER, M.D.,*

JOSEPH M. DiNATALE, B.S.,*

A. MARC GILLINOV, M.D., WILLIAM E. CURTIS, M.D.**

*and DUKE E. CAMERON, M.D.**

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Previous experiments in our laboratory have demonstrated that leukocyte depletion improves *early* postischemic ventricular performance in a neonatal model of global myocardial ischemia. However, the rate at which leukocytes return to the circulation after cardiopulmonary bypass (CPB), their possible late accumulation in the myocardium and their subsequent effect on the functional recovery of left ventricle is not known.

The present study examined the effect of leukocyte depletion on myocardial performance during the 6 hour period post CPB in an *in situ, in vivo* porcine model of neonatal cardiac surgery. Median sternotomy was performed on thirteen 3-5 day old piglets, 6 controls and 7 leukocyte depleted animals (LD), and left ventricular (LV) short-axis sonomicrometry crystals and an in-traventricular micromanometer positioned. Piglets were cooled to 22 °C prior to 90 minutes hypothermic ischemia after a single dose of cold crystalloid car-dioplegia. Mechanical leukocyte depletion was achieved using Pall RC100 filters. Granulocyte counts in the initial coronary reperfusate were reduced to 0.8% of controls (2±1 cells/ml, mean±SEM, $p<0.001$). However, circulating granulocyte counts progressively increased throughout the period of myocardial reperfusion reaching 66% of controls (874±356/ml) after 6 hours ($p<0.3$). Control piglets demonstrated an immediate reduction in postischemic *left ventricular performance* (measured by preload recruitable stroke work, PRSW) to 87 ± 6% baseline. PRSW further declined to a nadir of 73 ± 8% at 4 hours before improving to 86 ± 6% 6 hours postoperatively. In contrast, LV performance in LD animals were 94 ±6% baseline immediately post CPB and did not decline throughout the period of observation, remaining 98 ± 5% and 98 ± 4% baseline at 4 and 6 hours, respectively ($p<0.02$). Left ventricular *systolic function* (measured by end-systolic pressure-volume relationship) and *ventricular compliance* similarly were better preserved in the LD group throughout the postoperative period ($p<0.05$ and $p<0.04$, respectively). This improvement in postischecmic ventricular function was associated with decreased myocardial water content at 6 hours (79.6 ± 0.9%) compared to the control group (80.9 ± 0.8%, $p<0.02$), and with reduced tissue myeloperoxidase concentration (46% of controls,$p<0.05$), representing less myocardial leukocyte accumulation.

These data show that leukocyte depletion during initial reperfusion reduces myocardial leukocyte accumulation and results in *sustained* improvement of postischemic LV function. Temporary manipulation of the leukocyte population during cardiopulmonary bypass can reduce myocardial injury and improve myocardial protection following prolonged reperfusion despite rapid return of granulocytes to the circulation within 6 hours.

*By Invitation

F7. Alternative Methods of Retrograde Cardioplegia Delivery: Effects on Preservation of the Ischemic Left Ventricle After Acute Coronary Artery Occlusion and Reperfusion

JAMES T. DIEHL, M.D. MICHAEL PONTORIERO, M.D.*,*

*RAYMOND CONNOLLY, Ph.D.**

STEVEN SCHWARTZ, M.D. and*

RICHARD J. CLEVELAND, M.D.

Boston, Massachusetts

Myocardial protection may be enhanced by altering the flow (intermittent vs continuous) and temperature (cold vs warm) of retrograde blood car-dioplegia. 24 dogs were randomized into 4 cardioplegia groups (n = 6): Group I - intermittent cold antegrade, Group II - intermittent cold retrograde, Group III - continuous warm retrograde, and Group IV - continuous cold retrograde. Preservation was assessed with a model of left ventricular ischemia and reperfusion induced by acute occlusion of the LAD. Functional parameters were collected at baseline, at 60 minutes of LAD occlusion (ischemia), and following reperfusion. All dogs were maintained normother-mic during cardiopulmonary bypass (CPB) and all data was collected off CPB. Regional and global LV

function is reported as the load independent slope (E max) of fiber segment and minor LV axis dimension vs LV end systolic pressure respectively. Group I demonstrated no recovery of global LV function (#).Recovery of global LV function was similar for Grp II, III, and IV (*). Regional LV function demonstrated significant functional recovery only for Group IV, continuous cold retrograde cardioplegia (**). 2-D echo wall motion studies confirm this data.

E max	**GrpI**(n = 6)	**GrpII**(n = 6)	**GrpIII** (n = 6)	**GrpIV** (n = 6)
Global Ischemia	18±2	22±2	20±3	18±2
Post-reperfusion	17±5#	36 ±3*	38 ±2*	38 ±5*
Regional				
Ischemia	49 ±10	47 ±10	51±4	43 ±6
Post-reperfusion	48 ±26	72 ±48	112±29	129 ±28**

NS vs ischemia

*P<0.05 vs ischemia and vsGrpI post-reperfusion

"P<0.05 vsGrpIV ischemia - All data expressed as mean ± SEM

Conclusions:

1. Retrograde cardioplegia provides superior global LV preservation during ischemia when compared with antegrade cardioplegia.
2. Preservation of regional myocardium beyond an occluded coronary artery is best achieved with cold continuous retrograde cardioplegia.

*By Invitation

F8. Detrimental Effects of Interrupting Warm Blood Cardioplegia During Coronary Revascularization

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XI MING YANG, M.D., SAMUEL RIVERS, B.S.*,*

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Warm Blood Cardioplegia (WBC) has emerged as an alternative method of myocardial protection to Cold Blood Cardioplegia (CBC). However, the continuous infusion of blood required in this technique may obscure the operative field necessitating that the WBC be interrupted. The effects of interrupting WBC during coronary revascularization are unknown. This experimental study was therefore undertaken to determine whether interrupting WBC during coronary revascularization will result in increased myocardial damage.

In 30 adult pigs, the second and third diagonal vessels were occluded with snares just beyond the LAD for 1 ½ hours. All animals were then placed on cardiopulmonary bypass and underwent 45 minutes of cardioplegic arrest. Following aortic unclamping, the coronary snares were released and all hearts were reperfused for 3 hours. During the period of cardioplegic arrest, 10 pigs received intermittent, antegrade/retrograde CBC (4°C), 10 animals received continuous, retrograde WBC (37°C) at 100 ml/min; and 10 animals received continuous, retrograde WBC but had the infusion stopped for three 7 minute periods during the 45 minute crossclamp period. The effectiveness of myocardial protection in the area at risk was assessed by myocardial pH measured during cardioplegic arrest using tissue pH probes, Wall Motion Scores (WMS) using 2-D echo (4 = normal to -1 = dyskinesia), and the Area of Necrosis/Area of Risk (AN/AR) using histochemical staining. Results are Mean±SE;*p<.05 from Antegrade/Retrograde CBC; $_{+}$p<.05 from Retrograde WBC.

	Antegrade/Retrograde CBC	**Retrograde WBC**	**Interrupted Retrograde WBC**
pH	6.98 ± .17	6.45 ± .12*	6.20 ± .16
WMS	3.3 ± .4	2.8 ± .4	2.06 ± .30*
AN/AR (%)	21 ± 2	25 ± 2	38 ± 5$_{+}$*

Interrupting Retrograde WBC resulted in more tissue acidosis during acrioplegic arrest, lower wall motion scores, and increased tissue necrosis. We conclude that interrupting WBC during coronary revascularization diminishes the effectiveness of WBC and results in increased ischemic damage.

*By Invitation

F9. Complete Prevention of Myocardial Stunning, Low-Reflow and Edema After Heart Transplantation by Blocking Leukocyte Adhesion Molecule During Reperfusion

JOHN G. BYRNE, M.D., LAWRENCE H. COHN, M.D.,*

WENDEL J. SMITH, M.D., MICHAEL P. MURPHY, M.D.*,*

GREGORY S. COUPER, M.D. and*

*ROBERT F. APPLEYARD, Ph.D.**

Boston, Massachusetts

Following heart preservation and transplantation, reperfusion-induced microvascular inflammation with release of leukocyte-derived toxic mediators (oxygen free radicals, enzymes) has been suggested as an underlying mechanism leading to edema, "low-reflow" and subsequent *diastolic* dysfunction ("diastolic stunning"). For lekocytes to mediate inflammation and release their toxic mediators they must first adhere to either each other or to the endothelium. We therefore hypothesized that *preventing leukocyte adhesion,* by blocking leukocyte membrane adhesion molecule CD18, would reduce myocardial inflammation and edema and improve reflow and diastolic function after heart preservation and transplantation.

Methods: After cardioplegia and insertion of an LV balloon, rabbit hearts were heterotopically transplanted into recipient rabbits either immediately (Immediate, *n* = 12) or after preservation in

4°C saline (3 hrs ischemia, *n* = 23). Recipients of preserved hearts received, 45 min before reperfusion, either IV saline (Placebo, *n* = 13) or IV anti-CD18 monoclonal antibody R15.7 (2 mg/Kg) (Anti-CD18, *n* =10). During 3 hours reperfusion the slope of the end-systolic pressure-volume relation (Emax), the exponential elastic coefficient of the end-diastolic pressure-volume relation (Î²), the unstressed ventricular volume (V_0) and the time constant of the exponential LV pressure fall after dP/dt_{min} (Ï„) were serially measured. Myocardial blood flow was measured with microspheres from which coronary vascular resistance (CVR) was calculated. After explantation the degree of myocardial inflammation, estimated by tissue leukocyte sequestration (Myeloperoxidase assay, MPO), and myocardial water content ($\%H_2O$) were determined.

		E_{max}	□	V_0	□	CVR	MPO	$\%H_2O$
Group	n	(mmHg/ml)		(ml)	(msec)	U/g	(□**U/g**)	
Immediate	12	63. 3 ± 2.7	3.4 ± 0.3	0.3 ± 0.2	37.9 ± 3.8	70.5 ± 10.6	1712 ± 552	75.6 ± 1.3
Placebo	13	67.6 ± 3.1	3.1 ± 0.3	-0.5 ± 0.3	157.7 ± 4.4*	1 15.5 ± 13.4#	3380 ± 456#	79.8 ± 0.4#
Anti-CD18	10	57.6 ± 3.6	4.3 ± 0.3	0.5 ± 0.3	32.7 ± 5.0†	64.8 ± 4.5‡	1100 ± 308‡	75.2 ± 1.7‡

Values: Mean ± SEM;*p<0.01 vs Immediate; †p<0.01 vs Placebo; #p<0.05 vs Immediate; ‡p<0.05 vs Placebo (ANOVA)

Increased MPO, prolonged T, elevated CVR and increased $\%H_2O$ observed in Placebo hearts were all completely averted by Anti-CD18 treatment, demonstrating complete prevention of myocardial inflammation, diastolic stunning, low-reflow, and edema.

Conclusion: Leukocyte adhesion is a critical initiating event in reperfusion-induced myocardial damage after heart transplantation and should be addressed in treatments directed at limiting reperfusion injury.

*By Invitation

F10. A Long-Term Ventricular Assist System

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GERSON ROSENBERG, Ph.D., WILLIAM WEISS, M.S.*,*

WALTER E. PAE, JR., M.D. and

JOHN A. WALDHAUSEN, M.D.

Hershey, Pennsylvania

While cardiac transplantation is excellent therapy for certain patients with end-stage heart disease, the number of donor hearts is far less than the need. A permanent left ventricle-to-aortic assist pump has the potential of providing long-term systemic circulatory support in patients for whom donor hearts cannot be obtained.

Our multidisciplinary group is developing an implantable, electrically powered pump that will provide tether-free circulatory support. The blood pump consists of a seamless polyurethane sac within a polysulfone case. Bjork-Shiley monostrut valves provide unidirectional flow. The blood sac is compressed by a pusher plate actuated by a brushless DC electric motor-motion translator.

The system has gradually evolved from one in which a tube crossed the skin, serving as a conduit for electrical wires, to the most recent model in which the unit is completely sealed and inductive coupling is used to transfer electrical energy across the intact skin. The current model has an implantable miniature control system as well as a battery that provides 30 minutes

of operation when the external coil is disconnected. However, during normal operation, the pump is powered by a portable battery pack or by house current.

The pump has a stroke volume of 62 ml and is capable of pumping 8.5 *l*/min, (l0mmHg filling pressure, 120mmHg outlet pressure). Extensive mock loop testing has demonstrated progressive improvement in system reliability.

Twenty-six chronic animals have had circulatory support with an average period of pumping of 62 days, the longest period of support being eight months. Experiments were terminated in 18 animals for pump related problems (electromechanical 8, moisture related 6, sac rupture 4) and in 7 for animal related problems (thromboembolic 3, bleeding 2 and infection 2). One animal is ongoing.

Studies to date are very encouraging and suggest that, with further refinement, a reliable assist pump can be developed that will have important clinical application.

*By Invitation

TUESDAY MORNING, April 28, 1992

9:00 a.m. SCIENTIFIC SESSION - Los Angeles Ballroom

13. Aortic Valve Replacement With Pulmonary Homografts: Early Experience

GINO GEROSA, M.D., DONALD N. ROSS, F.R.C.S.*

PETER E. BRUECKE, M.D., ANTONI J. DZIATKOWIAK, M.D.,*

SOPHIA MOHAMMAD, MSc and*

*DINO CASAROTTO, M.D.**

Verona, Italy; Linz, Austria; Krakow, Poland;

London, England

The increased use of aortic homografts (AH) as aortic valve substitute and the limited availability of donor valves prompted us to consider the pulmonary homograft (PH) as an alternative substitute for aortic valve replacement (AYR). The aim of our study is to compare the morphologic, ultrastructural and biomechanical properties of PH leaflets with the AH leaflets and to present the early phase results using PH for AYR. Light, scanning and transmission electron microscopy have shown that PH leaflets are thinner than the aortic with a lesser content of elastic tissue in the ven-tricularis layer. Moreover there were not marked differences in the ultrastruc-ture. Uniaxiale tensile tests were carried out on 36 cusps from human pulmonary and aortic valves using an Instron test machine. The strain at 200 KPa was found to be similar for both pulmonary and aortic leaflets (approx 26%) cut radially. Circumferential strips appear to be more extensible in pulmonary leaflets, than in aortic (15% and 9% respectively). The ultimate tensile strength (UTS) for circumferential strips was found to be one and a half times as large for aortic when compared with pulmonary, but there was relatively little difference between the radial strips. As far as clinical experience is concerned, from September 1988 through September 1991, 107 consecutive patients 20-78 years old received either fresh-antibiotic or cryopreserved PH for AYR. The PH's were inserted in place of patients' diseased aortic valve with two different techniques: freehand in subcoronary position or as a "short cylinder" inside the aortic root. There,were 3 hospital deaths (2.8%). Follow-up was complete (1-36 mo.), all surviving patients have been followed with serial color flow Doppler echo.cardiography. There were no late deaths. 3 patients (2.9%) underwent

reoperation because of severe aortic regurgitation (1,4 and 15 mo. post-op.) due to technical problem (mismatch in size between PH and aortic annulus) in 2 cases and probably due to graft rejection in 1 case. Mild aortic regurgitation has been detected in 3 patients (2.9%). No patients incurred in thromboembolicepisodes or infective endocarditis. According to our results the PH has shown to have ultrastructural and biomechanical properties similar to that of the AH. Furthermore PH has shown to be more pliable and easier to insert giving promising short term results.

*By Invitation

14. Infant Repair of Complete Atrioventricular Canal Defects: 20 Year Trends

FRANK L. HANLEY, M.D., RICHARD A. JONAS, M.D.,*

JOHN E. MAYER, JR., M.D. and

ALDO R. CASTANEDA, M.D.

Boston, Massachusetts

Three hundred nineteen infants with complete atrioventricular canal defects have undergone surgical repair at our institution from 1972 to 1991. Analysis of these cases by 5 year intervals reveals a number of important institutional trends.

	1972-77	**1977-81**	**1982-86**	**1987-91**
Caseload	13	63	107	136
Assoc. Lesions	16%	22%	22%	30%
1° Repair	56%	81%	87%	88%
Reoperations Within 6 mo	31%	19%	15%	8%
Age 0-6 mo. (Hosp. Deaths)	6(2)	26(7)	48(8)	70(1)
Age 7-12 mo. (Hosp. Deaths)	7(4)	37(11)	59(5)	66(3)

These trends show increasing volume and complexity of cases over time. In spite of this the number of cases undergoing primary repair increased and the need for early reoperation and the in hospital mortality have decreased markedly. The most recent 5 year experience reveals a 1.5% mortality in patients aged 0 to 6 months at the time of surgery and a 4.5% mortality in patients aged 7 to 12 months (overall 2.9% in hospital mortality).

These data support primary repair in infancy as the procedure of choice and suggest that repair before 6 months may provide further benefit. Further analysis suggests that precise surgical technique, primarily with avoidance of residual mitral regurgitation and to a lesser extent with avoidance of residual ventricular level shunting, is an important factor in the improved outcome and can be reliably achieved in early infancy.

*By Invitation

15. Deep Hypothermia and Circulatory Arrest: Determinants of Stroke and Early Mortality in 656 Adult Patients

LARS G. SVENSSON, M.D., E. STANLEY CRAWFORD, M.D.,*

KENNETH R. HESS, M.S., JOSEPH S. COSELLI, M.D.**

*and HAZIM J. SAFI, M.D.**

Houston, Texas

Deep hypothermia with circulatory arrest are being used more often for complicated cardiovascular surgery, particularly for repair of the aortic arch and acute aortic dissection. There are, however, few large studies that have documented the safety of this technique in adults. We have therefore evaluated our results for this type of adjunct to determine the independent predictors of early death and postoperative stroke by logistic regression analysis. Of the 656 patients operated upon between 7/7/79 and 1/30/91, 43% (N = 283) were female, median age was 64 years (range 10 to 88 years), 12% (N = 77) had acute dissection, 26% (N = 173) had previously had cardiac or ascending aortic surgery, and 13% (N = 85) had a history of cerebrovascular disease. The median circulatory arrest time was 31 minutes (range 7 to 120 minutes). The univariate predictors of transient or permanent stroke, either global or hemiparetic, which occurred in 44 patients (7%), were ($p<0.05$): increasing age, history of cerebrovascular disease, circulatory arrest time (7-29 minutes = 12/298 [4%]; 30-44 minutes = 15/201 [7.5%]; 45-59 minutes 9/84 [10.7%]; 60-120 minutes 7/48 [14.6%]), cardiopulmonary bypass time, and concurrent descending thoracic aorta repair. The independent determinants for stroke were ($p < 0.05$): history of cerebrovascular disease, previous aortic surgery distal to the left subclavian artery, and cardiopulmonary bypass time. A history of aortic valve incompetence was associated with a lower risk (adjusted odds ratio 0.42, $p=0.015$). The independent determinants for increased risk of early death, which occurred in 66 (10%) of patients, were ($p < 0.05$): increasing age, Mar-fan syndrome, concurrent distal aortic aneurysm, previous ascending aortic surgery, cardiopulmonary bypass time, cardiac complications, renal complications, and stroke. We conclude that deep hypothermia with circulatory arrest is a safe technique for the repair of complex aortic problems provided circulatory arrest time and cardiopulmonary bypass time is not excessive. Furthermore, the determinants of stroke and death are predominantly related to the patients clinical characteristics.

10:00 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

10:45 a.m. SCIENTIFIC SESSIONS - Los Angeles Ballroom

16. Intra-Aortic Balloon Pumping in Cardiac Surgical Patients: Risk Analysis and Long Term Follow-Up

KEITH S. NAUNHEIM, M.D., MARC T. SWARTZ, B.A.*,*

D. GLENNPENN1NGTON, M.D.,

*GEORGE C. KAISER, M.D., LAWRENCE R. McBRIDE, M.D.**

*and ANDREW C. FIORE, M.D.**

St. Louis, Missouri

The intraaortic balloon pump (IAB) is usually the first mechanical device inserted for perioperative cardiac failure, however, little current data is available regarding short and long-term effectiveness. From Jan. 1983 through Nov. 1990, 6,856 adult patients (pts) underwent cardiac surgical procedures, 580 of whom (8.5%) had lABs inserted preoperatively (preop) 107 pts, intraoperatively 419 pts, or postoperatively 54 pts. There were 374 males, 206 females with a mean age of 63.9 yrs (range 19-88). Operations included 336 CABG, 75 mitral, 54 aortic, 15 double valve replacements and 100 other procedures. Operative mortality for IAB pts was 44%. Univariate and multivariate analysis of 26 parameters revealed 6 independent predictors of mortality.

Variable	Univariate p Value	Multivariate p value
Preop NYHA	<0.0001	<0.0001
Transthoracic IAB	<0.0004	<0.0001
Preop IV nitroglycerine	<0.007	<0.001
Patient age	<0.006	<0.001
Female gender	<0.01	<0.001
Preop IAB	<0.013	<0.001

There were 96 (16.5%) lAB-related complications of which 43 required IAB removal for ischemia and 53 required surgical intervention. lAB-related surgical procedures included thrombectomy (34), vascular repair (15), aortic repair (2) and fasciotomy (2). Univariate analysis demonstrated no relationship between any of the lAB-related complications and survival. Only 34 of the 580 pts (6%) were lost to follow-up. There were 75 late deaths, the etiology of which was cardiac in 41 (55%), noncardiac in 20 (27%) and unknown in 14 (19%). Actuarial survival at 1, 5 and 9 years is 51%, 42% and 33%. Of the 217 hospital survivors still alive and contacted, 81% were in NYHA Class I (114) or II (60).

These data show 1) operative mortality for pts requiring IAB in the perioperative period remains high; 2) perioperative risk factors can be identified; 3) IAB complications do not effect survival; 4) operative survivors can achieve prolonged survival with excellent functional results; and 5) consideration for alternative methods of circulatory support is justified.

*By Invitation

17. The Effect of Coronary Reoperation on the Survival of Patients With Stenoses in Saphenous Vein to Coronary Bypass Grafts

BRUCE W. LYTLE, M.D., FLOYD D. LOOP, M.D.,

PAUL C. TAYLOR, M.D., ROBERTONOVOA, M.D.*,*

MARLENE GOORMASTIC, M.P.H. and*

DELOS M, COSGROVE, M.D.

Cleveland, Ohio

Coronary reoperations (reop) have not yet been shown to improve survival. To examine the question of whether coronary reop improves the survival of patients with stenoses in saphenous vein to coronary bypass grafts (SVG), we retrospectively reviewed 1117 patients who had coronary bypass surgery then underwent a postoperative coronary angiogram

(stenotic cath) that documented a stenosis (20-99%) of at least one SVG. Reop within one month of the stenotic cath was performed for 394 patients (reop group) whereas 723 patients (med group) received initial medical treatment (no reop or PTCA within 1 year). Compared with the med group, patients in the reop group were older, more symptomatic, had a higher incidence of left main stenosis and fewer patent bypass grafts (all $p < 0.001$). In-hospital mortality for the reop group was 3.8%. Mean post cath follow-up of the entire group was 80 months.

Based on the interval between the primary operation and the stenotic cath, patients were designated as having early (< 5 yrs.) or late (> 5 yrs.) SVG stenoses. Univariate and multivariate analyses were used to identify factors influencing the survival of these subgroups. Reoperation was not identified as a variable improving the survival of patients with early SVG stenoses.

For patients with late SVG stenoses, moderate or severe impairment of left ventricular function, advanced age, triple-vessel or left main stenosis and stenosis in a SVG to the left anterior descending artery (LAD) (all $p < 0.001$) decreased survival while reoperation ($p = 0.0015$) improved survival. The benefit of reop was very strong for patients with SVG-LAD stenoses with survival of 84% and 74% for the reop group versus 76% and 53% for the med group at 2 and 4 post cath years, respectively ($p = 0.004$). Even for patients with Class I or II symptoms, reoperation prolonged survival ($p = 0.02$ with multivariate testing). This is the first study showing that coronary reoperation improves the survival of any patient subset and documents enhanced survival with reoperation for patients with late SVG stenoses, particularly those with SVG-LAD stenoses.

11:25 p.m. ADDRESS BY HONORED SPEAKER

Coronary Artery Bypass Graft Surgery; Twenty-five Years Later. Some Landmarks

Rene G. Favaloro, M.D., Buenos Aires, Argentina

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

CARDIOTHORACIC RESIDENTS' LUNCHEON - Century Room

*By Invitation

TUESDAY AFTERNOON, APRIL 28, 1992

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - ADULT CARDIAC SURGERY - Los Angeles Ballroom

18. Prolonged Orthotopic Xenoheart Transplantation in Infant Baboons

KAWAUCHI MOTOHIRO, M.D., STEVEN R. GUNDRY, M.D.,*

JAVIER ALONSO de BEGONA, M.D.,*

FRANCOIS BOUCHART, M.D., ANEES J. RAZZOUK, M.D.*,*

NORI FUKUSHIMA, M.D., ARTHUR J. HAUCK, M.D.**

DOUGLAS A. WEEKS, M.D.,*

SANDRA NEHLSEN-CANNARELLA, Ph.D. and*

LEONARD L. BAILEY, M.D.

Lotna Linda, California

The donor pool for infant heart transplantation is severely limited. Many infants die each year awaiting suitable donor hearts. To study the feasibility of an animal donor "bridge" to allotransplantation, we examined Orthotopic concordant xenotransplantation in a juvenile primate model. Eighteen donor rhesus monkeys weighing 2.4-3.8 kg (mean 2.9 kg) were matched with juvenile baboons, aged 9-19 months (mean 12.7 mo) and weighing 3.2-4.8 kg (mean 3.9 kg) using ABH blood type and mixed lymphocyte culture. Rhesus monkey hearts were orthotopically transplanted without immunosuppression into six control baboons (Or. 1). In five baboons (Gr. 2), 4 mg/kg/day of an-tilymphocyte globulin (ALG) was given for 3 days preoperatively and 5 days postoperatively. Splenectomy was also performed, and 18 mg/kg/day of oral FK506 was administered. Intravenous Methotrexate and/or Methylpred-nisolone were used as rescue therapy. Seven baboons (Gr. 3), received the same immunosuppression as Gr. 2, but an intravenous dose of Methotrexate (0.1-5 mg) was given twice weekly to suppress the proliferative response monitored by in vitro immunologic assays.

Baboons in Gr. 1 survived 6,7,8,8,9 and 10 days (mean 8 days); all died from rejection. Baboons in Gr. 2 survived 25,32,53,57 and 75 days (mean 48.4 days) ($p<0.05$ vs Gr. 1). Two died during rescue therapy for rejection and three died from CMV infection. Two baboons in Gr. 2 revealed mild rejection at autopsy. Three baboons in Gr. 3 succumbed at 35,43 and 96 days; one from pulmonary infection, one from CMV pneumonia, and one from renal failure aggravated by Gancyclovir. Only one of the 3 deceased baboons in Gr. 3 showed mild rejection at autopsy. Four baboons in Gr. 3 remain alive and well 43,105,113 and 127 days posttransplantation (mean 80 days).

FK-506 coupled with low-dose maintenance Methotrexate has produced host survival in this xenotransplant model. Results suggest that concordant xenotransplantation would be a suitable biologic bridge to allotransplanta-tion in infant recipients.

*By Invitation

19. Defibrillator Therapy in Ischemic and Non-Ischemic Ventricular Arrhythmias

T. BRUCE FERGUSON, JR., M.D.,*

BRUCE D. LINDSAY, M.D., MICHAEL E. CAIN, M.D.**

and JAMES L. COX, M.D. St. Louis, Missouri

The role of the implantable cardioverter-defibrillator (ICD) is non-ischemic ventricular arrhythmias (tachycardia-VT, fibrillation-VF, and sudden cardiac death-SCD is well established. However, its role in ischemic VT that is refractory to conventional medical therapy is less clear; ablative surgical procedures and amiodarone provide alternative therapies to the ICD for VT. Over the past four years, we have followed the treatment algorithm that 1) ablative surgery for cure of VT is preferable in patients with discrete aneurysms and preserved regional ventricular function; 2) all other patients who are not candidates for direct surgery (e.g., severe global dysfunction) undergo ICD implantation, if VT criteria are met; and 3) due to the side effects of the drug, amiodarone is used only if an ICD is not indicated. Potentially, however, this approach might increase the mortality in the ICD group by selecting out the best VT patients for ablative surgery and by including patients in whom amiodarone should be the preferred treatment. Therefore, our experience with 93 consecutive patients undergoing complete ICD system placement for ischemic (ISCH) and non-ischemic (NI) VT/VF was examined. Data are expressed as mean ± SD; (significance: $p<0.05$, independent Student's t test):

	ISCHEMIC	**NON-ISCHEMIC**	
Patients	50	54	p = NS
Age(yrs); Range	59.6 ± 11.1 (37-80)	53.6 ± 19.0 (10-83)	p = NS
Ejection Fr (%)	25.2 ± 10.8	30.9 ± 13.7	p<0.05
LESD(J);* Range	18.6 ± 5.0 (10-30)	16.2 ± 4.0 (8-28)	p<0.05
Operative Mortality	3/50 (6.0%)	1/43 (2.3%)	p = NS
K-M Survivah:SCD (48m)	88.0	88.4	p = NS
K-M Survivah:Death (48m)	69.8	77.6	p = NS

*(LESD = least energy for 3 successful defibnllations)

Eight total (5 ISCH, 3 NI) patients were transplant candidates. 19 ISCH patients underwent concomitant coronary bypass grafting. All perioperative deaths in the ISCH group were due to myocardial infarction and ventricular failure; the NI death was due to amiodarone toxicity. In all patients with manifestations of amiodarone toxicity, surgical mortality for ICD implantation was 50%. Kaplan-Meier analysis (Mantel-Haenszel) demonstrated that freedom from SCD and any death was not different between the groups.

Therefore, despite worse ventricular function, concomitant coronary disease and higher LESDs, the surgical mortality, long-term survival and freedom from SCD following ICD implantation in ISCH patients is not significantly different from that for ICD patients with NI disease; the somewhat higher mortality in the ISCH group is directly related to the underlying coronary disease and not the VT. ICD therapy in these ischemic patients who do not meet criteria for ablative and potentially curative surgery is comparable to ICD therapy in non-ischemic patients; this ICD patient stratification should become useful when less surgically invasive implant techniques are considered.

*By Invitation

20. Clinic, Hemodynamic and Electrophysioiogic Results of 200 Left Ventricular Patch Reconstructions for Post Infarction Left Ventricular Aneurysm

VINCENT DOR, M.D., MICHEL SABATIER, M.D.,*

FRANCOISE MONTIGLIO, M.D., MOHAMED SAAB, M.D.**

PHILIPPE COSTE, M.D.,*

PHILIPPE ROSSI, M.D. and*

*MARISA DiDONATO, M.D**

Monte Carlo, Monaco

From 1987 to 1991, 200 consecutive pts (181 men and 19 women, mean age 58 ±9 yr), with postinfarction left ventricular (LV) aneurysm (187 anterior and 13 posterior) underwent LV reconstruction (LVR). Surgical technique consisted of endoventricular circular patch plasty with septal exclusion, using a circular patch of synthetic or autologous tissue anchored

inside the LV in the contractile myocardium, after the resection of the endocardial scar and resection or exclusion of the aneurysm.

Methods: all pts underwent complete hemodynamic study (right and left heart catheterization, ventricular angiography and coronary arteriography) before and early after surgery (9-12 days). 115/200 pts underwent programmed right ventricular stimulation (PVS), before and after surgery. At present, clinical follow up is available in 148 of the surviving pts; 60 of them underwent complete hemodynamic study, including PVS, after 1 yr.

Preoperative data: 33 pts were operated in emergency. 145 pts were in NYHA class III-IV (72%); 31/200 pts had spontaneous VT (15%) and 46/115 had inducible VT (40%). Mean EF was 36 ± 14% (EF <40% in 116 pts and <20% in 18 pts) Contractile EF was 44 ± 11%.

Postoperative results: LVR was performed with a synthetic patch in 115 pts and with autologous patch in 85. Non guided subtotal endocardectomy was performed in all pts with spontaneous and inducible VT, 24 of them had associated cryotherapy. Complete myocardial revascularization was performed in 189 pts (95%). Global peroperative mortality was 6.5% (13 pts).

Early control: Mean EF significantly increased (49 ± 13%, $p<0.01$); no pt had EF <20%; 29 pts had EF <40%. When LV function has been evaluated by pressure-volume and pressure length loops, preop abnormal morphology and orientation of the loops tend to normalize after surgery. 42 out of the 44 surviving pts with preop inducible VT had non inducible VT after surgery (95% $p<0.01$); 29 out of the 30 surviving pts with preop spontaneous VT had no spontaneous or provoked arrythmias after surgery. 8 pts without preop inducible VT had postop inducible VT and 1 pt without preop spontaneousVT had postop spontaneous VT.

Late control: In the 60 pts controlled after 1 year, EF was still significantly increased (44 ± 13, $p<.05$). VT was induced in 3 pts and no spontaneous episodes of VT have been recorded. Late mortality (interval 3m-4yr) was 11/148 pts (7.4%); in 6 pts it was not of cardiac origin. 88% of pts were in NYHA class I-II. One pt underwent redux surgery for a false aneurysm after 1 year.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - ADULT CARDIAC SURGERY - Los Angeles Ballroom

21. Aprotinin Partially Inhibits the Contact and Platelet Activation Systems During Extracorporeal Perfusion

YANINA T. WACHTFOGEL, M.D.,*

UMBERTO KUCICH, Ph.D., C. KIRK HACK, M.D.*,*

STEFAN NIEWIAROWSKI, M.D.,*

ROBERT W. COLMAN, M.D. and*

L. HENRY EDMUNDS, JR., M.D.

Philadelphia, Pennsylvania

Aprotinin reduces blood loss after cardiac surgery by inhibiting plasmin and decreasing bleeding times. The mechanism of action of aprotinin on the contact system of plasma proteins, which mediates part of the "whole body inflammatory response", and on platelets is not clearly understood. We investigated the effect of aprotinin at four different doses on the contact activation system, neutrophil degranulation, and platelet release and aggregation during ex vivo extracorporeal circulation.

Fresh heparinized human blood was recirculated at 37 °C for two hours in a membrane oxygenator-roller pump perfusion circuit. Changes in platelet count, leukocyte count, platelet response to ADP, plasma p-thromboglobulin (BTG), kallikrein-C1 inhibitor complex (Kal-C1), C1-C1 inhibitor complex (C1-INH) and neutrophil elastase were measured before and at 5, 30, 60 and 120 minutes of recirculation in 30 studies at 0, 0.015, 0.03, 0.06 and 0.12 mgm/ml aprotinin.

Without aprotinin plateletes decreased to 36 ± 12% of control at 5 min and increased to 56 ±13% at 120 min. Plasma BTG increased progressively to 2.10 mg/ml at 120 min. Aprotinin did not affect these values at a dose of 0.12 mgm/ml but did increase the sensitivity of platelets to ADP. Kal-Cl-INH and C1-C1-INH complexes increased progressively to 0.533 ± 0.135 μ/ml and 2.20 ± 1.17 n/ml respectively at 120 min without aprotinin. Kal-C1-INH complexes were completely inhibited at aprotinin concentrations of 0.03 mg/ml or greater. The increase in C1-C1-INH was not affected by any dose of aprotinin. Release of neutrophil elastase was partially but not completely inhibited at the highest dose of aprotinin and was 50% inhibited at 0.03 mg/ml concentrations.

We conclude that aprotinin in high dose completely inhibits kallikrein-induced activation of neutrophils but does not inhibit the classical complement pathway. Aprotinin does not affect platelet adhesion or release but improves platelet function. Since this system does not generate plasmin, the effect on platelets may be mediated through neutrophil products. The data indicate that aprotinin partially preserves platelet function, totally inhibits contact activation, and partially inhibits neutrophil release, and thus attenuates the whole body inflammatory response.

*By Invitation

22. Venovenous Compares Favorably to Venoarterial Access for Extra-Corporeal Membrane Oxygenation in Neonatal Respiratory Failure

RALPH DELIUS, M.D., ROBERT H. BARTLETT, M.D.,*

HARRY ANDERSON, III, M.D.,*

ROBERT SCHUMACHER, M.D., MICHAEL SHAPIRO, M.D.*,*

TETSURO OTSU, M.D. and JENNIFER HIRSCH**

Ann Arbor, Michigan

Venoarterial ECMO with jugular and carotid cannulation has become standard treatment for severe respiratory failure in the newborn. Venovenous access has theoretical advantages, and is now possible using jugular cannulation with a double lumen catheter. We retrospectively compared 22 VV ECMO patients with 20 patients supported with traditional VA bypass. The two groups of patients were selected to be comparable in terms of diagnosis and severity of respiratory insufficiency. The diagnoses in both groups were limited to meconium aspiration syndrome or persistent pulmonary hypertension of the newborn. The average oxygenation index in both groups were similar (46.6 VV, 47.2 VA, $p<0.05$). Both VV and VA allowed flow rates > 100 ml/kg/min, which was adequate for gas exchange support. One patient required

conversion of VV to VA bypass due to hemodynamic instability. The average time of support was 115 hours (range 24 to 338 hours) for VV ECMO and 134 hours (range 47 to 361 hours) for VA ECMO ($p>0.05$). The time to extubation after decannulation from ECMO was 133 hours (range 38 to 720 hours) for VV and 100 hours (range 27 to 192 hours) for VA ($p>0.05$). One patient supported with VA ECMO had an intracranial hemorrhage. There were no documented neurological injuries in the patients managed with VV ECMO. There were no deaths in either group. Venovenous ECMO through a double lumen cannula provides adequate respiratory support for neonates and pulmonary failure and avoids ligation of the common carotid artery.

*By Invitation

23. Successful Restoration of Cell Mediated Immune Response Following Cardiopulmonary Bypass by Immunomodulation

ANDREAS MARKEWLTZ, M.D., EUGEN FAIST, M.D.*,*

STEPHAN LANG, M.D., STEPHAN ENDRES, M.D.*,*

DIETMAR FUCHS, M.D. and*

*BRUNO REICH ART, M.D.**

Munich, Germany

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

Stanford, California

The most common pathogens found in patients (pts) with septic multi organ failure after cardiopulmonary bypass (CPB) are opportunistic microorganisms indicating a depression of cell mediated immunity (CMI). It was therefore the purpose of our prospective randomized trial to study the immunologic changes due to CPB and the possible influence of im-munomodulation on CMI parameters. **Patients and Methods:** 32 male and 8 female pts with a mean age of 63.3 years undergoing coronary artery bypass grafting (n = 31) or valve replacement (n = 9) were included in the study. 20 pts received conventional therapy (Gr. 1), another 20 pts had additional im-munomodulatory treatment (Gr. 2) with the cyclooxygenase inhibitor in-domethacin (Indo) (3x50 mg i.v. daily for 5 d) and the thymomimetic substance thymopentin (TP-5) (50 mg s.c. 2 hours prior to operation, on d2 and d4). Indo blocks synthesis of prostaglandin E2 which downregulates CMI, TP-5 enhances T-lymphocytic reactivity. Immunologic screening was carried out preop., on dl, d3, and d7. CMI parameters studied included in vitro phenotyping of T-helper (T4) - and T-suppressor (T8) cells, mitogen induced lymphoproliferation (LP) and synthesis of interleukin (IL)-l, IL-2, tumor necrosis factor (TNF), and interferon (IFN)-gamma. Delayed type hypersensitivity (DTH) response to an antigen skin test battery served as in vivo parameter for CMI reactivity.

Results: When compared to preop baseline data on dl, T4/T8 ratio, LP, IL-1, TNF, IL-2, and IFN-gamma synthesis were significantly reduced in controls (Gr. 1). Depression of CMI parameters persisted until d7; in addition DTH response was significantly impaired on d7. In Gr. 2 pts (Indo and TP-5 treatment) only LP were found to be significantly reduced on dl (Gr. 1 vs. Gr. 2: $p< .05$ for T4/T8 ratio, LP, IL-1, IL-2, TNF). On d3 all parameters studied had returned to preop baseline (Gr. 1 vs. G. 2: $p<.05$ for T4/T8 ratio, LP, IL-2, TNF, IFN-gamma) and remained normal until d7 (Gr. 1 vs. Gr. 2: $p<.05$ for T4/T8 ratio, LP, IL-2, IFN-gamma). DTH response showed no change on d7 as compared to preop. (Gr. 1 vs. Gr. 2: $p<.05$). **Conclusions:** 1. CMI response is seriously impaired following CPB. 2. Immunomodulation with Indo and TP-5 can

successfully counteract the depression of CMI; this is - to our knowledge - the first time that a restoration of CMI response could be demonstrated following CPB.

*By Invitation

24. Redo Cardiac Surgery: Late Bleeding Complications From Topical Thrombin Induced Factor V Deficiency

BRIAN L. CMOLIK, M.D., JOEL A. SPERO, M.D.*,*

GEORGE J. MAGOVERN, M.D. and

RICHARD E. CLARK, M.D.

Pittsburgh, Pennsylvania

Bovine thrombin induced Factor V (FV) deficiency was thought to be a very rare acquired coagulopathy with only one case documented in the literature. We report the first series of 7 patients over a 2 year period who developed this unique coagulopathy 1-2 weeks following cardiopulmonary bypass. All patients had normal coagulation profiles preoperatively. The coagulopathy was characterized by a markedly elevated prothrombin time (PT) (26.6-69.9 sec.), an elevated activated partial thromboplastin time (APTT) (68-M80 sec.), a positive lupus anticoagulant study (7/7), and markedly decreased level of Factor V (<="" p="">

Patient Procedure	**Admission PT(sec)**	**Highest PT(sec)**	**Lowest Factor V (% of Normal)**	**Outcome/ Complications**
1 Redo CABG	13.5	69.9	<1	Alive/GI Bleeding
2 Redo CABG	12.7	47.6	<1	Alive
3 Redo MVR	13.6	30.6	6	Death/Bleeding
4 Redo MVR	13.5	64.2	4	Alive/Bleeding
5 AVR (S/P CABG)	11.3	31.2	8	Alive
6 Redo CABG	11.7	26.6	28	Alive
7 Asc. Ao. Repl.	12.9	39.6	2	Alive
MEAN ± -SEM	12.7 ± 0.3	44.2 ± 6.5	7.1 ± 3.6	

The coagulopathy failed to respond to intravenous Vitamin K, fresh frozen plasma or platelet transfusions. Intravenous gamma globulin (IVIG), resulted in transient improvement in abnormal PT, APTT, and FV levels. Four patients never demonstrated clinical evidence of bleeding. Three patients (3/7) developed a significant bleeding diathesis. One patient developed coagulopathic bleeding which contributed directly to death by cardiac arrhythmia. One patient suffered from spontaneous bleeding into surgical wounds which significantly prolonged her hospital stay, and one developed minor gastrointestinal bleeding. Six of seven patients were discharged from the hospital.

Bovine thrombin induced FV deficiency was previously unrecognized. This syndrome should be suspected in patients undergoing redo cardiac surgery who develop marked elevations in their PT 7-10 days after their exposure to bovine thrombin. The resulting coagulopathy has produced bleeding complications and death. Current treatment modalities are ineffective in correcting this coagulopathy, although plasmapheresis may have a role in its management. These observations have resulted in a more selective use of bovine thrombin as a topical hemostatic agent at our institution.

SPECIAL PRESENTATION - Los Angeles Ballroom 75 Years Ago the Incredible Beginnings of Thoracic Surgery and the AATS

Andreas P. Naef, M.D., Pully-Lausanne, Switzerland

4:50 p.m. EXECUTIVE SESSION (Members Only)

7:00 p.m. 75th ANNIVERSARY DINNER/DANCE (Black Tie)

*By Invitation

TUESDAY AFTERNOON, April 28, 1992

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - GENERAL THORACIC SURGERY - Beverly Hills Room

25. Intrathoracic Stomach: Presentation and Results of Operation

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

CLAUDE DESCHAMPS, M.D. and*

PETER C. PAIROLERO, M.D.

Rochester, Minnesota

Between January 1, 1980 and December 31, 1990, 147 patients (96 females and 51 males) were found to have an intrathoracic upside-down stomach. Median age was 69 years (range 34-89). Signs and symptoms occurred in 141 patients (95.9%) and were primarily obstructive. They included postprandial pain in 87 (59.2%) patients, vomiting in 46 (31.3%), and dysphagia in 44 (29.9%). Only 23 (15.6%) presented with reflux. Anemia was present in 31 (21.1%).

One hundred and twenty patients underwent elective surgical repair. The hernia was known to be present for a median of 60 months (range 0-420) prior to operation. A transthoracic uncut Collis-Nissen repair was done in 79 patients (65.8%), a Belsey in 19 (15.8%), a transthoracic Nissen in 12 (10.0%), a transabdominal Nissen in four (3.3%), and other procedures in six (5.0%). Twenty-three patients (19.2%) had complications. There were no operative deaths. Median follow-up of 116 patients (96.7%) was 46 months (range 1-139). Seventy-one patients (61.2%) had excellent results, 38 (32.8%) good, six (5.2%) fair, and one (0.9%) had poor results. Five patients had emergent operations for suspected strangulation with one death. Of the remaining four, only two had excellent results. Twenty-two other patients were followed for a median of 96 months (range 12-268) with medical management. Two developed progressive symptoms and one died following aspiration.

We conclude that patients with an intrathoracic upside-down stomach presenting with obstructive symptoms should be repaired and that elective operation is safe and effective.

*By Invitation

26. Surveillance Endoscopy for Barrett's Esophagus: Does It Help?

JOHN M. STREITZ, JR., M.D.,*

CHARLES W. ANDREWS, JR., M.D. and*

F. HENRY ELLIS, JR., M.D.

Burlington, Massachusetts

Patients with Barrett's esophagus (BE) are at increased risk for the development of adenocarcinoma of the esophagus. Although endoscopic surveillance is commonly practiced, it is not known whether it will improve post-resection survival compared with patients whose carcinoma develops while not under surveillance. This is our initial report of 19 patients in whom either high grade dysplasia or invasive adenocarcinoma developed in a known pre-existing benign BE.

Seventy-nine cases with adenocarcinoma arising in BE were seen at our institution from January 1973 to October 1991, 19 of whom were known previously to have had BE. Surveillance endoscopy was carried out in these 19 patients at intervals ranging from 1 month to 4 years, with a median interval of 6 months. Two patients had a surveillance interval greater than 12 months and presented with advanced tumors 3 and 4 years after their last endoscopy. All patients underwent esophagogastrectomy except one who refused operation when severe dysplasia was diagnosed and who returned 1 year later with an unresectable carcinoma.

Comparison of the pathologic stages of these 19 tumors with those of the 60 patients not under surveillance at the time of discovery showed a significant difference with 11 (58%) vs 9 (15%) in stages 0 and 1, 4 (21%) vs 17 (28%) in stage 2, 4 (21%) vs 30 (50%) in stage 3, and 0 vs 4 (7%) in stage 4 ($p = 0.002$).

Adjusted postoperative survival for 16 patients who underwent endoscopy at least every 12 months and who underwent resection when advised was compared with the group not under surveillance. The difference was significant with survival at 1 year being 93% vs 67%, at 3 years 84% vs 36%, and at 5 years 67% vs 22% (Tarone-Ware $p = 0.02$).

Our data strongly suggest that surveillance endoscopy allows early detection of developing malignancy in BE and will improve long-term postoperative survival.

*By Invitation

27. Epiphrenic Diverticulae, Why is Operation Necessary?

NASSER K. ALTORKI, M.D., DA VID B. SKINNER, M.D.*

and M. SUNAGAWA, M.D.*

New York, New York

Diverticulae of the thoracic esophagus are uncommon disorders. The indications for surgical intervention in asymptomatic or minimally symptomatic patients are unclear. Among 20 patients referred over 20 years there were 6 males and 14 females with a median age of 65 years. Two had had previous diverticulectomies. Dysphagia was present in 9 (45%) and regurgita-tion in 11 (55%). Nine patients had severe nocturnal cough with symptoms of aspiration. In two of these nine and in three other patients (25%) pulmonary symptoms were the only manifestation of disease with no or minimal esophageal symptoms. One patient was misdiagnosed as bronchial asthma for several years, one had massive aspiration prior to a

hernia repair, one developed a bronchoesophageal fistula and lung abscess and 2 had severe persistent cough. All patients had a diagnostic barium esophagogram and en-doscopy. Operation was performed in 17 patients while 3 declined. There was one hospital mortality. Follow-up is complete on 17/19 patients until June 1991. All operative survivors but one are asymptomatic. Of three patients refusing surgery, one died from aspiration pneumonia, another died from a myocardial infarction and one lives with severe dysphagia.

Because of the high incidence of aspiration (45%) and the potential for life threatening pulmonary complications in some patients (15%), we conclude that operative intervention should be undertaken in all patients with thoracic esophageal diverticulae regardless of the presence or absence of symptoms.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION B -GENERAL THORACIC SURGERY - Beverly Hills Room

28. Omentopexy and Early Postoperative Corticosteroid in Clinical Lung Transplantation

JOHN MILLER, M.D.*
Toronto, Ontario

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

Early success in clinical lung transplantation was believed in part due to the technique of bronchial anastomosis, routine bronchial omentopexy and avoidance of early postoperative corticosteroid. The most recent 16 month consecutive experience of two programs with single (SLTx) or bilateral (BLTx) lung transplant was compared to study the current short-term effect of these perioperative strategies. In Center A, of 31 patients undergoing LTx, 29 had telescoped bronchial anastomoses, coverage of the bronchus with local tissue only (no omentopexy), and routine perioperative corticosteroid. In Center B, of 50 patients having LTx, 44 had end-to-end bronchial anastomoses wrapped in omentum and received no routine perioperative corticosteroid. These 29 patients from Center A and 44 patients from Center B comprise the experience described below.

	SLTx	BLTx	Bronchial Anast.			Biopsy Proven	Operative	1 Yr.
			At Risk	Dilatation	Stent	Acute Reject	Mortality	Act. Surv.
Center A								
n = 29	4	25	54	2	1	14	5	81%
Center B								
n = 44	21	23	67	1	-	19	4	82%

In Center A, septic lung disease was the most frequent indication (15 of 29 patients) whereas in Center B obstructive lung disease was the most frequently encountered condition (24 of 44 patients). Sepsis accounted for 3 of 5 early deaths in Center A (all due to resistant pseudomonas cepacia infection in cystic fibrosis recipients) and for 2 of 4 perioperative deaths in Center B (one pseudomonas, one cadida). In Center A, CMV prophylaxis was administered to all patients except negative recipients receiving grafts from negative donors. CMV infection requiring treatment was encountered in 5 of 29 patients at Center A in comparison to 23 of 44 recipients at Center B where only D+ and R- mismatches received prophylaxis. Routine

omentopexy is not required for successful lung transplantation. Early postoperative corticosteroids do not impair airway healing but neither do these agents appear to protect against acute rejection episodes. While routine corticosteroids do not predispose the recipient to CMV infection, their use may increase the likelihood of postoperative bacterial sepsis.

*By Invitation

29. Comparative Outcome of Heart-Lung and Lung Transplantation for Pulmonary Hypertension

ALAIN R. CHAPELIER, M.D.,*

PHILIPPE G. DARTEVELLE, M.D.,*

PASCAL R. VOUHE, M.D., JACQUES CERRINA, M.D.*,*

*FRONCOIS LE ROY LADURIE, M.D.**

*and GERALD SIMONNEAU, M.D.**

Le Plessis Robinson, France

Sponsored by: Jean DesLauriers, M.D., St. Foy, Quebec

While several lung transplantation (LT) procedures have been developed during the recent years, the most accurate for pulmonary hypertension (PHT) remains controversial. Out of a total series of 72 LT performed by the same team since 1986, 27 were for end-stage lung vascular disease (primary PHT, n = 22; chronic pulmonary embolism, n = 3; histiocytosis x, n = 2).

There were three groups according to the transplantation procedure: Group A, with heart-lung transplantation (HLT), 19 patients aged 37 ± 11 years, operated between 1986 and 1991; Group B, with en-bloc double lung transplantation (DLT), 7 patients aged 36 ± 8 years, operated since October 1990; Group C, with single lung transplantation (SLT), 1 patient aged 35 years operated in December 1990. In Group B the airway anastomosis was performed by double proximal bronchial anastomoses which resulted in 2 cases in stenosis corrected with stents; the associated ASD was closed and correction of major pulmonary artery (PA) size discrepancy was achieved using anastomosis of the recipient's left PA to the donor's main PA.

In Group A, the cumulative survival rate is 78% and *53%* at 1 and 4 years respectively (4 post-operative deaths). In Group B, 1 patient died in postoperative course and in Group C, the patient is alive 10 months later. Hemodynamic values are similar in Groups A and B.

	HLT (ht-lung)		DLT (double)		SLT (single)	
	Pre-op.	Post-op.	Pre-op.	Post-op.	Pre-op.	Post-op.
P.A.P.? (mm Hg)	66.2 ± 17.4	20.6 ± 3.3	7 1.4 ± 22.7	19.5 ± 3.3	80	42
C.I. (1/m2)	2.1 ± 0.4	3.3 ± 0.7	1.9 ± 0.3	3.3 ± 0.7	1.9	3
P.V.R. (Wood U.)	27.4 ± 13.6	3.2 ± 1.4	32.4 ± 8.2	3.1 ± 1.6	38.4	8

The respiratory functionn tests are similarly favorable in Groups A and B. Three patients in Group B developed early and reversible left ventricle dysfunction. The SLT patient had difficult hemodynamic post-operative course and six months later is providing high PAP levels (27 mm Hg) and maximum work load of 60 W compared with a mean 120 W in Groups A and B.

Conclusion: 1) En-bloc DLT should be preferred to HLT being followec by immediate right ventricular function recovery and avoided of cardiac specific graft complications despite transient left ventricle dysfunction. 2) DLT should also be preferred to SLT, procedure complicated with a critica post-operative course and with modest functional results.
*By Invitation

30. Obliterative Bronchiolitis After Lung or Heart-Lung Transplantation for Primary Pulmonary Hypertension

SARA J. SHUMWAY, M.D., MARSHALL I. HERTZ, M.D.*,*

JOSE JESSURUN, M.D., RAOUF NAKHLEH, M.D.*,*

MICHAEL PETTY, R.N., B.S.N. and*

R. MORTON BOLMAN, III, M.D.

Minneapolis, Minnesota

Since the advent of heart-lung (HLT) and lung (LT) transplantation at the University of Minnesota in May 1986, 24 HLT, 26 SLT, 1 double lung, and 3 bilateral SLT have been performed. Nine patients underwent HLT and four SLT for primary pulmonary hypertension (PPH). Of these 53 patients, 13 have developed obliterative bronchiolitis for an overall incidence of 24.5%. Among these 13 patients with OB, seven have died, one has undergone successful bilateral SLT after a SLT for PPH, and one died following an unsuccessful attempt at SLT following HLT for PPH. Five are living with OB on medical management; one is awaiting retransplantation and one is considering retransplantation. Of patients transplanted for PPH, 6 of 13 (46%) have developed OB (2 of 4 after SLT, 4 of 9 after HLT). The incidence of OB following transplantation for etiologies other than PPH is 17.5% (7 of 40 patients). Only one of 7 patients (14%) with alpha-1 antitrypsin deficiency developed OB after SLT, and only one of the 9 heart-lung recipients (11%) presenting with Eisenmenger's complex has developed OB. Compared to the incidence of OB following transplantation for other etiologies, the rate of OB after transplantation for PPH does not reach statistical significance, however the trend definitely suggests that patients with PPH may be at increased risk to develop OB after lung or heart-lung transplantation. Furthermore, the disease-free interval before the onset of OB is longer when a HLT is done in contrast to a single lung transplant for PPH (mean of 18 months for 4 patients versus mean of 7 months for 2 patients, respectively). Given this trend toward the increased development of OB following transplantation for PPH, we believe that replacement of both lungs may provide the patient transplanted for PPH a longer disease-free interval from obliterative bronchiolitis than replacement of a single lung.
*By Invitation

31. A Decade of Pediatric Cardiac Transplantation and the Impact of FK506

BARTLEY P. GRIFFITH, M.D., JOHN M. ARMITAGE, M.D.,*

FREDERICK J. FRICKER, M.D.,*

ROBERT L. HARDESTY, M.D. and
*THOMAS E. STARZL, M.D., Ph.D.**

Pittsburgh, Pennsylvania

The application of lung transplant technology to the pediatric population was a natural extension of the success realized in our adult transplant program which began in 1983. Fourteen pediatric patients (age range 3 to 15 years) have undergone heart-lung (8) or double-lung (7) transplantation (1985-1991). The etiology of end stage lung disease in this pediatric group was primary pulmonary hypertension (4), congenital heart disease (4), cystic fibrosis (2), pulmonary arteriovenous malformation (2), graft versus host disease (1) and desquamative interstitial pneumonitis (1). There were 3 perioperative deaths and no late deaths: sepsis (1), cytomegalovirus (CMV) (1) and donor organ dysfunction (1). The overall survival was 78% with follow-up of 6 years (2), 4 years (1), 3 years (1), 2 years (1), 1 year (1), and 6 months (5). Immunosuppression in our first 9 patients was cyclosporine based therapy with azathioprine, steroids and rabbit antithymocyte globulin (RATG). Encouraged by the dramatic ‘immune advantage' and paucity of side effects in the FK506 trial in pediatric cardiac transplantation we have initiated the trial of FK506 with azathioprine and low dose steroids in the pediatric pulmonary transplant recipients. The last 5 lung transplant recipients' immunosuppression has been FK 506 based triple drug therapy without antilymphocyte agents. Additionally one of the recipients was switched from cyclosporine to FK506 due to severe hirsutism and hypertension which developed three months post-transplant. Rejection was diagnosed by pulmonary function tests (PFT), donor specific primed lymphocyte response and lung biopsy. Nine of 11 patients (80%) were treated with intravenous steroids for episodes of acute rejection, however, only 2/11 recipients (18%) had chronic rejection and 1/11 patient (9%) had significant obliterative bron-chiolitis. Lung functions, measured by serial home and hospital based PFT's, have remained good (average FEV1 90%predicted) in all the children. Complications in the group have been few: phrenic paresis 2/11, bronchial stenosis 1/11, and post-transplant lymphoproliferative disease 1/11. Viral infections represented a constant threat to the pediatric lung recipient: CMV (4), Epstein Barr Virus (2), and Coxsackie virus (1). Since 1989 all lung recipients received intravenous ganciclovir for 2-4 weeks post transplant and oralacyclovir for six months. This anti-viral therapy has reduced the incidence of CMV to 1 of the last 7 recipients. Cardiac rejection and graft coronary arteriopathy have not been observed in the heart-lung recipients. Airway management, the diagnosis of rejection, lung biopsy and follow-up have posed unique problems in the pediatric age group. However, pulmonary transplantation holds real hope for children afflicted by both primary and secondary lung disease.

SPECIAL PRESENTATION - Los Angeles Ballroom

75 Years Ago the Incredible Beginnings of Thoracic Surgery and the AATS

Andreas P. Naef, M.D., Pully-Lausanne, Switzerland

4:50 p.m. EXECUTIVE SESSION (Members Only)

7:00 p.m. 75th ANNIVERSARY DINNER/DANCE (Black Tie)

*By Invitation

TUESDAY AFTERNOON, April 28, 1992

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - CONGENITAL HEART DISEASE - Santa Monica Room

32. Surgical Treatment of Coarctation of Aorta in Infants

L. W. ERNEST van HEURN, M.D., MARC R. DeLEVAL, M.D.*,*

JAROSLAV F. STARK, M.D., CM. WONG,*

O.J. SPIEGELHALTER, M.D. and*

MARTIN J. ELLIOTT, M.D.*

London, England

Despite advances in recent years there remains controversy over the appropriate surgical treatment of coarctation in infants. Thus we reviewed our recent experience.

One hundred fifty-one infants less than three months of age underwent repair of coarctation between 1985 and 1990. In 25% there was hypoplasia of the isthmus and in 33% of the transverse arch. Surgical therapy used was subclavian flap angioplasty (SFA) in 15, resection with a traditional end to end anastomosis (E-E) in 43 and resection with an extended end to end anastomosis into the arch (EE-EA) in 77. In 30 the extension was proximal to the origin of the left carotid artery (REE-EA). Other procedures were used in 16.

Mortality (13 early and 12 late deaths) was related to the presence of associated major heart defect, to preoperative resuscitation and to direct postoperative gradient over the arch. The gradient was significantly lower after EE-EA and REE-EA if there was a hypoplastic isthmus and after REE-EA if the transverse arch was hypoplastic. Actuarial freedom from recoarcta-tion at 4 years was 57% (CL 28%-78%) after SFA, 77% (CL 60%-87%) after E-E, 83% (CL 66%-92%) after EE-EA and 96% (CL 77%-100%) after REE-EA.

We conclude that the extended end to end anastomosis and radical end to end anastomosis offer the best prognosis to treat coarctation in infants irrespective of the arch morphology.

*By Invitation

33. Results of Surgical Repair of Supravalvular Aortic Stenosis in Children

JOHN L. MYERS, M.D. and

JOHN A. WALDHAUSEN, M.D.

Hershey, Pennsylvania

From 1975 to 1991 thirteen patients (7 female, 6 male) have undergone surgical correction of supravalvular aortic stenosis. Preoperative catheteriza-tion demonstrated left ventricular to aortic arch gradients of 25 to 110 mmHg (mean 62 mmHg). The patients were operated on at 2 days to 14 years of age (mean 6 yrs). Repair was accomplished by one of two techniques. 1) A bifurcated patch extending from the ascending aorta down into the right and non-coronary sinuses of valsalva (N = 7) or 2) transection of the aorta at the level of stenosis and incising into all three sinuses of valsalva; counter incisions were made in the distal ascending aorta, allowing advancement of these flaps into each sinus of valsalva (N = 6). This "Y" to "V" flap advancement enlarges the supravalvular area with autologous aortic tissue and eliminates the stenosis. The cross-clamp time was 22 to 104 minutes (mean 67 min). The mean reduction in gradient postoperatively was 47 mmHg.

There was one postoperative death (8%) in a two day old infant who had complete relief of her stenosis but died secondary to severe hypertrophic car-diomyopathy. The average postoperative length of stay was seven days. There are no late deaths. One patient had successful reoperation for residual stenosis distal to the aortic patch. Follow-up has been one month to 11.7 years (mean 3.4 yrs). Echocardiograms performed at most recent follow-up demonstrate mild aortic insufficiency (AI) in five patients, and trace or no AI in six patients. Eight patients have a gradient less than 25 mmHg, and three have gradients of 25 to 40 mmHg. All three of these patients had the bifurcated patch technique.

The technique of aortic division and advancing aortic flaps into all three sinuses of valsalva produces a more anatomic repair and has provided good relief of supravalvular aortic stenosis and deserves broader use.

*By Invitation

34. Management of Severe Subaortic Stenosis, Ventricular Septal Defect and Aortic Arch Obstruction in the Neonate

ARA K. PRIDJIAN, M.D., EDWARD L. BOVE, M.D.,*

L. LUANN MINICH, M.D., FLAVIAN M. LUPINETTI, M.D.**

*and A. REBECCA SNIDER, M.D.**

Ann Arbor, Michigan

Neonates with ventricular septal defect (VSD) and aortic arch obstruction frequently have severe subaortic stenosis (SAS) secondary to posterior deviation of the infundibular septum. The optimal method of repair is uncertain and the use of conduits to bypass the obstruction has been recommended because direct relief of the outflow tract obstruction through the standard transaortic approach is difficult. From September '89 to August '91, 6 neonates and VSD, coarctation (n = 4) or interrupted aortic arch (n = 2) and severe SAS underwent repair using a new operative technique. The infundibular septum was partially removed via the right atrium by resecting the superior margin of the VSD up to the level of the aortic annulus. The resulting enlarged VSD was then closed with a patch to widen the subaortic area. In each patient, the aortic arch was repaired by direct anastomosis. The patients ranged in age from 4 to 28 days (median, 11 days) and in weight from 1.3 to 5.4 kg (mean, 3.2 kg). The preoperative ratio of the diameter of the left ventricular outflow tract to the descending aorta in systole was 0.5 ± 0.1 mm (SD) and ranged from 0.42 mm to 0.65 mm. All patients survived operation with one late death 3 months after operation from noncardiac causes. The survivors remain well from 1 to 12 months following repair (mean, 6 months). All are in sinus rhythm and none has an important residual arch gradient or VSD. No patient has residual SAS although one has significant valvar aortic stenosis (Doppler gradient, 85 mmHg). Mild aortic regurgitation is present in one patient. This series suggests that in neonates with VSD and severe SAS secondary to posterior deviation of the infundibular septum, direct relief can be satisfactorily accomplished from a right atrial approach. This method provides effective widening of the left ventricular outflow tract and is superior to palliative techniques or conduit procedures.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - CONGENITAL HEART DISEASE - Santa Monica Room

35. Neonatal Repair of Truncus Arteriosus

ALDO R. CASTANEDA, M.D., FRANK L. HANLEY, M.D.,*

JOHNE. MAYER, JR., M.D. and

RICHARD A. JONAS, M.D.

Boston, Massachusetts

Between 1/87 and 10/91 patients with truncus arteriosus were surgically managed. 57 of 60 patients (95%) underwent primary repair and 44 of 60 (73%) were less than 3 months of age at repair. Among these 44 patients there has been a recent trend towards earlier repair:

	# Cases	Age (days)	Hospital Deaths
1987	5	49	0
1988	5	47	2
1989	12	40	2
1990	11	29	3
1991	11	12	1

Three of 6 patients (50%) with associated interrupted aortic arch and 4 of 8 patients (50%) with significant truncal valve dysfunction died. These lesions therefore were associated with 7 of 8 (88%) of the hospital deaths. Other important associated lesions, including branch pulmonary artery stenosis or anomalous origin, multiple VSDs, anomalous pulmonary venous return, and coronary artery anomalies were present in 9 additional patients and were not associated with hospital mortality. Over the past two years, in association with early neonatal repair, there have been no cases of post operative pulmonary hypertensive crisis and mean pulmonary artery/right ventricular pressure has been uniformly below 30% systemic pressure. Medium term follow-up (2 mo - 24 mo) was available in 22 of 36 hospital survivors and revealed no further mortality.

Conclusions: Interrupted aortic arch and truncal valve dysfunction remain important risk factors for surgical death. In the absence of these two associated lesions, truncus arteriosus can be repaired with excellent surgical outcome in the early neonatal period (29/30 survivors, 97%). Repair in the early neonatal period appears to reduce the incidence of post operative pulmonary hypertension and pulmonary vascular liability.

*By Invitation

36. Results of a Protocol of Early Repair for Truncus Arteriosus

EDWARD L. BOVE, M.D., FLAVIAN M. LUPINETTI, M.D.,*

ARA K. PRIDJIAN, M.D., JON N. MELIONES, M.D.**,

LOUISE B. CALLOW, R.N. and*

*A. REBECCA SNIDER, M.D.**

Ann Arbor, Michigan

Although the mortality for repair of truncus arteriosus has decreased, routine repair in the neonate has not been widely adopted. Since 1986, we have followed a protocol of early repair, generally within the first month of life. From 1/86 to 10/91, 37 patients underwent repair of truncus arteriosus. Ages ranged from 1 day to 7 months (median, 13 days) and weights from 1.9 to 5.4 kg (mean, 3.2 kg). Repair was done beyond 30 days of age in only 7 patients due to

late referral (6) or noncardiac problems (1). Associated lesions were: interrupted aortic arch (n = 4), nonconfluent pulmonary arteries (n = 3), infracardiac TAPVR (n = l) and hypoplastic pulmonary arteries (n = 3). Truncal valve regurgitation was absent in 18 patients, mild to moderate in 14 and severe in the remaining 5 patients. Truncal valve replacement was performed in the 5 patients with severe regurgitation, 3 of whom also had transvalvar gradients exceeding 30 mmHg. The truncal valve was replaced with a mechanical prosthesis in 2 patients and with a homograft in 3. Right ventricle to pulmonary artery reconstruction was performed with homograft conduits in 32 patients (range, 8 to 15 mm), valved porcine conduits in 4 (range, 12 to 14 mm) and with a nonvalved gortex tube in 1 patient (10 mm). Actuarial survival at 1, 6 and 12 months was 84%, 78% and 78%, respectively. There was 1 death among the 7 patients with interrupted arch or nonconfluent pulmonary arteries. Late death occurred in 3 patients (one noncardiac) with no additional mortality after the fourth postoperative month. Reoperations were required in 4 patients for conduit obstruction (3 months to 3.5 years after initial repair) and in 2 for residual VSD. Despite the high incidence of major associated anomalies, early repair has resulted in excellent survival. We continue to recommend repair promptly after presentation, optimally within the first month of life.

*By Invitation

37. Anatomical Subtype of Hypoplastic Left Heart Syndrome Influences Survival After Palliative Reconstruction

RICHARD A. JONAS, M.D., DOLLYHANSEN, M.D.,*

NANCY COOK, Ph.D. and DAVID WESSEL, M.D.**

Boston, Massachusetts

The mortality of the reconstructive surgical approach to hypoplastic left heart syndrome (HLHS) has remained high in many centers, including those consistently achieving extremely low mortality rates for virtually all other congenital heart anomalies. Accordingly, interest in neonatal heart transplantation for HLHS has strengthened though widespread application is limited by donor supply. We conducted a retrospective study of 78 consecutive patients undergoing palliative reconstructive surgery for HLHS between 1983 and 1991 to identify predictors of mortality which might enable more appropriate triage of patients to either reconstruction or transplantation. Of the 78 patients, 29 had aortic atresia, mitral atresia (AA,MA) (37%), 18 had aortic stenosis and mitral stenosis (AS,MS) (23%), 20 had aortic atresia and mitral stenosis (AA,MS) (26%), and 11 had other variants (14%). Aortic reconstruction was with a homograft gusset in 28 (36%), a homograft tube graft in 9 (12%), a synthetic tube in 29 (37%) and other forms of reconstruction in 12 (15%). A right sided Blalock shunt was used in 68 (87%) and a central shunt in 10 (13%). One patient was lost to follow-up to August, 1991. There were 29 hospital deaths (37%). The product-limit (PL) survival estimate among hospital survivors was 25% at five years. Analysis of hospital deaths revealed no strong predictors of hospital mortality though there was a suggestion that subgroup AA,MS may be at greater risk of hospital death irrespective of surgical procedure (p=0.06). Age at surgery, ascending aortic diameter, shunt type, method of aortic reconstruction and year of surgery did not influence outcome. Anatomical subtype was a predictor of late survival with a PL survival estimate at 3 years of 11 % in the AA,MA group with a trend to worse survival with a very small ascending aorta, of 25% in the AA,MS subgroup and of 76% in the AS,MS subgroup (logrank p = 0.03). This also held true (p = 0.05) when analysis was undertaken according to survival to next procedure following neonatal palliation, thereby eliminating the mortality of the Fontan operation which was evolving in this time frame.

We conclude that patients with AA.MA in particular those with an ascending aortic dimension of less than 2 mm, may be better served by heart transplantation than a reconstructive approach. The longterm outlook for the subgroup with aortic stenosis and mitral stenosis, however, would appear to support a reconstructive approach for this subgroup. We speculate that there are inherent limitations of the current neonatal procedure reflected in a high uniform early mortality over time and between institutions which should be addressed by innovative surgical or ancillary procedures.

*By Invitation

38. High Dose Steroids Prevent Placenta! Dysfunction After Fetal Cardiac Bypass

JOSEPH F. SABIK, M.D., FRANK L. HANLEY, M.D.*,*

MARKUS K. HEINEMANN, M.D. and*

*RENATO S. ASSAD, M.D.**

Boston, Massachusetts

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

Boston, Massachusetts

Surgical treatment of certain congenital heart lesions in utero may have therapeutic advantage over post natal repair or palliation. To perform fetal heart surgery a method to support the fetal circulation will need to be developed. Early experimental attempts at fetal cardiac bypass were unsuccessful secondary to increased placental vascular resistance during and after fetal cardiac bypass. This increase in placental vascular resistance led to decreased placental blood flow, fetal asphyxia and death. Our laboratory has demonstrated that the administration of indomethacin (a cyclooxygenase inhibitor) during fetal cardiac bypass prevents this increase in placental vascular resistance during and after fetal cardiac bypass. The mechanism by which indomethacin achieves this effect is either by inhibiting the production of a placental vasoconstrictive prostaglandin, or by diverting substrate from the cyclooxygenase pathway to the lipooxygenase pathway, thereby increasing production of a placental vasodilating leukotriene. To determine which of these mechanisms is responsible for preventing the increase in placental vascular resistance after fetal cardiac bypass, we inhibited both prostaglandin and leukotriene synthesis at the phospholipase stage with high dose steroids. Fourteen fetal lambs were used in the study. Six animals received indomethacin (3 mg/kg), four animals received high dose steroids (solumedrol 50 mg/kg), and four animals were used as controls. Observations were made during a one hour pre-bypass period, a thirty minute bypass period, and a two hour post bypass period. Placental blood flow and placental vascular resistance were calculated at four times during the experiments: Pre-Sternotomy; Post-Sternotomy; during bypass at thirty minutes; and thirty minutes after cessation of bypass.

Similar to indomethacin, high dose steroid administration during fetal cardiac bypass, prevents the rise in placenta! vascular resistance and preserves placental blood flow during and after fetal cardiac bypass. This study clearly demonstrates that the production of a placental

vasoconstrictive prostaglan-din is responsible for the increase in placental vascular resistance and decrease in placental blood flow observed after fetal cardiac bypass.

SPECIAL PRESENTATION - Los Angeles Ballroom

75 Years Ago the Incredible Beginnings of Thoracic Surgery and the AATS

Andreas P. Naef, M.D., Pully-Lausanne, Switzerland

4:50 p.m. EXECUTIVE SESSION (Members Only)

7:00 p.m. 75th ANNIVERSARY DINNER/DANCE (Black Tie)

*By Invitation

WEDNESDAY MORNING, APRIL 29, 1992

7:30 a.m. FORUM SESSION II - General Thoracic Surgery

Los Angeles Ballroom

F11. Improved Ultrastructural Lung Preservation With Prostaglandin E1 As Donor Pre-Treatment in a Primate Heart Lung Transplant Model

ROBERT S. D. HIGGINS, M.D., GEORGE V. LETSOU, M.D.*,*

JUAN SANCHEZ, M.D., RICHARD EISEN, M.D.*,*

KENNETH L. FRANCO, M.D.,*

GRAEME L. HAMMOND, M.D. and

JOHN C. BALDWIN, M.D.

New Haven, Connecticut

Donor pre-treatment utilizing Prostaglandin E_1 (PGE1) as a pulmonary vasodilator has developed as a simple, effective means to provide excellent preservation in heart-lung transplantation. This study was undertaken to investigate the Ultrastructural preservation of the lung using prostaglandin El and other pulmonary vasodilators in a primate heart-lung transplantation model.

Methods: Heart-lung transplantation was performed in 14 African green monkeys. Donor cardiac preservation was achieved with cold crystalloid car-dioplegic solution (10 ml/kg). Lung preservation was achieved with cold, modified Euro-Collins solution (8meq MgSO4 + 65 cc 50% Dextrose added to standard solution) delivered into the main pulmonary artery (60ml/kg/ total). Vasodilator agents were administered systemically 15 minutes prior to aortic cross-clamping. Central venous, femoral and pulmonary arterial pressures were monitored during infusion. The heart-lung grafts were stored at 4°C for 6 hrs. Three groups of animals were studied: 5 donors with PGE1 (0.1 to 4.0 meg/kg/min), 5 donors with prostacyclin (0.1 to 0.35 meg/kg/min), and 4 donors with nitroprusside (0.8 to 5.0 mcg/kg/min). After transplantation, arterial blood gases and lung biopsies were obtained at 1-3 hrs. and at conclusion of the study. Five formalin blocks per specimen were sectioned for hematoxylin and cosin staining. Cellular architecture and endothelial cell

swelling were evaluated using electron microscopy. The specimens were graded for alveolar hemorrhage, endothelial cell swelling (grade 1 = minimal to grade 3 = severe) and preservation of cellular architecture and a mean score was obtained for each preservation agent.

Results: Endothelial cell swelling correlated with the degree of cellular preservation. PGEi specimens demonstrated the least amount of endothelial swelling (mean score .8) compared to PCI and NTP specimens (mean score 1.4 and 2.7 respectively). All NTP specimens demonstrated moderate to severe endothelial cell swelling. Interstitial and alveolar hemorrhage was noted in poorly preserved specimens, but there were no significant differences between groups.

Conclusions: Prostaglandin El provides improved cellular preservation by decreasing the extent of endothelial cell swelling on electron microscopy.

*By Invitation

F12. The Effect of Corticosteroids on Bronchial Blood Flow After Lung Transplantation

KENJI INUI, M.D., H. J. SCHAFERS, M.D.*,*

VERA BECKER, M.D. BIRTE ONGSIEK, M.D.*,*

AXEL HAVERICH, M.D. and HANS G. BORST, M.D.*

Hannover, Germany

The prophylactic administration of corticosteroids has been discussed controversially in clinical lung transplantation for fear of an increased prevalence of bronchial complications. In an experimental investigation, the effect of prednisolone on bronchial blood flow was investigated, using a model of modified unilateral lung transplantation in pigs. The bronchial anastomosis was created between the donor trachea and recipient left main bronchus. Immunosuppression consisted of cyclosporine (15 mg/kg/d) and azathioprine (2 mg/kg/d) in group I (n = 6); in group II (n = 6), prednisolone (1 mg/kg/d) was added. The animals were studied on the 7th postoperative day. Bronchial blood flow was estimated using laser doppler velocimetry (LDV) and radioisotopic studies (RI). Segments of the graft airway and parenchyma were assessed for signs of ischemia or rejection.

In group I, bronchial blood flow (in percent of reference flow) at the graft main carina was markedly reduced (LDV: 34.9 ± 5.8%; RI 33.3 ± 19.8%). By comparison, blood flow in group II was significantly improved (LDV: 55.3 ± 3.9%, $p<0.005$; RI: 57.1 ± 18.3%, $p<0.05$). Histologic classification of acute pulmonary rejection resulted in similar scores in both groups. In group I, necrotic changes at the graft main carina were observed in 4 of 6 cases with extension into the muscular and cartilagenous portions of the airway wall. By comparison, only limited loss of epithelium with preservation of the deeper layers of the airway were seen in all cases of group II.

Addition of prednisolone to immunosuppression with azathioprine and cyclosporine results in decreased ischemia and significantly improves bronchial blood flow after lung transplantation. Prophylactic administration of corticosteroids is expected to alleviate bronchial ischemia in clinical lung transplantation and thus reduce the incidence of postoperative airway complications. This is confirmed by our clinical experience with 46 lui.£ transplantation.

*By Invitation

F13. Re-establishment of Lymphatic Drainage After Lung Transplantation in Canine Model

RENATO RUGGIERO, M.D., JAROSLAW MUZ, M.D.*,*

ROBERTFIETSAM, M.D.,*

GREGORY A. THOMAS, M.D., ROBERT J. WELSH, M.D.*,*

JAMES E. MILLER, M.D.,*

LARRY W. STEPHENSON, M.D. and

*FRANK A. BACIEWICZ, JR., M.D**

Detroit, Michigan

The lymphatic channels between the lung and the mediastinum are interrupted during lung transplantation. Although clinical and radiologic impressions suggest that lymphatic connections are re-established in the postoperative period, this has not been proven. The purpose of this investigation is to document lymphatic regeneration after lung transplant. A first group of 6 control dogs had transthoracic injection in the periphery of the left upper and lower lobes of 0.2 ml of antimony sulphide colloid (ASC) tagged with Technetium-99m (Tc-99m). ASC is a particle 2-15 nm in size which is absorbed only through lymphatic channels and concentrated in the tributary lymph nodes. 24 hours after injection the animals underwent scintigraphic studies and two-dimensional images of the lung and mediastinum. A second group of 4 dogs underwent division and reanastomosis of the left main bronchus. The left pulmonary artery and veins were dissected from all surrounding tissue. On the second postoperative day and then weekly for 4 weeks, the animals underwent Tc-99m-ASC injection and were studied 24 hours later as above. A third group of 4 dogs underwent left lung allotransplant using standard techniques including immunosuppression with CSA 50 mg/kg/day and AZA 1 mg/kg/day. The animals were studied with Tc-99m-ASC injection and lung lymphoscintigraphy every week for 6 weeks. All control dogs demonstrated mediastinal lymph nodes at 24 hours. 3 of 4 dogs in the reimplantation group showed mediastinal lymph nodes in all studies after 8 days. In the transplant group, mediastinal nodes were visualized for the first time 2 to 4 weeks after the surgery, and at every study from then on. In conclusion, lung lymphoscintigraphy with transthoracic injection of Tc-99m-ASC is a reliable technique for the study of pulmonary lymphatic flow. This experiment conclusively shows for the first time that lymphatic drainage after lung transplantation is re-established beginning as early as the second postoperative week.

*By Invitation

F14. Growth Potential of Reduced Size Mature Pulmonary Lobar Transplants

JOHN A. KERN, M.D., IRVING L. KRON, M.D.,*

CURTIS G. TRIBBLE, M.D., TERRY L. FLANAGAN, MPH*,*

BARRYB.K. CHAN, M.D. and*

WALTER W. SCOTT, B.S.*
Charlottesville, Virginia

The use of mature pulmonary lobes for pediatric lung transplantation has recently been described and represents a potential avenue for solving the severe pediatric donor lung shortage. To provide adequate long term function, however, transplanted mature lobes need to grow in proportion to the recipient. The total number of alveoli in the human lung reaches adult levels by 8-10 years and in the pig by 12 weeks. Whether or not an already mature lobe can grow by forming new alveolar units, following transplantation into a developing recipient, is not known. To study the growth potential of mature lobar transplants, we measured functional residual capacity (FRC), fixed lung volume, alveolar airspace percent, alveolar size, and total number of alveoli in isolated mature left lower lobe lung transplants 12 weeks following implantation into growing piglets. Comparisons were made to age matched control left lower lobes (studied immediately after a left upper lobectomy) to determine if functional or morphologic growth had occurred. The transplanted lobes, and lobes which served as controls were all explanted from 6 month old animals. Helium dilution was used to determine FRC and standard morphologic techniques utilizing the point counting method were used for determination of alveolar airspace percent, and alveolar numbers. Recipients of the transplanted lobes were 9 weeks old and weighed 20 ± 1 kilograms initially. By the end of the 12 week holding period, the animals had increased their body weight by approximately 4 times (88 ±2 kg). Results: values are means ± SEM:

	FRC (cc)	Fixed LungVolume (cc)	Alveolar AirspacePercent	Alveolar Size (Diameter in um)	Alveolar Number ($x10^6$)
Lobe					
Transplanted (n = 5)	579 ± 42	914 ± 50	72 ± 3	72 ± 3	112 ± 11
Control (n = 6)	570 ± 89	685 ± 35*	74 ± 2	71 ± 2	96 ± 9

*p<0.05 by unpaired t-test

No statistically significant differences were seen in FRC or morphologic analysis of alveoli in the transplanted versus control lobes. **Conclusions:** Transplantation of mature lobes into growing animals did result in significant growth of the lobes as determined by fixed volume, though not an increase in alveolar number of functional capacity. Reduced size mature lobar transplantation results in compensatory lobar growth which is not due to a statistically significant increase in functional gas exchanging units.

*By Invitation

F15. Allograft Replacement of the Trachea: Experimental Synchronous Revascularisation of Composite Thyro-Tracheal Transplantation

JOSEPH F. KHALIL-MARZOUK, M.D.,*

PETER COLOSTRAW, FRCS and*

*MERLY GRIFFITHS, MRCPath**

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

The purpose of this study is to assess the structural integrity of the transplanted trachea following composite Thyro-Tracheal allograft replacement in dogs, utilizing microvascular anastomoses based on the blood supply of the related thyroid gland.

Twelve-ring segments of the cervical trachea and the thyroid gland were resected in 18 Beagle dogs. They were grouped as follows: 6 dogs underwent non-vascularised transplants (Group A), 12 dogs received revascularised Thyro-Tracheal composite allografts establishing the anastomoses of the cranial thyroid arteries to the ipsilateral common carotid arteries, 6 dogs did not receive any immunosuppressive agents (Group B) and 6 dogs were given Cyclosporin A in the dose of 25 mg/kg body weight (Group C). Transplant operations were performed in pairs of dogs in the form of double swap procedures, half were implanted in a fresh state in all 3 groups and half were preserved in cold Hartman's solution at 4°C for a period averaging 3 hours.

ALL animals survived the operation, they were sacrificed at intervals (at Humane end-point) between 3 and 28 days. Gross anatomy and light microscopic studies were performed in all cases. Group A demonstrated total loss of structural integrity of the tracheal cartilages and soft tissues as early as 3 days in all animals. Group B showed adequate preservation of viability of tracheal cartilages but necrosis presumed rejection of the soft tissues. Group C showed preservation of tracheal cartilages and soft tissues in all but one case, there was no evidence of cartilage necrosis in the form of empty lacunae, the tracheal muscles and the thyroid gland remained histologically intact, however the lining mucous membrane was colonised by microorganisms and was partially disrupted but there was evidence of early epithelial regeneration at the tracheal anastomoses.

We concluded that revascularisation of the transplanted trachea using the thyroid arteries does maintain the vascularity and hence viability of the tracheal cartilages enabling a reliable substitute for long segment tracheal resections. We predict that the clinical success of this technique will solve a major problem in surgery of the airways.

*By Invitation

F16. Lazaroid U74500A as an Additive to UW Solution for Pulmonary Grafts in the Rat Transplant Model

RYO AEBA, M.D., WILLIAM A. KILLINGER, M.D.*,*

ROBERT J. KEENAN, M.D,, SAMUEL A. YOUSEM, M.D.*,*

ROBERT L. HARDESTY, M.D. and

HARTLEY P. GRIFFITH, M.D.

Pittsburgh, Pennsylvania

Lazaroids, a class of novel 21 amino-steroids, have been reported to be potent inhibitors of iron-dependent lipid peroxidation, which is a major contributing factor to ischemia-reperfusion injury in lung. The aim of this study was to determine the preservation effect of a lazaroid U74500A for pulmonary grafts.

The pulmonary arteries of Lewis rats were flushed with either cold (0 °C) standard University of Wisconsin solution (UW) or UW with 30μM of U74500A substituted for the dexamethasone (UW-L) and then the heart-lungs blocks were stored in the same solution. After 6 or 12 hours of cold storage at 0 °C the left lungs were orthotopically transplanted into isogeneic recipient rats and reperfused for 1 hour. Pulmonary function was assessed by measuring oxygen (PaO_2) and carbon dioxide ($PaCO_2$) tensions in arterial blood on 100% oxygen after removal of the right lung. Tissue

lipid peroxide (LP) levels in the transplanted lungs were measured as a thiobarbiturate-acid reactive substances. There were 6-8 animals in each group. The results were expressed as mean ± SE.

	UW (6 hours)	UW-L (6 hours)	UW (12 hours)	UW-L (12 hours)
PaO_2 (mmHg)	309 ± 81	436 ± 30	27 ± 3	339 ± 70*
$PaCO_2$ (mmHg)	28.2 ± 2.3	24.4 ± 3.8	47. 7 ± 7.0	24.3 ± 2.7**
LP (μmol/g)	.88 ± .07	.54 ± .07*	1.30 ± .09	.69 ± .07**

*:p<0.01 **:p<0.001 vs UW

We conclude that U74500A in the flush and storage solution enhances the preservation of the pulmonary graft in this model.

*By Invitation

F17. NR-LU-10 Monoclonal Antibody Scanning: A Helpful New Adjunct to CT in Evaluating Non-small Cell Lung Cancer

HOMER MACAPINLAC, M.D., VALERIE W. RUSCH, M.D.*,*

ROBERT HEEL AN, M.D., ELISSA KRAMER, M.D.*,*

STEVEN LARSON, M.D. and*

ROBERT J. GINSBERG, M.D.

New York, New York

CT scanning has improved noninvasive staging of lung cancer patients, but has the deficiency of not distinguishing benign from malignant lesions. This prospective trial evaluated the usefulness of a new monoclonal antibody (MoAb) imaging technique as an adjunct to CT by assessing (1) its clinical applicability; (2) its accuracy in detecting malignancy in primary lung tumors and mediastinal nodes. The MoAb, NR-LU-10 (NeoRx corporation), is a murine IgG2b Ab labeled with Tc-99m which recognizes a 40 kD glycopro-tein expressed in non-small cell lung cancers (NSCLC). **Methods:** (1) patients (pts) with potentially resectable NSCLC were eligible; (2) all pts had contrast-enchanced chest CT scans; (3) 25-30 mCi of MoAb were infused I.V. followed by whole body and SPECT imaging 14-17 hours later; (4) subsequent mediatinoscopy or thoracotomy with complete mediastinal nodal mapping provided pathologic correlation.

Results: 24 pts entered. Men:Women = 14:10. No allergic reactions or other side effects of MoAb were seen. Interference from prior V/Q scan precluded adequate imaging in 1 pt, but high quality MoAb images were obtained in the other 23 pts (96%). There was no background activity in lung, and little in the mediastinum. Pathologic correlation:

	MoAb Uptakein 1° **tumor**	Mediastinal **MoAb**	Nodal Involvement**CT**
True(+)	22	8	9

True(-)	1*	7	7
False (+)	0	6	6
False (-)	0	1	1

* = 1 ° lung lesion subsequently proven to be inflammatory

Conclusions: (1) NR-LU-10 MoAb scanning is safe, is easily performed, and produces high quality images of lung and mediastinum; (2) it is very accurate in detecting 1 ° NSCLC; (3) it is as accurate as CT in assessing involvement of mediastinal nodes. NR-LU-10 MoAb holds promise as an adjunct to CT by distinguishing benign from malignant lung lesions.

*By Invitation

F18. The Prognostic Significance of Flow Cytometry in Lung Cancer

THOMAS W. RICE, M.D., THOMAS W. BAUER, M.D.*,*

GORDON N. GEPHARDT, M.D.,*

SHARON V. MEDENDORP, MPH,*

DENISE A. McLAIN, B.S. and*

THOMAS J. KIRBY, M.D.*
Cleveland, Ohio

Sponsored by: Delos M. Cosgrove, M.D., Cleveland, Ohio

The prognostic significance of stage in lung cancer is well known but the significance of other factors, including DNA ploidy analysis, is unclear. To clarify the value of DNA ploidy analysis, we prospectively studied single parameter flow cytometry of fresh tissue from 278 patients with resected primary lung cancer from whom adequate tissue was present. These results as well as age, sex, cell type, histologic grade, and AJCC stage were correlated with survival.

The mean age of the patients was 65.4 years; 65% were male. Cell types were: adenocarcinoma 107 (38.6%); squamous cell 100 (36%); large cell 56 (20%); adenosquamous 8 (3%); small cell 6 (2%); and giant cell (0.4%). Histologic grades were: I (well differentiated) 15 (5%); II100 (36%); and III 163 (59%). AJCC stages were: I 154 (56%); II 39 (14%); III 76 (27%); and IV 9 (3%). Mean follow-up was 18.9 months (range <1-59). One hundred eighty-one patients (65%) were alive at last follow-up. Survival at one year was 75% (±3%) and at three years survival was 53% (±6%).

Each flow cytometry histogram was classified as follows: 1) no detectable aneuploidy 48 (17%); 2) hyperdiploid 152 (55%); 3) hypodiploid 4 (1%); 4) hypertetraploid 16 (6%); 5) multiple aneuploid populations 49 (18%); and 6) multiple aneuploid populations with one hypertetraploid population 9 (3%). For tumors with no detectable aneuploidy, mean S-phase was 4% (range 1-12%) and mean S + G2M phase was 9% (range 0.3-34%).

Multivariate analyses using the Cox Proportional Hazards Model for survival showed that 1) increasing AJCC stage ($p<0.001$) and male sex ($p = 0.007$) were the only factors of independent (negative) prognostic significance and 2) neither the presence nor absence of DNA aneuploidy ($p = 0.71$), the classification of DNA histogram ($p = 0.81$), nor the results of cell cycle analysis (S phase:$p = 0.83$ and S + G2M:$p = 0.47$) showed significant correlation with survival in this group of patients.

*By Invitation

F19. Assessment of Impaired Diaphragmatic Function After Thoracotomy

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MARIE-DOMINQUE FRATACCI, M.D.,*

WILLIAM K. KIMBALL, M.D. and M. ZAPOL, M.D.**

Boston, Massachusetts

Sponsored by: Hermes C. Grillo, M.D.,

Boston, Massachusetts

Experimental and clinical studies have suggested that diaphragmatic function is impaired after pulmonary resection. Previous clinical studies have assessed diaphragmatic function indirectly. However, direct, reliable diaphragmatic length measurements can be obtained with implantable sonomicrometry crystals. To directly assess diaphragmatic function in humans, sonomicrometry crystals were implanted in the costal diaphragm after thoracotomy and pulmonary resection in 6 patients (4 right thoracotomy, 2 left thoracotomy). Following placement of an esophageal balloon catheter and recovery from anesthesia, measurements of resting diaphragmatic muscle length and percent shortening, changes in trans-diaphragmatic pressure (Pdi) and in tidal volume (Vt) were made during mechanical ventilation and during spontaneous respiration before and after a dose of epidural anesthetic (BEp, PEp, respectively).

	mechanical	**spontaneous**	
		BEp	**PEp**
resting length	16.3 ± 2.2	15.0 ± 1.9	15.5 ± 1.8
% shortening	7.9 + 3.0	1.6 ± 1.6*	-.9 ± 2.6
Pdi	-5.4 ± 1.6	7.7 ± 1.5**	11.5 ± 1.9#
Vt	458 ± 102	390 ± 78	555 ± *ISttt*

*$p < 0.05$ versus mechanical, **$p < 0.01$ versus mechanical

#$p < 0.05$ versus BEp, ##$p < 0.01$ versus BEp

Conclusions: Diaphragmatic shortening is significantly impaired during spontaneous respiration post-thoracotomy. Although epidural anesthesia significantly improves spontaneous tidal volume, this is **not** due to increased diaphragmatic shortening.

*By Invitation

F20. Barrett's Esophagus - A Functional Foregut Disorder?

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*HUBERT J. STEIN, M.D.**

Los Angeles, California

Understanding the pathophysiology of Barrett's esophagus will provide better use of subject therapy to treat this complicated reflux problem. To understand the development of Barrett's esophagus in patients with gastroesophageal reflux disease we compared symptoms

(severity score 0-3), the gastric secretory state, esophageal acid exposure (pH < 4, < 3, and < 2), lower esophageal sphincter (LES) function, and circadian esophageal motor activity in 15 patients with Barrett's esophagus to 24 patients with esophagitis, and 25 normal volunteers.

Results: Compared to patients with esophagitis, patients with Barrett's esophagus had less heartburn and regurgitation, but an increased frequency and duration of reflux episodes (pH < 4), and % time pH < 4, pH < 3, and pH < 2 in the esophagus (table). This was associated with an increased basal and maximum gastric acid output (BAO and MAO), decreased LES pressures, and increased frequency of ineffective contractions in the distal esophagus (i.e., isolated contractions or contractions < 30 mmHg) on 24-hour ambulatory esophageal motility monitoring (table).

Table:	**Normals**	**Esophagitis**	**Barrett's**
Heartburn	0 ± 0	2.3 ± 0.2	1.4 ± 0.3*
Regurgitation	0 ±	2.2 ± 0.3	1.6 ± 0.2*
# reflux episodes	21.2 ± 1.9	72.5 ± 6.9	110.2 ± 12.4
# reflux episodes >5 minutes	1.3 ± 0.2	8.1 ± 1.2	15.9 ± 2.2*
%time pH < 4	2.5 ± 0.6	10.3 ± 2.9	24.5 ± 4.8*
% time pH < 3	1.2 ± 0.2	3.4 ± 0.4	10.2 ± 1.9*
%time pH < 2	0.4 ± 0.2	2.1 ± 0.5	4.9 ± 0.9*
BAO	-	2.7 ± 0.4	6.3 ± 1.3*
MAO	-	13 ± 3	23 ± 5*
LES pressure	12.1 ± 2.1	8.2 ± 1.7	4.3 ± 0.7*
% ineffective contr.	18.7 ± 2.3	22.9 ± 4.2	47.9 ± 7.8*

Means ± SEM, *:p < 0.01 vs patients with esophagitis

Conclusion: Despite less symptoms patients with Barrett's esophagus have a markedly increased esophageal exposure to higher concentrated gastric juice compared to patients with esophagitis. This appears to be due to an increased frequency and prolonged duration of reflux episodes with concentrated acid secondary to a combination of acid hypersecretion with amechanically defective lower esophageal sphincter and inefficient esophageal clearance function. Planned surgical therapy should reduce esophageal acid exposure to normal by improving sphincter function with *a* Collis gastroplas-ty and a partial fundoplication so as not to potentiate the detrimental effect of reduced body function.

*By Invitation

WEDNESDAY MORNING, April 29, 1992

9:00 a.m. SIMULTANEOUS SCIENTIFIC SESSION D - ADULT CARDIAC SURGERY - Los Angeles Ballroom

39. The Right Gastroepiploic Artery Graft: Clinical and Angiographic Mid-Term Results in 200 Patients

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YASUSHI TERADA, M.D., SACHITO FUKUDA, M.D.*,*

TETSURO TAKA YAMA, M.D. and*

SHOICHIFURUTA, M.D.

Tokyo, Japan

Sponsored by: Donald B. Doty, Salt Lake City, Utah

From March 1986 to September 1991, the right gastroepipicic artery(GEA) graft has been used for CABG in 200 patients (171 males, 29 females, mean age 58 ranged from 6 to 80) and they were followed up from 2 to 66 months with a mean of 23 months.

There were 16 reoperation cases and 176 patients had triple vessel or left main disease. GEA (182 in-situ and 18 free grafts) was anastomosed to 11 anterior descending, 3 diagonal, 26 circumflex and 160 right coronary arteries. The internal thoracic artery was concomitantly used in 192 patients and mean number of distal anastomoses was 3.3 including additional saphenous vein grafts. Postoperative angiography was performed in 143 patients within 6 postoperative months (mean 2 months) and second angiography was made at 1 to 3 postoperative years (mean 2 years) in 38 patients sequentially.

There were 6 early and 3 late deaths. New Q wave was noted in 4 patients. Duration of surgery and postoperative complication did not increase with use of GEA. Relief of angina was noted in 196 patients. GEA patency was 95% (136/143) at early and 95% (36/38) at late potoperative period. There were 4 successful PTCA through in-situ GEA graft for GEA anastomotic stenosis. In stress myocardial scintigraphy sequentially performed at preoperative, and immediate, 1 year and 2 year postoperatively in 11 patients, washout rate (%)of GEA grafted area improved from 35 ± 10 to 45 ± 15 ($P<0.05$) and was maintained in 43 ± 6 and 48 ± 9 at respective periods. By implantable doppler flow study, in situ GEA graft flow increased with meal.

In conclusion, GEA is a suitable arterial conduit for CABG in terms of low surgical risk, high patency rate and excellent patient's outcome.

*By Invitation

40. Aorto-Coronary Bypass Graft Patency After High Dose Aprotinin

BENJAMIN P. BIDSTRUP, FRACS*
London, England

Sponsored by: J.C.R. Lincoln, FRCS, London, England

High dose aprotinin has been shown in several single centre studies to dramatically reduce postoperative blood loss. Remarkedly few side effects of this drug have been reported in these studies and in reports of routine clinical use. However concern has been expressed that the use of haemostatic agents during or after aorto-coronary bypass graft (ACBG) surgery might affect patency of vein grafts. The aim of this prospective study was to determine if high dose aprotinin resulted in increased vein graft occlusion after primary ACBG.

Patients and Methods: In a placebo controlled, double blind study, 96 male patients (mean age 58.9 years) received a total of 363 bypass grafts. High dose aprotinin (6 x 10^6 kallikrein inhibitory

units approximately) was administered intra-operatively to 47, the remainder receiving placebo. The operative procedure, bypass and postoperative care was maintained uniformly within limits imposed by the clinical course. All procedures were carried by a single surgical service. Vein graft patency was assessed at 6 - 12 days postoperatively by magnetic resonance imaging, using a spin-echo sequence in 90 (93%) of the patients. Both groups were similar with respect to age, weight, bypass time and number of grafts placed. All patients received low dose aspirin beginning the first postoperative day. Patency of vein grafts to endarterectomized arteries was analyzed separately.

Results: At the conclusion of the study, 269 vein grafts were assessed, 131 in the aprotinin group and 138 in the placebo group. In the aprotinin group, 96.2% of grafts were patent vs 97.1% in the placebo group (70% CL 93.7 -97.8% vs 94.8 - 98.5%). This difference was not significant when tested using the ratio estimate procedure, assuming non-independence of grafts ($p > 0.05$). All grafts were patent in the aprotinin group in 86.1 % of patients compared with 89.4% (70% CL: 78.6 - 91.5% vs 82.8 - 93.9%) in the placebo group. Postoperative hemoglobin loss was reduced by 50% in the aprotinin treated patients and homologous blood transfusions by 45%.

Conclusions: Early graft occlusion is due to thrombosis and is determined by the quality of the artery and its run-off, the quality and type of the conduit used, the surgeon and the technical adequacy of the anastomoses, and the interaction of the coagulation system. Early (7-10 day) vein graft patency, as determined by MR imaging is not adversely affected by high dose aprotinin use. At the same time, similar reductions in blood loss and blooduse as have been previously described, were achieved. The improvement in haemostasis afforded by aprotinin is not achieved by the creation of a pro-thrombotic state. Rather, haemostasis after cardiopulmonary bypass is less disturbed, thereby reducing the bleeding tendency.

*By Invitation

41. Left Ventricular Function in Experimental Mitral Regurgitation With Intact Chordae Tendineae

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CHRISTOPHER D. STONE, M.D. and*

RICHARD E. CLARK, M.D.

Bethesda, Maryland

Left ventricular (LV) function in mitral regurgitation (MR) has typically been studied in models that either sever the chordae tendineae or create a ventriculo-atrial shunt. These methods may have adverse effects on LV function independent of the regurgitant lesion itself. An animal model of chronic MR was therefore developed that both preserves annulo-ventricular continuity and avoids the use of external shunts. A circular 0.16-0.24 mm/kg defect was created in the anterior mitral valve leaflet of weaning sheep under direct vision using cardiopulmonary bypass. Six animals were studied pre- and immediately postoperatively (AMR-C and AMR groups, respectively), and 20 animals were studied 8.1 ±0.2 (mean±SD) months postoperatively (CMR group). CMR animals were compared to an age and weight matched, normal, nonoperated control group (CMR-C, n = 7). Volumetric data were derived from digitalized cineangiographic images, and Emax calculated from pressure-volume loops obtained from the simultaneous recording of LV pressure by micromanometer tipped LV catheters, and volumes obtained from digitalized images of epicardial echocardiographic recordings. The data showed that there was severe MR in both the AMR and CMR groups, with a calculated regurgitant fraction of 37 ±7%.

GROUP	CI	LV dP/dt	EDV	ESV	EF	Emax
AMR-C	102 ± 19	1567 ± 432	110 ± 17	45 ± 7	59 ± 6	11.8 ± 1.2
AMR	113 ± 26*	1376 ± 245	121 ± 23*	46 ± 10	62 ± 7	11.4 ± 1.7
CMR-C	114 ± 29	1588 ± 334	1456 ± 34	63 ± 20	57 ± 9	10.3 ± 0.9
CMR	72 ± 28[b]	806 ± 293[b]	202 ± 32[b]	104 ± 17[b]	48 ± 6[b]	7.8 ± 0.7[b]

Where: CI = cardiac index (ml/min/kg); dP/dt = mm Hg/sec; EDV and ESV = LV end diastolic and systolic volumes (ml); EF = ejection fraction (%); and E_{max} = maximal elastance at end-systole (mm Hg/ml).

*P < 0.01 compared to corresponding control group, bp < 0.01 compared to both control and AMR groups.

These results demonstrate that LV elastance and contractile function are preserved in acute MR with intact chordae tendineae, but decline progressively in chronic MR. Furthermore, the acute MR produced in this model is well tolerated, results in progressive LV dysfunction, and is analogous to that seen in human disease.

*By Invitation

42. Metabolic Evidence for Fibroblast Injury During Cardiac Valve Harvesting, Antibiotic Disinfection and Cryopreservation: Protection with Inhibitors of Andenosine Deaminase and Adenine Nucleotide Transport

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ANWAR S. ABD-ELFATTAH, Ph.D.,*

*RICHARD A. HOPKINS, M.D.**

PATRICK W. DOMKOWSKI, M.S.,*

DONALD G. CRESCENZO, M.D. and*

ROBERTB. WALLACE, M.D.

Washington, DC

Human cardiac valves are increasingly used in the reconstruction of ventricular outflow tracts and have been shown to offer performance advantages over porcine and mechanical prostheses; the long-term durability of these conduits is believed related to leaflet fibroblast viability at implantation. Preparation for their use as cryopreserved homografts entails multiple steps which are each potentially injurious to the leaflet cells. These include: (1) obligate harvest associated warm ischemia, (2) 24 hours of 4°C antibiotic disinfection and (3) DMSO protected cryopreservation at -1°C/min to -170°C. Using 118 semilunar porcine valves as a model of cadaveric harvest with a fixed 40 minutes of warm ischemia, leaflet cell high energy phosphates were assayed to quantify the associated metabolic injury. 58 control (Co) leaflets were processed according to the cryopreservation protocol artificially abbreviated at staggered degrees of completion to analyze stepwise catabolic damage. 60 additional leaflets were treated (Rx) at procurement with the adenine nucleotide transport inhibitor p- nitrobenzy-thioinosine and the adenosine deaminase inhibitor erythro-9-(2-hydroxy-3-nonyl) adenine to attempt nucleotide salvage of processing-incurred high energy phosphate losses. Control and treated valves of Group I were placed immediately in liquid N_2 after cardiotomy (baseline). Following harvest, Groups II and IV valves were placed for 24 hours in RPMI nutrient media (4°C) while III and V were similarly maintained with the addition of antibiotics (cefoxitin 240 µg/ml, lincomycin 120 µg/ml. polymyxin B 100 µg/ml,vancomycin 50 µg/ml). After the 24 hours, Groups II and III valves were metabolically quenched by immersion in liquid N_2 while IV and V were dimethylsulfoxide-

cryopreserved at 1° C/min to - 170°C. The efficacy of nucleotide salvage was evaluated through each step by comparing High Performance Liquid Chromatography assayed ATP, ADP, and AMP and Co and Rx-treated groups. Results normalized to leaflet protein (Lowry) as nanomoles nucleotide/mg protein (± SEM).

GROUP		I (N)	II (N)	III (N)	IV (N)	V (N)	KEY:
ATP	Co	1.79 ± .31 (12)	1.48 ± .15 (12)	0.91 ± .19* (11)	0.46 ± .12*§ (11)	0.25 ± .10* (11)	I - 40 min warm ischemia
	Rx	2.02 ± .21 (12)	1.20 ± .26 (12)	1.49 ± .11* (12)	1.58 ± .32* (12)	1.47 ± .32* (12)	II - 1-24 hrs cold ischemia
TAN	Co	2.59 ± .34 (12)	2.81 ± .34 (12)	2.27 ± .50 (11)	0.67 ± .13*§ (11)	0.67 ± .13*§ (11)	III - II with antibiotics
	Rx	3.79 ± .36 (12)	2.84 ± .35 (12)	2.57 ± .32 (12)	2.19 ± .24* (12)	2.19 ± .24* (12)	IV = II + cryopreservation
TAN = ATP + ADP + AMP (ANOVA) * p < .05 vs Group I & II							V - III + cryopreservation
** p < .05 vs corresponding control* § p < .05 vs Group III							

High energy nucleotide depletion of control valves occurs independently and most significantly with antibiotic disinfection and cryopreservation (Groups I and II > III, IV, and V, p < 0.05). High energy phosphates were maintained in corresponding *Rx-treated valves* indicating that nucleotide salvage therapy completely protects leaflet cells from this depletion during all homograft preparation steps. Such strategies may be clinically important in the long-term performance of homograft cardiac valve transplants.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By invitation

11:05 a.m. SIMULTANEOUS SCIENTIFIC SESSION D - ADULT CARDIAC SURGERY - Los Angeles Ballroom

43. Dynamic Cardiomyoplasty: A Seven Year Clinical Experience

ALAIN F. CARPENTIER, M.D.,

JUAN-CARLOS CHACHQUES, M.D.,*

PIERRE A. GRAND JEAN, M.S.,*

JOHN Y.M. RELLAND, M.D., CHRISTOPHE A CAR, M.D.**

*and DANIELLE BENSASSON, M.D.**

Paris, France

Since January 1985, the date of the first dynamic cardiomyoplasty, until October 1991, 45 patients (pts) (38 males and 7 females), aged 15 to 68 years (mean 50 years) were operated upon in our Institution. The mean pre-operative NYHA functional class was 3.3 and the mean ventricular ejection fraction (EF) was 17 ± 3%at rest. Mean cardiothoracic ratio (CTR) was 54 ± 5%. Maximal oxygen consumption was 13.7 ± 3.5 ml/min/kg. The number of hospitalizations due to congestive heart failure the year prior to surgery was 2.4 hosp./pt/year. Etiology of cardiac failure was ischemic cardiomyopathy (29), idiopathic dilated cardiomyopathy (13), cardiac tumor (2), congenital post-Fontan (1). Ventricular failure involved the LV (31), the RV (3), or both ventricles (10). In 40 pts,

the cardiomyoplasty was the first cardiac operation, while 5 pts had a previous operation. Surgical techniques consisted of ventricular reinforcement (32), ventricular substitution (11) and atrial reinforcement (1). The left latissimus dorsi muscle (LDM) was wrapped around the ventricles in a clockwise fashion in 41 pts, and in a counterclockwise fashion in 3 pts. Associated procedures in 20 pts comprised LV aneurysm resection (9), valve surgery (7), cABG (6), tumor resection (2). Thirteen pts were assisted perioperatively with an IABP and one with a VAD.

Pre-assist mortality rate before full LDM stimulation was 7/13 pts (54%) in the 1985-1987 period and 4/32 (12.5%) in the 1988-1991 period. The cause of death were heart failure (4), multiorgan failure (3), septicemia (2), ventricular fibrillation (1), sudden death (1). Multivariate analysis of factors influencing hospital mortality showed that age, cardiac suture technique, associated surgical procedure, biventricular heart failure, and pts hemodynamically unstable on inotropic drug support were predictors of unfavorable outcome.

All pts were followed from 3 months to 6.5 years (mean 21 months). Post-assist mortality rate was 7/34 (20.5%). Causes of deaths included heart failure (4), ventricular fibrillation (1), myocardial infarction (1), gastricbleeding (1). Pre-operative risk factors influencing long-term mortality are NYHA functional class IV, biventricular heart failure, atrial fibrillation, CTR>60%,EF< 15%.

Actuarial survival at 6 years was 11%(pre assist mortality excluded). Surviving pts were in mean NYHA functional class 1.8 (preop 3.3, $p < 0.05$). The average EF (rest/stress) were 25/28% at 1 years, 26/30% at 2 years, 23/28% at 3 years. Average CTR ratio were 57 ± 3% at 1 years, 56 ± 2% at 2 years, 57 ± 2.5% at 3 years. Catheterization obtained in 20 pts showed no significant changes at rest of capillary wedge pressure, pulmonary artery pressure, and diastolic left ventricular pressure at rest when compared to preoperative pressures. Average EF increased from 24 to 30.6%. Maximal oxygen consumption increased from 13.5 ± 3.5 to 17.5 ± 3 ml/min/kg. The number of rehospitalizations due to congestive heart failure was reduced to 0.4 hosp./pt/year (preop: 2.4, $p < 0.05$). In 62% of the pts, pharmacological therapy was reduced after surgery. Three pts required orthotopic heart transplantation 6 months, 4 years and 5 years after cardiomyoplasty. All are alive and well (mean follow-up 6 months).

Conclusions: 1) Continuous non fatiguable LDM contraction at heart rate has been obtained in the human for periods up to 6.5 years. 2) Cardiomyoplasty effectively improves the functional capacity and the survival of patients suffering from severe chronic heart failure. 3) It stops the process of continuous ventricular dilatation. 4) Hemodynamically proven benefit however remains unconstant although significant progress has been made as a result of better patient selection, new surgical techniques and better perioperative management.

*By invitation

44. Acceleration of Neointima Formation in Vascular Prostheses by Transplantation of Autologous Venous Tissue Fragments: Applicability to Small Diameter Grafts

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YOSHIHISA YAMANE, Vet, Ph.D.,*

SHINICHISATOH, M.D., TAKOFUMI OKOSHI, M.D.**

*and AKIHIKO MA TSUMOTO, M.D.**

Yokohama, Japan

Sponsored by: D. Craig Miller, M.D., Stanford, California

We developed a method to accelerate neointima formation in synthetic vascular prostheses by transplantion of autologous venous tissue fragments. A canine jugular vein was resected, minced into tissue fragments, and suspended. This was sieved through the wall of a highly porous fabric vascular prosthesis (MICROKNIT: Water porosity:4,000 ml/cm^2) by pressurized injection causing tissue fragments to be trapped in the graft wall. Tissue fragmented (TF) grafts (7 mm ID, 5.7 cm long) were implanted into the thoracic aorta of 35 dogs. Small TF-grafts (4 mm ID, 4 cm long) were pretreated with heparin and implanted into both carotid arteries in 16 dogs (32 grafts). Preclotted grafts without TF were implanted into the thoracic aorta (25 dogs) and carotid arteries (6 dogs, 12 grafts) as controls. Grafts were explanted from one hr to 495 days after implantation. In the TF-grafts, host cells migrated and proliferated actively into the fibrin inner capsule. A single layer of endothelial cells formed on the luminal surface, covering multiple layers of smooth muscle cells. New arterial wall formation was complete throughout the TF-grafts within 2 weeks; however, in the control grafts, neointima formation was limited just to the anastomotic sites even after 2 months. Twenty small TF-grafts in the carotid position were patent, but all control grafts were occluded within one week. These results demonstrate that neointima formation in dogs can be enhanced in synthetic fabric prostheses; furthermore, long-term patency of small-caliber vascular grafts is possible using this tissue fragmentation technique in dogs.

*By Invitation

45. The Importance of Exploration of the Aortic Arch in Type "A" Dissection

*TIRONE E. DAVID, M.D., JOANNE BOS, R.N.**

and ZHAO SUN, MS*

Toronto, Ontario

Since 1986, we have systematically explored the aortic arch of patients (pts) with type A dissection when the false lumen extends into the arch. Intimal tears are repaired or the arch is replaced; the distal anastomosis between the Dacron graft and arch or ascending aorta is performed under circulatory arrest, and cardiopulmonary bypass is re-established using antegrade arterial perfusion by cannulating the aortic arch through the Dacron graft.

From May 1981 to June 1991, 55 pts with type A aortic dissection were operated on. There were 41 men and 14 women whose mean age was 56 years, range 21 to 76. Twenty-four pts (Group I) were operated on before 1986 and 31 pts (Group II) were operated on after the new surgical approach was introduced. Preoperatively, 2 pts from Group I and 3 pts from Group II were known to have an intimal tear in the aortic arch. The false lumen involved the aortic arch in 19 pts (79%) in Group I and in 26 pts (84%) in Group II. The aortic arch was repaired or replaced in only 2 pts (8%) in Group I, and in 15 pts (48%) in Group II. There were 2 operative deaths, one in each group. Both deaths occurred in pts with extensive false lumens. The operative morbidity was similar in both groups. Contrast-enhanced CAT scans were performed in all operative survivors. Persistent false lumen was detected in 12 pts (66%) in Group I and,in only 1 pt (4%) in Group II. During a

mean follow-up of 46 months further aortic surgery was necessary in 4 pts; all 4 had persistent false lumen. There were 9 late deaths; 6 were cardiovascular-related. Five of these 6 deaths occurred in pts with persistent false lumen. The overall actuarial survival at 5 years was 83% +/-5%.

Our data suggest that intimal tears in the aortic arch are common in pts with type A dissection. Repair of these tears and antegrade perfusion through the Dacron graft obliterates the false lumen in most pts, decreases the probability of further aortic surgery and may enhance survival.

12:10 p.m. ADJOURN

*By Invitation

WEDNESDAY MORNING, April 29, 1992

9:00 a.m. SIMULTANEOUS SCIENTIFIC SESSION E - GENERAL THORACIC SURGERY - Beverly Hills Room

46. Medical Tumors of the Chest Wall: Plasmacytoma and Ewing's Sarcoma

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OZURU OKOHA, M.D. and*

ROBERT J. GINSBERG, M.D.

New York, New York

Solitary plasmacytoma and Ewing's sarcoma of the chest wall are relatively uncommon tumors and data concerning treatment and results are sparse. In order to assess the results of therapy we reviewed over 40 year experience.

Methods: Records of 24 patients with solitary plasmacytoma and 62 with Ewing's sarcoma arising in chest wall admitted to our institution from 1949 to 1989 were reviewed. Survival was calculated by Kaplan-Meier method; comparisons by log rank analysis; significance defined as $p < 0.05$.

Results: Plasmacytoma (n = 24): Age: 35-75 yr (median 59); M:F 2.4:1. Presenting complaint was pain and/or mass in 92% (22/24). Primary therapy was local only in 5 (resection in 3, radiotherapy (RT) in 2), chemotherapy in 16 (with resection in 4, RT in 11, alone in 1), and none in 3. Subsequent multiple myeloma developed in 75% (18/24). Overall 5 year survival was 38% (median: 56 mos). Age, sex, site of primary, and local therapy did not significantly impact on survival. **Ewing's sarcoma (n = 62):** Age 2-39 yr (median 16); M:F 1.6:1. Presenting complaint was pain and/or mass in 90% (56/62). Primary therapy was local in 32 (resection in 22, RT in 7, resection + RT in 3) and chemotherapy in 30 (with resection in 19, RT in 5, alone in 6). Overall 5 year survival was 48% (median: 57 mos). Age, sex, and site of primary did not significantly impact on survival. Patients who developed distant metastases (n = 48) had a significantly decreased survival (5 yr: 28%) compared to those who did not (n = 14; 5 yr: 100%).

Conclusion: Plasmacytoma of chest wall, even if solitary at presentation, should be considered a systemic disease and therapy directed as such. For Ewing's sarcoma, although resection may offer

local control, because of the high incidence of distant metastases (77%), systemic therapy should be considered an integral part of treatment.

*By Invitation

47. N2 Lung Cancer: Outcome in Patients With False Negative Chest CT Scans

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L. JACK PALING, M.D., JAMES T. DIEHL, M.D.*,*

MARK S. BANKOFF, M.D. and*

*DANIEL D. KARP., M.D.**

Boston, Massachusetts

Over the past 13 years, 681 consecutive patients with lung cancer have undergone computed tomographic (CT) and surgical staging of the mediastinum. Five hundred and one had negative mediastinal lymph node staging by CT and of these, 37 had cancerous mediastinal lymph nodes at mediastinoscopy (1) or thoracotomy (36). In order to determine the consequences of missing mediastinal adenopathy in patients with false negative chest CT scans, we analyzed the survival in this group of patients according to T status, central or peripheral location of tumor, cell type, areas of mediastinum involved, and the extent of nodal involvement with tumor. Twelve patients had central tumors: T2 - 5 pts., T3 - 5 pts., and T4 - 2 pts. Twenty-five had peripheral tumors: T1 - 12 pts., T2 -12 pts., and T3 -1 pt. Two of the patients in the central group died postoperatively and only two others remain alive whereas 12 of the 25 patients in the peripheral group remain alive. Four of the 37 patients, two in each group, were not resected and all are dead. One patient in each group is alive with disease. All but two of the resected 31 survivors received postoperative adjuvant XRT (23 pts.), chemotherapy (1 pt.), or XRT and chemotherapy (5 pts.). The projected two and five year survivals (Kaplan Meir) for all patients combined was 40% and 21%; for those resected 44% and 24%; for resected central tumors 40% and 0%; and for resected peripheral tumors 47% and 47%. None of these differences are significant ($P>0.10$). Cell type, location or number of locations of involved nodes, and the average percentage or maximum and average size or maximum size percentage of mediastinal nodes involved with tumor did not impact on survival. These data suggest that definitive thoracotomy with appropriate lymph node sampling or dissections is justified in all patients with negative chest CT scans since even patients with unsuspected N2 tumors can usually undergo resection with satisfactory 2 year survival in all patients and prolonged survival in patients with peripheral cancers.

*By Invitation

48. Comparison of Thoracoscopic Talc Pleurodesis With Standard Chest Tube Using Tetracycline and Bleomycin for Control of Malignant Pleural Effusion

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KENNETH A KESLER, M.D. and*

JOHN W. BROWN, M.D.

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Adequate treatment of malignant pleural effusions requires repeated aspirations of pleural fluid or drainage and sclerosis to relieve the distressing symptoms of breathlessness. We prospectively studied the use of in-tracavitary Talc insufflated by a Thoracoscope and compared this to historical controls; patients who participated in a randomized controlled study in which standard chest tube drainage with either Bleomycin or Tetracycline was instilled. The 25 patients in the Talc group underwent Thoracoscopy with local anesthesia and intravenous sedation. There ages ranged from 41 to 88 yrs. We recorded clinical characteristics, laboratory data, findings, and any complications associated with the procedure. Of the evaluable patients in the Talc group, 95% had a successful pleurodesis at 30 days, 92%at 60 days and at 88% at 90 days. In comparison, the bleomycin group had successful pleurodesis of 64% at 30 days and 70% at 90 days; the tetracycline group had successful pleurodesis of 33% at 30 days and 47% at 90 days. In the Talc group, one patient had extraluminal compression of the right lower lobe bronchus preventing lung reexpansion and subsequent pleurodesis. Another patient in Talc group had severe unexplained chest pain and required intensive care monitoring. In conclusion, thoracoscopically administered intracavitary Talc is a safe procedure and superior to tetracycline and bleomycin in the control of malignant pleural effusions.

*By Invitation

49. Successful Surgical Resection of Both Stages IIIA and IIIB Non-Small Cell Lung Cancer After Intensive Preoperative Chemoradiotherapy: A Southwest Oncology Group Trial

VALERIE W. RUSCH, M.D., KATHYS. ALBAIN, M.D.*,*

JOHNCROWLEY, Ph.D., THOMAS RICE, M.D.*,*

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JOHN R. BENFIELD, M.D.

New York, New York; Sacramento, California;

Seattle, Washington and Cleveland, Ohio

Recent studies suggest that preoperative chemo ± radiotherapy can improve the historically poor resectability and survival rate of Stage IIIA non-small cell lung cancer (NSCLC), but sometimes with significant associated morbidity/mortality. Such treatment has not been studied in IIIB NSCLC - usually considered unresectable. This Phase II trial tested the feasibility of intensive preoperative chemoradiotherapy for both IIIA and IIIB NSCLC in a multi-institutional setting that included university and community hospitals.

Methods: (1) patient (pts) with **pathologically** documented Tl-4 N2-3 (except pleural effusion) were eligible; (2) induction therapy was cisplatin 50 mg/m2days 1,8,29,36 + VP-16 50 mg/m^2 days 1-5, 29-33 + **concurrent RT** (4500 cGy, 180 cGy/fx); (3) resection was attempted 3-5 weeks after induction if the response was stable, partial, or complete; (4) complete nodal mapping at thoracotomy was required. **Results:** 122 pts entered. Complete clinical data currently available on 75 pts: median age = 58 (32-75); M:F = 49:26. 68/75 (91%) pts were eligible for surgery; 63/75 (84%) pts underwent thoracotomy; 55/75 (73%) pts, including 12 pts with "stable" response preop, had a complete resection; 4/63 (6%) pts died postoperatively.

	elig. for resection	complete resection	"complex"* resection	mean op time (hrs)	mean bid. loss (ml)	mean # days in hospital
IIIA	40	35 (88%)	13/35 (37%)	3.2 ± 1.36	741 ± 564	11.3 ± 8.7
IIIB	28	20 (72%)	6/20 (30%)	3.2 ± 1.64	547 ± 352	8.84 ± 4.53

*e.g. intrapericardial pneumonectomy, lobe + chest wall or vertebral body resection.

Complete pathology data currently available on 53 pts: 11 (21%) had no residual tumor; 20 (30%) pts had only rare microscopic foci of tumor. 2 year survival: 30% for IIIA and IIIB.

Conclusions: This intensive therapy is well tolerated; it leads to a better resectability rate and pathologic CR than most other reported regimens. Contrary to usual resection criteria this approach is applicable even to IIIB NSCLC. Surgical risk is similar to that for early stage lung cancer, even though extensive operations are often necessary. A planned randomized trial using this regimen will further assess the impact of resection on survival in IIIA/IIIB NSCLC.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

11:05 a.m. SIMULTANEOUS SCIENTIFIC SESSION E -GENERAL THORACIC SURGERY - Beverly Hills Room

50. Indications, Risks and Results of Completion Pneumonectomy

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JEAN DesLAURIERS, M.D.

St. Foy, Quebec

Completion pneumonectomy refers to an operation intended to remove what is left of a lung partially resected during previous surgery. The procedure is seldom indicated and, based on current literature, it carries a significantly higher risk of operative mortality/morbidity than that of standard pneumonectomy. Over the past 20 years, 60 consecutive patients aged 17 to 70 (avg: 49.7 years) and initially diagnosed as having lung cancer (N = 43), or benign pleuro-pulmonary disease (N = 17) underwent completion pneumonectomy (Table). The interval between the first operation and completion pneumonectomy averaged 31 months for carcinoma patients and 215 months for patients with benign disease.

Table - Indications for surgery

First resection (N)	Completion pneumonectomy: n	
Lung cancer (N = 43)	Local recurrence:	28
	Second primary:	12
	Benign disease:	3
Benign disease (N = 17)	Benign disease:	16
	Carcinoma:	1

For all patients, the previous thoracotomy incision was re-opened in its full length and manoeuvres such as rib resection, extra-pleural lung mobilization, intra-pericardial vessel ligation, division of the bronchus first, local application of glues and hemostatic agents, and bronchial re-

inforcement were routinely used. Six patients (10%) died intra-operatively (N = 2) or postoperatively (N = 4) and an additional 16 patients (21%)suffered one or more major non fatal complications. The operative mortality was higher for carcinoma patients (11.6%) than for patients with benign diseases (5.9%). Actuarial 5 year survival rates (including op. mortality) were 48% for the entire population, 33% for cancer patients, and 88% for patients with benign disease. Based on this data, we think that completion pneumonectomy can be safely done and with an operative risk similar to the one reported for standard pneumonectomy (6-10%). In addition, patients undergoing completion pneummonectomy have a reasonable prospect for long-term survival.

*By Invitation

51. Physiologic Evaluation of Pulmonary Function in the Lung Resection Candidate

JOSEPH I. MILLER, M.D.

Atlanta, Georgia

From 7/1/74 to 12/31/90, 2,340 patients (pts) who underwent pulmonary resection were evaluated by comprehensive analysis of pulmonary function. Pulmonary function test (PFT) criteria for resection were (1) pneumonec-tomy: FEV1 > 2L; FEV 25-75 > 1L; MBC > 55%; (2) lobectomy: FEV1 > 1L; FEV 25-75 > 0.6L; MBC > 40%; (3) wedge or segmental resection: FEV1 > 0.6L; FEV 25-75 > 0.6L; MBC > 40%. Pts with FEV1 < 0.6L; FEV 25-75 < 0.6L; MBC < 35% were considered inoperable for elective resection. Types of resection were (1) exploratory thoracotomy 503; segmental resection 116; wedge 769; lobectomy 785; pneumonectomy 161. Hospital mortality was thoracotomy 3 (0.59%); segmental resection 0%; wedge 1 (0.13%); lobectomy 3 (0.39%); pneumonectomy 8 (4.97%). Split perfusion scans were performed in 503 pts (21.5%); Reichel stress exercise testing was done in 217 pts (9.3%). Less than 40 pts (1.7%) were turned down for surgery on basis of PFT. A detailed analysis of each subgroup will be given.

*By Invitation

52. Early Surgical Intervention Improves Survival in Patients With Pulmonary Mucormycosis

MARK TEDDER, M.D., JOHN A. SPRATT, M.D.*,*

MARK P. ANSTADT, M.D., SANJAY S. HEGDE, B.A.*,*

STEPHEN D. TEDDER, M.D. and*

JAMES E. LOWE, M.D.

Durham, North Carolina

Mucormycosis is an opportunistic fungal infection with a predilection for the respiratory tract. The purpose of this study was to define the clinical presentation of pulmonary mucormycosis and to determine the effectiveness of surgical and medical treatments. The English literature (n = 225) and thirty patients at our institution were reviewed; patients with minimal pulmonary symptomatology and an incidental postmortem diagnosis were excluded. This represents the largest review in the literature as well as the largest single institutional series. For the combined groups (n = 255), the mean age was 41 ± 21 (SD) years with a male to female ratio of 7:3. Associated conditions included leukemia or lymphoma (37%), diabetes mellitus (32%), chronic renal failure

(18%), history of organ transplant (1.6%), and a known solid tumor (5.6%). The most common event precipitating hospitalization and subsequent diagnosis was pneumonia (61%) followed by diabetic ketoacidosis (12%). The overall in-hospital mortality was 80% (65% for patients with isolated pulmonary mucormycosis and 96% for those with disseminated disease). Of the 45% of patients who were diagnosed ante mortem, 63% were treated medically and 37% surgicall, with or without antifungal agents. Of the patients treated surgically, 61% underwent lobectomy, 11% pneumonectomy, 8% wedge resection, and 20% had an unspecified type of resection. The mortality in patients treated surgically was 11% (4/35), and significantly lower than the 71% (38/56) mortality in those treated medically ($p<0.0001$). In the subset of patients with disease *confined* to the lungs, the mortality rates were 9.4% (3/32) and 50% (15/30), respectively ($p =0.0006$). The most common causes of death were fungal sepsis (42%), respiratory insufficiency (27%), and hemoptysis (13%). Pulmonary mucormycosis most commonly presents in the immunocompromised patient and is associated with a high mortality. Treatment for patients with localized pulmonary mucormycosis should include anitfungal agents and early surgical resection.

12:10 p.m. ADJOURN

*By Invitation

WEDNESDAY MORNING, April 29, 1992

9:00 a.m. SIMULTANEOUS SCIENTIFIC SESSION F - CONGENITAL HEART DISEASE - Santa Monica Room

53. Intra-Ventricular Repair for Taussig-Bing Anomaly

*HIKARU MA TSUDA, M.D.**, *YASUNARU KAWASHIMA, M.D.,*

*TOSHIKATSU YAGIHARA, M.D.**,

*YASUHISA SHIMAZAKI, M.D.**, *FUMIO YAMAMOTO, M.D.**

*and KYOICHINISHIGAKI, M.D.**

Osaka, Japan

Between 1969 and 1990, 35 patients (pts) with Taussig-Bing anomaly underwent corrective surgery. Several kinds of operative procedures were utilized in the early series but recently either arterial switch operation (ASO) or intraventricular repair (IVR) was utilized. Before 1984, 18 pts underwent corrective surgery with 10 operative and 3 late deaths. Since 1985, 17 pts underwent repair with 1 operative and 1 late death. At present, basic indication for ASO is pts with aorta anterior and pulmonary artery posterior and that for IVR is side-by-side relation of the great arteries. Thus, 11 pts ranging in age from 1 month to 7 years (yrs) with an average of 2 yrs underwent IVR. Ten pts survived the operation and followed up to 21 yrs with an average of 6 yrs. No late death was reported and all are asymptomatic with two pts having residual VSD. Ten and 20 mmHg of LV-Ao systolic pressure gradients were found in 2 pts and 16 mmHg or RV-PA gradients in 2 pts at late postoperative study. These pressure gradients were zero or trivial in other pts. Postoperative left and right ventricular ejection fraction studied in 6 pts were on an average 68% and 41%. This type of corrective procedure is not always possible for Taussig-Bing anomaly but indicated for most of side-by-side type with satisfactory early and late postoperative results. The long term follow-up study in comparison with ASO is mandatory.

*By Invitation

54. Anatomic Repair of Transposition of Great Arteries With Ventricular Septal Defect and Aortic Arch Obstruction: Single Versus Two Stage Procedure

CLAUDE PLANCHE, M.D., JUAN V. COMAS, M.D.*,*

ALAIN SERRAF, M.D., FRANCOIS LACOUR-GAYET, M.D.*,*

JACQUELINE BRUNIAUX, M.D. and*

*ANITA TOUCHOT, M.D.**

Le Plessis Robinson, France

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

Birmingham, Alabama

Between Sept. 1, 1983 and Oct. 1, 1991, 37 patients (pts) underwent anatomic repair of Transposition of Great Arteries, Ventricular Septal Defect and Aortic Arch Obstruction (TGA, VSD and AAO). In group I, *25* pts (67.5%) underwent repair in a Two Stage Procedure [A] + [B]. Phase [A] = AAO repair with (15 pts) or without (10 pts) pulmonary artery banding through left thoracotomy (mean age 18.7 ± 23.4 days). There were 3 deaths and 3 reoperations. Phase [B] = Arterial switch operation with VSD closure (mean age 95.5 ± 122 days). There were 5 early deaths and no late deaths. Seven pts required reoperation. Mean delay between [A] to [B] was 77.5 ± 109 days. The overall mortality [A] + [B] was 8 early deaths (32%-70% CL 25%-44.2%) and 10 pts (40% - CL 32.3%-52.3%) required reoperation. The mean stay in Intensive Care Unit (ICU) was 24.7 ± 20 days. Mean follow-up of 59.6 ± 21.4 months was completed in all survivors. They were all but one in NYHA class I without medication. Actuarial survival rate and freedom from reoperation at 5 years were 67.5% and 60% respectively.

In group II 12 pts (32.4%) had a Single Stage Procedure through midster-notomy: Arterial switch operation with VSD closure and AAO repair (mean age 10.2 ± 5.5 days). There were 2 early deaths (16.6% - CL 10.8%-34.3%) and one late death after reoperation for overlooked multiple VSDs (10% -CL 6.7%-30%). Two pts (16.6% - CL 10.8%-34.3%) required reoperation. The mean stay in ICU was 11.7 ± 2.5 days. Mean follow-up of 22.4 ± 16.7 months was achieved in all survivors. They were all in NYHA class I without medication. Actuarial survival rate and freedom from reoperation at 3 years were 74.6% and 83.3% respectively.

Conclusions: The Single Stage Procedure: 1) Allows complete repair in neonates without need for multiple operations; 2) May decrease early mortality (16.6% vs 32% ns), reduces the reoperation rate (16.6% vs 40% ns) and the cumulative stay in ICU (11.7 days vs 24.7 days ns). However, surgical indications for Single or Two Stage Procedure need an accurate assessment of intracardiac and aortic arch anatomy.

*By Invitation

55. Two Ventricular Repair for Isomerism Heart With Common A-V Canal Utilizing Endocardial Cushion Prosthesis

YASUNARU KAWASHIMA, M.D.,

HIKARU MATSUDA, M.D., YASUAKI NAITO, M.D.*,*

TOSHIKATSU YAGIHARA, M.D., KEIJI KADOBA, M.D.**

*and OSAMU MATSUKI, M.D.**

Osaka, Japan

Isomerism heart (IH) is not always the candidate for two ventricular repair because of its complexity. Since 1983, we have repaired 6 IH with common atrioventricular canal (CAVC) utilizing endocardia! cushion prosthesis (ECP) which we are using since 1975 for repair of CAVC. Their ages ranged from 11 months to 10 years. Three were left isomerism and 3 were right isomerism. All patients (pts) were associated with DORV. Other associated anomalies were PS (3), azygos continuation (2), PL or PRSVC to LA (4), TAPVC (1), PAPVC (1), and dextrocardia (2). ECP composed of a vertical Dacron patch for the closure of septal defects with anterior extension of ventricular portion to construct a tunnel from LV to aortic valve and autologous pericardial wings sutured on both sides at the annular level to compensate the deficient AV valve leaflets was utilized.

Operations were performed through RA dividing common anterior and posterior leaflets. Anomalous systemic and pulmonary venous returns were repaired with or without additional prosthetic materials so as to separate them physiologically. All pts survived the operation and are doing well 1 to 9 years after surgery with no or trivial mitral regurgitation (MR) except one in whom moderate MR persisted.

Although IH is often associated with various anomalies and operated upon with Fontan type procedure, two ventricular repair with ECP was shown to have satisfactory operative as well as late results.

*By Invitation

56. Staged Modified Fontan Operation for Complex Cardiac Anomalies With Subaortic Obstruction

ROBERTO M. DiDONATO, M.D.,*

*ANTONIO AMODEO, M.D.**, *LORENZO GALLETTI, M.D.*,*

LUCIANO PASQUINI, M.D and*

CARLO MARCELLETTI, M.D.

Rome, Italy

Ventricular hypertrophy is a recognized risk factor for orthoterminal repair in cases of complex cardiac anomalies with subaortic obstruction. Between 1986 and 1991 we have treated 17 such patients with a new type of palliation combining a main pulmonary artery-ascending aorta anastomosis with a bidirectional cavopulmonary anastomosis. Their age and weight averaged 48.3 months (7-13 months) and 18.5 kg (6.2-67 kg). Seven patients had (S,L,L) single ventricle and the other 10 had more complex cardiac anomalies unsuitable for biventricular repair. All had subaortic obstruction. There were 2 hospital deaths (11.7%), due to hemorrage and to critical pre-operative condition, respectively. Mean follow-up was 23.4 months (range 1-58 months). Among the 15 hospital survivors, the proportion surviving 4 years postoperatively was 82%. Control cardiac catheterization in 8 patients showed absence of subaortic gradient, low pressure in the cavopulmonary system (mean 9 mmHg) and fair arterial O_2 saturation (80%). Six patients underwent a modified Fontan operation 18 ± 14 months after palliation. There was 1 hospital death

due to underestimated pulmonary vascular resistance. Another patient died elsewhere, 4.5 years later, for hemorrage at sternal re-entry during attempted Fontan operation. The other 8 patients are waiting for Fontan operation. This staged approach normalizes both pressure and volume ventricular load and provides adequate oxygenation before Fontan operation. Therefore, we advocate its use for patients with complex cardiac anomalies and subaortic obstruction.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

11:05 a.m. SIMULTANEOUS SCIENTIFIC SESSION F - CONGENITAL HEART DISEASE - Santa Monica Room

57. The Tricuspid Valve and Outcomes in Pulmonary Atresia and Intact Ventricular Septum

JOHN W. KIRKLIN, M.D., FRANK L. HANLEY, M.D.,*

ROBERT M. SADE, M.D., ROBERT M. FREEDOM, M.D.,*

NAVIN NANDA, M.D., and*

EUGENE H. BLACKSTONE, M.D.

Boston, Massachusetts

Among 274 neonates, treated for pulmonary atresia and intact ventricular septum and entered prospectively into a multi-institutional study (1987-1991), survival for at least 1, 12 and 48-months was 81%, *69%,* and 64% respectively. The dominant patient-specific risk factor for death (hazard function domain) at any time after entry was small dimensions of the tricuspid valve ($P = .001$) which was correlated with right ventricular volume ($r = 0.68$, $P<.0001$) and with the probability of a right ventricular dependent coronary circulation ($P = .0002$). The effect of the procedural risk factors, determined in a parsimonious multivariable analysis which included patient-specific risk factors, is illustrated in the nomogram, in which Z (tricuspid valve) is the dimension in terms of standard deviations below normal for age:

A subsequent systemic-pulmonary artery shunt, after an initial procedure which did not include a shunt, was placed (multivariable analysis) in 39%, 66%, and 91% of those with tricuspid Z-values of 0, -2, and -4 respectively. A subsequent transannular patch (TAP) procedure was performed within 1 year in 22% of those who initially received a surgical valvotomy.

Inference: A valvotomy or, more often, transannular patch is indicated as the initial procedure, and with a concomitant shunt if the tricuspid Z-value is less than -2, unless 1) Z less than -4, or 2) the coronary circulation is right ventricular dependent, in which instances only a shunt (and later a Fontan operation) is indicated. Only about 25% of patients have tricuspid Z less than -4.

*By invitation

58. Bless the Babies: 100 Late Survivors of Heart Transplantation During the First Year of Life

LEONARD L. BAILEY, M.D., STEVEN R. GUNDRY, M.D.,

ANEES RAZZOUK, M.D. and NAN WANG, M.D.**

Loma Linda, California

There is a rapid growth of interest in heart transplantation therapy (HTx) during early infancy. Since November 1985, 122 orthotopic HTx procedures have been performed in 121 consecutive infants ranging in age from 3 hours to 12 months. Indications for HTx included: Hypoplastic Left Heart Syndrome (52%), other complex structural anomalies (43%), and myopathy (3°7o). Most recipients had ductus-dependent circulation and were maintained on continuous infusion of PGE-1. From 10-25% of infants listed for transplantation have died annually while awaiting a donor heart. Heart donors were usually victims of trauma, sudden infant death, or birth asphyxia. A donor-recipient weight ratio of 4.0 or less has been acceptable. Graft cold ischemic (C.I.) time has ranged from 51-497 minutes. Recipient survival and late graft function have been identical for donor hearts procurred distantly (>4 hrs. C.I.) and those obtained regionally (<4 hrs. C.I.). Procurement is facilitated by single dose cold crystalloid cardioplegia and cold immersion transport. Profound hypothermic circulatory arrest was used for graft implantation. One hundred and eight (90%) recipients survived HTx and were discharged from hospital. There have been 8 late deaths, resulting in an 83% overall survival. Five year actuarial survival is projected at 80%. Actuarial survival among newborn recipients (N = 52) at 5 years is 84%. Chronic immunomodulation is cyclosporine-based and steroid-free. Surveillance is non-invasive, relying heavily on echocardiography, electrocardiography, and clinical intuition. There have been no late lethal infections. Tumor has not been encountered. Coronary occlusive disease is known to exist in only 1 chronic survivor. We conclude that HTx results in excellent life quality and is highly effective and durable therapy when applied during early infancy.

*By Invitation

59. A Decade of Pediatric Cardiac Transplantation and the Impact of FK 506

JOHN M. ARMITAGE, M.D., FREDERICK J. PRICKER, M.D.*,*

HARTLEY P. GRIFFITH, M.D.,

ROBERT L. HARDESTY, M.D. and

*THOMAS E. STARZL, M.D., Ph.D.**

Pittsburgh, Pennsylvania

The decade from 1982 through 1991 witnessed tremendous growth in pediatric cardiac transplantation. At our institution 63 cardiac transplants were performed during this period (age range, 1 day to 17 years). The etiology of cardiomyopathy was congenital (29), idiopathic (28), myocarditis (2), adriamycin cardiotoxicity (2), cardiac tumor (1), and carnitine deficiency (1). Eight children (13%) required extracorporeal" membrane oxygenation (ECMO) prior to transplantation. The overall survival in the group was 67%. There were 12 perioperative (30 days post transplant) deaths, graft failure (8), infection (3), and stroke (1) and 8 late deaths, chronic rejection/coronary arteriopathy (5), posttransplant lymphoproliferative disease (2) and tumor recurrence (1). The improvement in both early and late survival with accrued experience in pediatric transplantation is reflected in the 36 orthotopic cardiac transplants performed since June, 1988: 3 perioperative deaths (8%) and 4 late deaths (12%). In October 1989 our immunosuppressive protocol for the children changed from cyclosporine, azathioprine and steroids to FK 506. Twenty-two children have been enrolled in this study (ages 1 day to 17 years). The survival in the FK series was 91%. The actuarial freedom from grade 3 A rejection

at 3 and 6 months posttransplant on FK 506 was 60% versus 20% and 12%, respectively, for the 15 children transplanted prior to the use of FK 506 on cyclosporine based triple-drug therapy ($p < 0.001$, Mantel-Cox). The incidence of posttransplant hypertension was 4% in the FK group versus 70% in the cyclosporine group ($p < 0.001$, Fischer). No coronary disease has been observed in 11 annual catheterizations. Cardiac function (average LVEF, 66%) and the quality of these children's lives have been excellent. Additionally, 5 children have undergone rescue therapy with FK 506 for refractory rejection and/or drug toxicity with improvement or resolution in each case. Notably absent in this patient group were complaints of gingival hyperplasia, hirsutism or facial bone growth abnormalities. The absence of these debilitating side effects together with the observed ‘immune-advantage' and steroid sparing effects of FK 506 hold tremendous promise for the young patient facing cardiac transplantation and a future wedded to immunosup-pression.

12:10 p.m. ADJOURN

*By Invitation

GEOGRAPHICAL ROSTER

NECROLOGY

Garrett M. Brownrigg, M.D. St. John's, NF

R. Adams Cowley, M.D. Baltimore, MD

Egbert H. Fell, M.D. Lacey, WA

Robert T. Fox, M.D. Friday Harbor, WA

Vincenzo Gallucci, M.D. Padova, Italy

Joffre-Andre Gravel, M.D. Quebec, PQ

Frank R. Johnston, M.D. Winston-Salem, NC

George H. C. Joynt, M.D. Toronto, ON

Archibald R. Judd, M.D. Hamburg, PA

William H. Oatway, M.D. Laguna Hills, CA

Irving A. Sarot, M.D. Westhampton Beach, NY

Herbert J. Sullivan, M.D. Hamilton, ON

The American Association for Thoracic Surgery, 1990-1991

(Listed by Countries, States, Provinces and Cities)

Geographical - UNITED STATES

USA

ALABAMA

Birmingham

CALIFORNIA

Anaheim

Main, F Beachley

Blackstone, Eugene H

Blakemore, William S

Kahn, Donald R

Kessler, Charles R

Kirklin, James K

Kirklin, John W

McElvein, Richard B

Pacifico, Albert D

Montgomery

Simmons, Earl M

ARIZONA

Green Valley

McClenathan, James E

Mesa

Fisk, R. Leighton

Paradise Valley

Nelson, Arthur R

Phoenix

Brown, Lee B

Cornell, William P

Scottsdale

Pluth, James R

Sun City

Read, C Thomas

Tucson

Burbank, Benjamin

Copeland, Jack G, III

Sanderson, Richard G

Sethi, Gulshan K

ARKANSAS

Jasper

Hudson, W A

Little Rock

Campbell, Gilbert S

Read, Raymond C

Williams, G Doyne

Capistrano Beach

Flynn, Pierce J

Chico

Becker, Ronald M

Coronado

Silver, Arthur W

Covina

Carter, P Richard

El Cajon

Long, David M, Jr

El Macero

Andrews, Neil C

Escondido

Mannix, Edgar P, Jr

Flintridge

Penido, John R F

Fresno

Evans, Byron H

Indian Wells

Salyer, John M

Inglewood

Lee, Myles E

Irvine

Connolly, John E

Miller, Don R

La Canada

Meyer, Bert W

La Jolla

Fosburg, Richard G

Hutchin, Peter

Loma Linda

Bailey, Leonard L

Gundry, Steven R

Wareham, Ellsworth E

Long Beach

Bloomer, William E

Carlson, Herbert A

Stemmer, Edward A

Los Angeles

Buckberg, Gerald D

Davis, Lowell L

DeMeester, Tom R

Fonkalsrud, Eric W

Holmes, E Carmack

Hughes, Richard K

Kay, Jerome Harold

Khonsari, Siavosh

Laks, Hillel

Lindesmith, George G

Longmire, William, Jr

Maloney, James V, Jr

Mandal, Ashis K

Matloff, Jack M

Mulder, Donald G

Stiles, Quentin R

Waters, Paul F

Los Osos

Aronstam, Elmore M

Martinez

Guernsey, James M

Montebello

Lui, Alfred H F

Oakland

Ecker, Roger R

Iverson, Leigh I G

May, Ivan A

Orange

Gazzaniga, Alan B

Wakabayashi, Akio

Oxnard

San Bernardino

Misbach, Gregory A

San Diego

Baronofsky, Ivan D

Chambers, John S, Jr

Daily, Pat O

Jamieson, Stuart W

Kaye, Michael P

Lamberti, John J, Jr

Moreno-Cabral, Ricardo

Trummer, Max J

San Francisco

Ellis, Robert J

Gardner, Richard E

Grimes, Orville F

Hill, J Donald

Leeds, Sanford E

McEnany, M Terry

Rankin, J Scott

Richards, Victor

Roe, Benson B

Thomas, Arthur N

Turley, Kevin

Ullyot, David J

San Jose

Oakes, David D

San Marino

Tsuji, Harold K

Santa Ana

Pratt, Lawrence A

Santa Barbara

Higginson, John F

Dart, Charles H, Jr

Palm Springs

Goldman, Alfred

Palo Alto

Cohn, Roy B

Fogarty, Thomas J

Jamplis, Robert W

Peters, Richard M

Wilson, John L

Pasadena

Ingram, Ivan N

Newman, Melvin M

Pebble Beach

Ramsay, Beatty H

Sacramento

Benfield, John R

Berkoff, Herbert A

Follette, David M

Harlan, Bradley J

Hurley, Edward J

Miller, George E, Jr

Smeloff, Edward A

Jahnke, Edward J, Jr

Lewis, F John

Love, Jack W

Santa Cruz

Fishman, Noel H

Santa Monica

Morton, Donald L

Nelson, Ronald J

Santa Rosa

Neville, William E

Seal Beach

Baisch, Bruce F

St Helena

Dugan, David J

Stanford

Mark, James B D

Miller, D Craig

Oyer, Philip E

Shochat, Stephen J

Shumway, Norman E

Stinson, Edward B

Tiburon

Heydorn, William H

Torrance

Carey, Joseph S

Cukingnan, Ramon A

Moore, Thomas C

State, David

Victorville

Jurado, Roy A

COLORADO

Aspen

Zaroff, Lawrence I

Norwich

Kelley, Winfield O

Sharon

Wylie, Robert H

Wilton

Pool, John L

Woodbridge

Stansel, Horace C, Jr

Stern, Harold

DELAWARE

Newark

Lemole, Gerald M

Colorado Springs

Burrington, John D

Denver

Brown, Robert K

Clarke, David R

Condon, William B

Eiseman, Ben

Grover, Frederick L

Grow, John B

Harken, Alden H

Hopeman, Alan R

Paton, Bruce C

Pomerantz, Marvin

Rainer, W Gerald

Wright, George W

Englewood

Kovarik, Joseph L

Lakewood

Swan, Henry

Littleton

Pappas, George

Pueblo

Bartley, Thomas D

Vail

Fuller, Josiah

CONNECTICUT

Avon

Maier, Herbert C

Bridgeport

Rose, Daniel M

Hartford

Kemler, R Leonard

New Haven

Baldwin, John C

Glenn, William W L

Hammond, Graeme L

Wilmington

Pecora, David V

DISTRICT OF COLUMBIA

Washington

Aaron, Benjamin L

Bowles, L Thompson

Katz, Nevin M

Keshishian, John M

Lefemine, Armand A

Midgley, Frank M

Randolph, Judson G

Simmons, Robert L

Wallace, Robert B

FLORIDA

Atlantic Beach

Stranahan, Allan

Belleair

Lasley, Charles H

Boca Raton

Seley, Gabriel P

Clearwater

Wheat, Myron W, Jr

Coconut Grove

Center, Sol

Coral Gables

Cooke, Francis N

Gainesville

Alexander, James A

Jacksonville

Koster, J Kenneth, Jr

Stephenson, Sam, Jr

Lakeland

Brown, Ivan W, Jr

Marathon

Mangiardi, Joseph L

Marco

Kopf, Gary S

Lindskog, Gustaf E

New Milford

Okinaka, Arthur J

Miami

Bolooki, Hooshang

Chesney, John G

Daughtry, Dewitt C

Gentsch, Thomas O

Greenberg, Jack J

Jude, James R

Kaiser, Gerard A

MacGregor, David C

Papper, Emanuel M

Reis, Robert L

Ripstein, Charles B

Subramanian, S

Thurer, Richard J

Miami Beach

Spear, Harold C

Naples

Battersby, James S

Linberg, Eugene J

Smyth, Nicholas P D

North Palm Beach

Dorsey, John M

Orlando

Scott, Meredith L

Sherman, Paul H

Ponte Vedra Beach

Gilbert, Joseph, Jr

Punta Gorda

Taber, Rodman E

St Petersburg

Schuster, Samuel R

Rivkin, Laurence M

Symbas, Panagiotis

Williams, Willis H

Augusta

Ellison, Robert G

Rubin, Joseph W

Chickamanga

Hall, David P

Macon

Dalton, Martin L, Jr

Sealy, Will C

Van De Water, Joseph M

Savannah

Langston, Hiram T

Yeh, Thomas J

HAWAII

Honolulu

Ching, Nathaniel P

Gebauer, Paul W

McNamara, Joseph J

IDAHO

Boise

Herr, Rodney H

ILLINOIS

Chicago

Barker, Walter L

Breyer, Robert H

Campbell, Charles D

Ebert, Paul A

Faber, L Penfield

Daicoff, George R

DeMatteis, Albert

Tallahassee

Kraeft, Nelson H

Tampa

Angell, William W

Seller, Hawley H

Winter Haven

Maurer, Elmer P R

Winter Park

Bloodwell, Robert D

GEORGIA

Atlanta

Craver, Joseph M

Guyton, Robert A

Hatcher, Charles, Jr

Hopkins, William A

Jones, Ellis L

King, Richard

Lee, Arthur B, Jr

Mansour, Kamal A

Miller, Joseph I

Ferguson, Mark K

Goldin, Marshall D

Hanlon, C Rollins

Hartz, Renee S

Head, Louis R

Hunter, James A

Idriss, Farouk S

Karp, Robert B

Kittle, C Frederick

Mavroudis, Constantine

Michaelis, Lawrence

Najafi, Hassan

Norman, John C

Raffensperger, John

Replogle, Robert L

Shields, Thomas W

Thomas, Paul A, Jr

Vanecko, Robert M

Downers Grove

Leininger, Bernard J

Elmhurst

Mason, G Robert

Evanston

Anderson, Robert W

Fry, Willard A

Tatooles, Constantine

Glencoe

Rubenstein, L H

Maywood

DeLeon, Serafin Y

Keeley, John L

Montoya, Alvaro

Pifarre, Roque

Sullivan, Henry J

Shawnee Mission

Adelman, Arthur

Wichita

Tocker, Alfred M

KENTUCKY

Lexington

Crutcher, Richard R

Dillon, Marcus L, Jr

Todd, Edward P J

Louisville

Gray, Laman A, Jr

Mahaffey, Daniel E

Oak Brook

Hudson, Theodore R

Ilbawi, Michel N

Javid, Hushang

Jensik, Robert J

Nigro, Salvatore L

Park Ridge

Baffes, Thomas G

Weinberg, Milton, Jr

Peoria

DeBord, Robert A

Springfield

Wellons, Harry A, Jr

Winnetka

Mackler, S Allen

INDIANA

Indianapolis

Brown, John W

King, Harold

King, Robert D

Mandelbaum, Isidore

Shumacker, Harris B, 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

Ransdell, Herbert, Jr

LOUISIANA

Alexandria

Knoepp, Louis F

Baton Rouge

Berry, B Eugene

Beskin, Charles A

Metairie

Ochsner, Alton, Jr

New Orleans

Blalock, John B

DeCamp, Paul T

Hewitt, Robert L

Lindsey, Edward S

McFadden, Paul M

Mills, Noel L

Moulder, Peter V

Ochsner, John L

O'Neill, Martin J, Jr

Pearce, Charles W

Rosenberg, Dennis M

Schramel, Robert J

Webb, Watts R

MAINE

Liberty

Hurwitz, Alfred

Portland

Bredenberg, Carl E

Drake, Emerson H

Rockport

Swenson, Orvar

Windham

Hiebert, Clement

MARYLAND

Baltimore

Attar, Safuh

Stanford, William

KANSAS

Cunningham

Allbritten, Frank F, Jr

Baker, R Robinson

Baumgartner, William A

Blair, Emil

Dodrill, Forest D

Gardner, Timothy J

Oott, Vincent L

Haller, J Alex, Jr

McLaughlin, Joseph S

Michelson, Elliott

Reitz, Bruce A

Salomon, Neal W

Turney, Stephen Z

Watkins, Levi, Jr

Bethesda

Jones, Michael

Chevy Chase

lovine, Vincent M

Mills, Mitchell

Ellicott City

MacManus, Joseph E

Towson

Brawley, Robert K

Worton

Walkup, Harry E

MASSACHUSETTS

Boston

Akins, Cary W

Austen, W Gerald

Barsamian, Ernest M

Berger, Robert L

Bougas, James A

Braunwald, Nina S

Buckley, Mortimer J

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

Watkins, Elton, Jr

Cambridge

Harken, Dwight E

Malcolm, John A

Chestnut Hill

Laforet, Eugene G

Strieder, John W

Concord

Boyd, Thomas F

Soutter, Lamar

Dover

Black, Harrison

Lynnfield

Wesolowski, Sigmund A

Medford

Desforges, Gerard

Methuen

Burke, John F

Castaneda, Aldo R

Cleveland, Richard J

Cohn, Lawrence H

Collins, John J

Daggett, Willard M

Daly, Benedict D T

Deterling, Ralph, Jr

Ellis, F Henry, Jr

Frank, Howard A

Gaensler, Edward A

Grillo, Hermes C

Hilgenberg, Alan D

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

Wilson, Norman J

North Andover

Cook, William A

Shrewsbury

Moran, John M

Springfield

Engelman, Richard M

Rousou, John A

Vineyard Haven

Malm, James R

West Roxbury

Bernhard, William F

Khun, Shukri F

Westport Harbor

Findlay, Charles W, Jr

Williamstown

Wilkins, Earle W, Jr

Winchester

Taylor, Warren J

Worcester

Vander Salm, Thomas J

MICHIGAN

Ann Arbor

Bartlett, Robert H

Bove, Edward L

Gago, Otto

Greenfield, Lazar J

Kirsh, Marvin M

Morris, Joe D

Neerken, A John

Orringer, Mark B

Prager, Richard L

Sloan, Herbert

Payne, W Spencer

Puga, Francisco J

Schaff, Hartzell V

Trastek, Victor F

St Paul

Lillehei, C Walton

Miller, Fletcher A

Waubun

DeNiord, Richard N

MISSISSIPPI

Carthage

Logan, William D, Jr

Birmingham

Timmis, Hilary H

Clarkston

Gerbasi, Francis S

Detroit

Arbulu, Agustin

Arciniegas, Eduardo

Levine, Frederick H

Silverman, Norman A

Steiger, Zwi

Stephenson, Larry W

Wilson, Robert F

Grand Rapids

Harrison, Robert W

Meade, Richard H

Rasmussen, Richard A

Tomatis, Luis A

Grosse Pointe

Benson, Clifford D

MINNESOTA

Minneapolis

Arom, Kit V

Bolman, R Morton, III

Emery, Robert W

Foker, John E

Gannon, Paul G

Garamella, Joseph J

Helseth, Hovald K

Humphrey, Edward W

Kiser, Joseph C

Molina, J Ernesto

Nicoloff, Demetre M

Rochester

Bernatz, Philip E

Danielson, G K

McGoon, Dwight C

Jackson

Hardy, James D

Johnston, J Harvey, Jr

Netterville, Rush E

MISSOURI

Bridgeton

Codd, John E

Chesterfield

Bergmann, Martin

Columbia

Bryant, Lester R

Curtis, Jack J

Silver, Donald

Kansas City

Ashcraft, Keith W

Benoit, Hector W, Jr

Borkon, A Michael

Holder, Thomas M

Killen, Duncan A

Mayer, John H, Jr

Padula, Richard T

Piehler, Jeffrey M

Reed, William A

Van Way, Charles W, III

Mount Vernon

Campbell, Daniel C, Jr

St Louis

Baue, Arthur E

Connors, John P

Cooper, Joel D

Cox, James L

Ferguson, Thomas B

Flye, M Wayne

Johnson, Frank E

Kaiser, George C

Kouchoukos, Nicholas T

McGregor, Christopher G A

Olsen, Arthur M

Orszulak, Thomas A

Pairolero, Peter C

Roper, Charles L

Spray, Thomas L

Weldon, Clarence S

Wfflman, Vallee L

MONTANA

Missoula

Oury, James H

NEBRASKA

Omaha

Fleming, William H

Schultz, Richard D

NEVADA

Las Vegas

Little, Alex G

Swain, Julie A

NEW HAMPSHIRE

Jaffrey

Woods, Francis M

NEW JERSEY

Alpine

Holswade, George R

Bellville

Gerard, Franklyn P

Browns Mills

Fernandez, Javier

McGrath, Lynn B

Camden

Camishion, Rudolph C

East Orange

Auerbach, Oscar

Lewis, J Eugene, Jr

Patterson, G Alec

Pennington, D Glenn

Newark

Abel, Ronald M

Donahoo, James

Gielchinsky, Isaac

Hochberg, Mark S

Swan, Kenneth G

Paterson

Bregman, David

Short Hills

Timmes, Joseph L

Tenafly

Gerst, Paul H

NEW MEXICO

Albuquerque

Edwards, W Sterling

Las Vegas

Thai, Alan P

Santa Fe

Davila, Julio C

Silver City

Waddell, William R

NEW YORK

Albany

Foster, Eric D

Bay Shore

Ryan, Bernard J

Bronx

Altai, Lari A

Brodman, Richard F

Fell, Stanley C

Hackensack

Hutchinson, John E, III

Jersey City

Demos, Nicholas J

Millbum

Parsonnet, Victor

Moorestown

Morse, Dryden P

Morristown

Parr, Grant V S

Mount Laurel

Pierucci, Louis, Jr

Neptune

Bailey, Charles P

Roberts, Arthur J

New Brunswick

Kunderman, Philip J

Lewis, Ralph J

MacKenzie, James W

Scholz, Peter M

Ford, Joseph M

Prater, Robert W M

Hirose, Teruo

Brooklyn

Cunningham, J N, Jr

Levowitz, Bernard S

Sawyer, Philip N

Buffalo

Adler, Richard H

Andersen, Murray N

Bhayana, Joginder N

Hoover, Eddie L

Lajos, Thomas Z

Cooperstown

Blumenstock, David A

East Meadow

Strevey, Tracy E, Jr

Fayetteville

Bugden, Walter F

Effler, Donald B

Floral Park

Crastnopol, Philip

Lido Beach

Hines, George L

Loudonvllle

Alley, Ralph D

New Hyde Park

Amato, Joseph J

Barner, Hendrick B

New Rochelle

Rubin, Morris

New York

Acinapura, Anthony J

Adams, Peter X

Patchogue

Finnerty, James

Plattsburg

Potter, Robert T

Rochester

Craver, William L

DeWeese, James A

Hicks, George L

Schwartz, Seymour I

Stewart, Scott

Roslyn

Thomson, Norman B, Jr

Anagnostopoulos, C E

Bains, Manjit S

Beattie, Edward, Jr

Bloomberg, Allan E

Boyd, Arthur D

Cahan, William G

Clauss, Roy H

Conklin, Edward F

Cracovaner, Arthur J

Culliford, Alfred T

Ergin, M Arisan

Friedlander, Ralph

Ginsberg, Robert J

Green, George E

Griepp, Randall B

Holman, Cranston W

Isom, O Wayne

Jaretzki, Alfred, III

King, Thomas C

Kirschner, Paul A

Krieger, Karl H

Lambert, Adrian

Litwak, Robert S

Martini, Nael

McCord, Colin W

McCormack, Patricia M

Nealon, Thomas F, Jr

Redo, S Frank

Reemtsma, Keith

Rose, Eric A

Skinner, David B

Spencer, Frank C

Spotnitz, Henry M

Steichen, Felicien M

Subramanian, Valavanur A

Tice, David A

Wisoff, B George

Saranac Lake

Decker, Alfred M, Jr

Scarsdale

Robinson, George

Scottsville

Emerson, George L

Slingerlands

Kausel, Harvey W

Staten Island

Garzon, Antonio A

Stony Brook

Dennis, Clarence

Soroff, Harry S

Syracuse

Brandt, Berkeley, III

Meyer, John A

Parker, Frederick, Jr

Valhalla

Moggio, Richard A

Reed, George E

NORTH CAROLINA

Ashevllle

Belts, Reeve H

Scott, Stewart M

Takaro, Timothy

Chapel Hill

Bowman, Frederick, Jr

Keagy, Blair A

Starek, Peter J K

Wilcox, Benson R

Charlotte

Robicsek, Francis

Selle, Jay G

Taylor, Frederick H

Durham

Tyras, Denis H

Veith, Frank J

Wichern, Walter, Jr

Wolff, William I

Wolfe, Walter G

Young, W Glenn, Jr

Greenville

Chitwood, Walter R

Isle of Palms

Mullen, Donald C

Oriental

Deaton, W Ralph, Jr

Pinehurst

Fischer, Walter W

Winston-Salem

Cordell, A Robert

Crosby, Ivan K

Hammon, John W, Jr

Hudspeth, Allen S

Meredith, Jesse H

Mills, Stephen A

NORTH DAKOTA

Grand Forks

James, Edwin C

OHIO

Canton

Wallsh, Eugene

Chagrin Falls

Cross, Frederick S

Cincinnati

Albers, John E

Callard, George M

Flege, John B, Jr

Gonzalez, Luis L

Jones, Robert H

Lowe, James E

Oldham, H N, Jr

Sabiston, David C

Kilman, James W

Meckstroth, Charles

Myerowitz, P David

Vasko, John S

Williams, Thomas E, Jr

Dayton

DeWall, Richard A

Pepper Pike

Mendelsohn, Harvey J

OKLAHOMA

Jenks

LeBeck, Martin B

Lawton

Barnhorst, Donald A

Oklahoma City

Elkins, Ronald C

Felton, Warren L, II

Fisher, R Darryl

Greer, Allen E

Munnell, Edward R

Williams, G Rainey

Zuhdi, M Nazih

OREGON

Days Creek

Miller, Arthur C

Portland

Cobanoglu, Adnan

Krause, Albert H

Okies, J Edward

Poppe, J Karl

Helmsworth, James A

Hiratzka, Loren F

Ivey, Tom D

Wilson, James M

Wright, Creighton B

Yee, Edward S

Cleveland

Ankeney, Jay L

Cosgrove, Delos M

Geha, Alexander S

Grondin, Claude M

Groves, Laurence K

Kay, Earle B

Loop, Floyd D

Lytle, Bruce W

Snow, Norman J

Van Heeckeren, Daniel W

Columbus

Clatworthy, H W, Jr

Davis, J Terrance

Kakos, Gerard S

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

Starr, Albert

PENNSYLVANIA

Abington

Frobese, Alfred S

Ardmore

Hargrove, W Clark, III

Bethlehem

Snyder, John M

Bryn Mawr

Haupt, George J

Mundth, Eldred D

Camp Hill

Pennock, John L

Carlisle

DeMuth, William, Jr

Darby

McKeown, John J, Jr

Erie

Kerth, William J

SOUTH CAROLINA

Charleston

Bradham, R Randolph

Crawford, Fred A, Jr

Parker, Edward F

Sade, Robert M

Columbia

Almond, Carl H

Landrum

Stayman, Joseph W

Spartanburg

Utley, Joe R

TENNESSEE

Addonizio, Paul V

Brockman, Stanley K

Deac, Radu C

DiSesa, Verdi J

Dunn, Jeffrey M

Edie, Richard N

Edmunds, L Henry, Jr

Fineberg, Charles

Kaiser, Larry R

MacVaugh, Horace, III

Nemir, Paul, Jr

Norwood, William I

Pittsburgh

Bahnson, Henry T

Clark, Richard E

Ford, William B

Griffith, Bartley P

Hardesty, Robert L

Magovern, George J

Pontius, Robert G

Rams, James J

Rosemont

Sink, James D

Templeton, John, III

Sayre

Sewell, William H

Wayne

Lemmon, William M

Wyncote

Mendelssohn, Edwin

Wynnewood

Wallace, Herbert W

Yardley

Sommer, George N, Jr

RHODE ISLAND

Providence

Knoxville

Blake, Hu Al

Brott, Walter H

Domm, Sheldon E

Memphis

Cole, Francis H

Eastridge, Charles E

Garrett, H Edward

Howard, Hector S, Jr

Hughes, Felix A, Jr

McBurney, Robert P

Pate, James W

Robbins, S Gwin, Sr

Rosensweig, Jacob

Skinner, Edward F

Watson, Donald C

Nashville

Alford, William, Jr

Bender, Harvey W, Jr

Gobbel, Walter G, Jr

Merrill, Walter H

Sawyers, John L

Scott, Henry W, Jr

Stoney, William S

Thomas, Clarence, Jr

Sparta

Labrosse, Claude C

TEXAS

Amarillo

Sutherland, R Duncan

Austin

Hood, R Maurice

Burnet

Ross, Raleigh R

Coppell

McPhail, Jasper L

Karlson, Karl E

Moulton, Anthony L

Singh, Arun K

Dallas

Adam, Maurice

Estrera, Aaron S

Holland, Robert H

Lambert, Gary J

Mills, Lawrence J

Paulson, Donald L

Platt, Melvin R

Razzuk, Maruf A

Ring, W Steves

Seybold, William D

Shaw, Robert R

Sugg, Winfred L

Urschel, Harold, Jr

Dilley

Hood, Richard H, Jr

El Paso

Glass, Bertram A

Fort Sam Houston

Zajtchuk, Rostik

Galveston

Conti, Vincent R

Derrick, John R

Tyson, Kenneth R T

Houston

Beall, Arthur C, Jr

Burdette, Walter J

Cooley, Denton A

Crawford, E Stanley

DeBakey, Michael E

Frazier, Oscar H

Heaney, John P

Treasure, Robert L

Trinkle, J Kent

Temple

Brindley, G V, Jr

Woodville

Harrison, Albert W

UTAH

Salt Lake City

Cutler, Preston R

Doty, Donald B

Gay, William A, Jr

Liddle, Harold V

Mortensen, J D

Nelson, Russell M

Wolcott, Mark W

VERMONT

Burlington

Coffin, Laurence H

Miller, Donald B

Chester Depot

Adams, Herbert D

West Dover

Humphreys, G H, II

White River Junction

Tyson, M Dawson

VIRGIN ISLANDS

St Thomas

Wilder, Robert J

VIRGINIA

Annandale

Hallman, Grady L

Henly, Walter S

Lawrie, Gerald M

Mattox, Kenneth L

Morris, George C, Jr

Mountain, Clifton F

Ott, David A

Overstreet, John W

Reul, George J, Jr

Roth, Jack A

Walker, William E

Wukasch, Don C

Kaufman

Davis, Milton V

Lubbock

Bricker, Donald L

Feola, Mario

San Antonio

Cohen, David J

Dooley, Byron N

French, Sanford, III

Akl, Bechara F

Lefrak, Edward A

Arlington

Conrad, Peter W

Klepser, Roy G

Charlottesville

Dammann, John F

Kron, Irving L

Minor, George R

Muller, William, Jr

Nolan, Stanton P

Lynchburg

Moore, Richmond L

Richmond

Bosher, Lewis H, Jr

Brooks, James W

Cole, Dean B

Gwathmey, Owen

Lower, Richard R

Wechsler, Andrew S

WASHINGTON

Bellingham

Varco, Richard L

Friday Harbor

Lawrence, G Hugh

IssaQuah

Jarvis, Fred J

Kirkland

Mills, Waldo O

Poulsbo

Malette, William G

Seattle

Anderson, Richard P

Morgantown

Graeber, Geoffrey M

Murray, Gordon F

Warden, Herbert E

Parkersburg

Tarnay, Thomas J

WISCONSIN

Delafield

Hausmann, Paul F

La Crosse

Gundersen, Erik A

Madison

Chopra, Paramjeet S

Ashbaugh, David G

Dillard, David H

Hill, Lucius D, III

Jones, Thomas W

Li, Wei-I

Mannas, Dev R

Mansfield, Peter B

Miller, Donald W, Jr

Rittenhouse, Edward A

Sauvage, Lester

Thomas, George I

Verrier, Edward D

Spokane

Berg, Ralph, Jr

WEST VIRGINIA

Charlestown

Walker, James H

Huntlngton

Gonzalez-Lavin, Lorenzo

Kroncke, George M

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

CANADA

ALBERTA

Calgary

Miller, George E

Edmonton

Callaghan, John C

Gelfand, Elliot T

Sterns, Laurence P

BRITISH COLUMBIA

Kelowna

Couves, Cecil M

Vancouver

Allen, Peter

Ashmore, Phillip G

Jamieson, W R Eric

Sudbury

Field, Paul

Walker, George R

Toronto

Baird, Ronald J

Bigelow, Wilfred G

Coles, John G

David, Tirone E

Delarue, Norman C

Goldberg, Melvyn

McKneally, Martin F

Mickleborough, Lynda

Pearson, F Griffith

Salerno, Tomas A

Tyers, G Frank O

Victoria

Stenstrom, John D

West Vancouver

Robertson, Ross

MANITOBA

Winnipeg

Barwinsky, Jaroslaw

Cohen, Morley

NOVA SCOTIA

Halifax

Landymore, Roderick W

Murphy, David A

Mabou

Thomas, Gordon W

ONTARIO

Collingwood

Heimbecker, Raymond

North York

Goldman, Bernard S

Nottawa

Key, James A

Ottawa

Keon, Wilbert J

Todd, Thomas R J

Scully, Hugh E

Trimble, Alan S

Trusler, George A

Weisel, Richard D

Williams, William G

Westbrook

Lynn, R Beverley

QUEBEC

Montreal

Blundell, Peter E

Chiu, Chu-Jeng (Ray)

Cossette, Robert

Dobell, Anthony R

Duranceau, Andre C H

Lepage, Gilles

MacLean, Lloyd D

Morin, Jean E

Mulder, David S

Pelletier, Conrad L

Scott, Henry J

Sainte-Foy

DesLauriers, Jean

Sillery

Grondin, Pierre

OTHER COUNTRIES

AFGHANISTAN

Kabul

Hankins, John R

ARGENTINA

Buenos Aires

Favaloro, Rene G

AUSTRALIA

SOUTH AUSTRALIA

FRANCE

Meudon

Cachera, Jean Paul

Paris

Binet, Jean-Paul

Blondeau, Philip

Cabrol, Christian E A

Carpentier, Alain F

Piccadilly

Sutherland, H D'Arcy, FRCS

VICTORIA

Melbourne

Nossal, Gustav J V

AUSTRIA

Puchenau

Bruecke, Peter E

Salzburg

Unger, Felix H

Vienna

Wolner, Ernst

BRAZIL

Sao Paulo

Jatene, Adib D

Zerbini, E J

ENGLAND

Bath, Avon

Belsey, Ronald

Cambridge

Kennedy, John H

Hereford

Thompson, Vernon C

Herefordshire

Smith, Roger A

London

Braimbridge, Mark V

Lennox, Stuart C

Lincoln, Christopher R

Ross, Donald N

Stark, Jaroslav F

Taylor, Kenneth M

Yacoub, Magdi

FINLAND

Kauniainen

Mattila, Severi P

Piwnica, Armand H

Pessac, Bourdeaux

Couraud, Louis

Fontan, Francis M

Suresnes

Bachet, Jean E

GERMANY

Aachen

Messmer, Bruno J

Hamburg

Rodewald, Georg

Hannover

Borst, Hans G

Munich

Sebening, Fritz

Neuss

Bircks, Wolfgang H

GUATEMALA

Guatemala City

Herrera, Rodolfo

INDIA

Raiputana

Van Allen, Chester M

IRELAND

Dublin

O'Malley, Eoin

ITALY

Bergamo

Parenzan, Lucio

Padova

Peracchia, Alberto

Rome

Marcelletti, Carlo

JAPAN

Kanazawa

Iwa, Takashi

Kitakyushu

Miyamoto, Alfonso T

Osaka

Kawashima, Yasunaru

Sendai

Mohri, Hitoshi

Tokyo

Imai, Yasuharu

Wada, Juro J

MONACO

Monte Carlo

Dor, Vincent

NEW ZEALAND

Auckland

Barratt-Boyes, Sir Brian

P.R. OF CHINA

Beijing

Ying-Kai, Wu

PORTUGAL

Lisbon

Macedo, Manuel E M

RUSSIA

Moscow

Bockeria, Leo A

Burakovsky, Vladimir I

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 O

SWITZERLAND

Arzier

Hahn, Charles J

Pully

Naef , Andreas P

Zurich

Senning, Prof Ake

Turina, Marko I

UNITED ARAB EMERIT

Abu Dhabi

Brom, A Gerard

VENEZUELA

Caracas

Tricerri, Fernando E

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

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

BY-LAWS OF

THE AMERICAN ASSOCIATION

FOR THORACIC SURGERY

ARTICLE I. Name

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

ARTICLE II. Purpose

The purposes of the Association shall be:

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

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

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

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

ARTICLE III. Membership

Section 1. There shall be four classes of members: Honorary, Senior, Active and, for a time, Associate. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws.

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

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

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

Section 5. Election to Honorary, Senior or Active Membership shall be for life, subject to the provisions of Section 9 following. There shall be no further additions to the Associate Membership. All new members shall be elected directly to Honorary or Active status.

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

Section 7. Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active or Senior Members. Such nomination must have been in the hands of the Membership Committee for not less than four months, and the name of the candidate must have been distributed to all members of the Association before final action may be taken on any new candidate for election to Active Membership. Provided the foregoing requirements have been met and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a regularly convened annual meeting for final action. A three-fourths vote of those present and voting shall be required to elect. Any candidate for membership in this Association who has failed of election for three successive years shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

Section 8. The report of the Membership Committee shall be rendered at the second executive session of each annual meeting of the Association. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; Candidates for Active Membership, Associate Members for re-election; members dropped from the rolls of the Association.

Section 9. Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving such member ample opportunity to appear in his own behalf.

Section 10. The Council shall recommend that any Active or Associate Member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from three consecutive annual meetings, shall have his membership terminated.

Section 11. Notwithstanding Section 10, any member of the Association over 65 years of age is excused from the attendance requirement and upon his specific request may likewise be excused from the payment of dues.

ARTICLE IV. Board of Directors ("Council")

Section 1. The Board of Directors of the Association shall be called the Council and shall be composed of the President, Vice-President, Secretary, Treasurer and Editor of the Association, and five Councilors. All members of the Council must be Active or Senior Members of the Association, except that the Editor may be an Honorary Member.

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

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

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

c. It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article III, Section 9.

d. It may not deplete the principal of the Endowment Fund.

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

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

ARTICLE V. Officers

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

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

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

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 a new 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.

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 the Editor of the Association, and at least two members-at-large appointed by the President. 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 six members: the President, Secretary, and Treasurer of the Association and three members-at-large, one member being appointed by

the President each year to serve a term of three years. The Chairman shall be the member-at-large serving his third year. The duties of the Committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and the Fellows, past, present, and future.

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

Section 9. The Ethics Committee shall consist of five members appointed by the Council. No member shall serve more than four years. The Ethics Committee shall advise the Council concerning alleged breaches of ethics. Complaints regarding alleged breaches of ethics shall be received in writing by the Ethics Committee and shall be investigated by it. In addition, the Ethics Committee may investigate on its own initiative.

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

ARTICLE VII Finances

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

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

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

Section 4. Funds derived from the payment of initiation fees shall not be available for current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact, and to be known as the Endowment Fund. The Council is responsible for the proper management of the Endowment Fund, and may divert any surplus in the current funds of the Association into this fund, but may not withdraw any of the principal of the Endowment Fund except in accordance with the provisions of Section 6, following.

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

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

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

ARTICLE VIII. Meetings

Section 1. The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council and the provisions of these By-Laws.

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

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

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

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

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

1. Appointment of necessary committees.

2. Miscellaneous business of an urgent nature.

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

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

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

3. Audit Report.

4. Report of the Necrology Committee.

5. Report of the Program Committee.

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

7. Action on recommendations emanating from the Council.

8. Unfinished Business.

9. New Business.

10. Report of the Membership Committee.

11. Election of new members.

12. Report of the Nominating Committee.

13. Election of officers.

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

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

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

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

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

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

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

ARTICLE IX. Indemnification and Directors and Officers

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

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

ARTICLE X. Papers

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

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

ARTICLE XL Initiation Fees, dues and Assessments

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

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

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

Section 4. Senior Members are exempt from dues.

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

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

Section 7. Associate and Active Members must subscribe to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY to retain their membership status.

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

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

ARTICLE XII. Parliamentary Procedure

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

ARTICLE XIII. Amendments

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

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

As amended, Tuesday, May 8, 1990

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

AWARDS

GRAHAM EDUCATION AND RESEARCH FOUNDATION
13 Elm Street, Manchester, Massachusetts 01944, (508) 526-8330

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

Vice President William A. Gay, Jr., M.D., Salt Lake City, Utah

Secretary-Treasurer William T. Maloney, Manchester, Massachusetts

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

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWSHIP

The Evarts A. Graham Memorial Traveling Fellowship was established in 1958 by The American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young surgeons from North America and abroad who have completed their formal training in general, thoracic, and cardiovascular surgery. The award allows the recipient to study a year to intensify his training in a program of special interest and to travel to several sites to broaden his overall training and increase his contacts with thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having the potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 40 young surgeons from 21 countries have completed their training at thoracic surgical centers.

1st	1951-52	L. L. Whytehead, M.D., F.R.C.S. 790 Sherbrooke St., Winnipeg, Manitoba, R3A 1M3 CANADA
2nd	1953-54	W. B. Ferguson, M.B., F.R.C.S. Royal Victoria Infirmary, Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley, M.Chir., F.R.C.S. St. Mary's Hospital, London, W.2, ENGLAND
4th	1955-56	Raymond L. Hurt, F.R.C.S. The White House, 8 Loom Lane, Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth, F.R.C.S. Brompton Hospital, London, S.W. 3, ENGLAND
6th	1957-58	Peter L. Brunnen, F.R.C.S. Department of Thoracic Surgery, Woodend General Hospital Aberdeen, SCOTLAND
7th	1958-59	N. G. Meyne, M.D. University of Amsterdam, Wilhelmina-Gasthuis,Amsterdam, HOLLAND
8th	1960-61	Godrej S. Karai, M.D. Calcutta, INDIA
9th	1961-62	Fritz Helmer, M.D. Second Surgical Clinic, University of Vienna, Vienna, AUSTRIA
10th	1962-63	Theodor M. Scheinin, M.D. Tammisalonitie 20, Helsinki, 00830, Finland
11th	1963-64	Masahiro Saigusa, M.D. National Nakano Chest Hospital, 3-14-20 Egata, Nakano-Ku, Tokyo 165, JAPAN
12th	1963-64	Adar J. Hallen, M.D. Department of Thoracic Surgery, University Hospital Uppsala, SWEDEN

13th	1964-65	Stuart C. Lennox, M.D.
		18 Alexander Sq., 5W3 2AX, London, ENGLAND
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S.
		Thessaloniki, GREECE
15th	1965-66	Gerhard Friehs, M.D.
		Chirugische University Klinik, Graz A-8036, AUSTRIA
16th	1965-66	Ary Blesovsky M.D.
		London, England
17th	1966-67	C. Peter Clarke, F.R.A.C.S.
		Ste. #4, 6th Floor, 55 Victoria Parade, Fitzroy 3065 AUSTRALIA
18th	1966-67	G. B. Parulkar, M.D.
		K.E.M. Hospital & Seth G.S., Medical College, Bombay 400 012, INDIA
19th	1967-68	Claus Jessen, M.D.
		Surg. Dept. D, Rigshospitalet, Blegdamsvej 9, Copenhagen, DENMARK
20th	1969-70	Peter Bruecke, M.D.
		AM Steinbruch, 29 Linz-Puchenau, A-4040, AUSTRIA
21st	1970-71	Michel S. Slim, M.D.
		New York Medical College, Division of Pediatric Surgery
		New York, New York 10595 USA
22nd	1971-72	Severi Pellervo Mattila, M.D.
		Department of Thoracic Surgery, Helsinki University CentralHospital, Helsinki 29, FINLAND
23rd	1972-73	Yasuyuki Fujiwara, M.D.
		Department of Cardiovascular Surgery, Tokyo Medical College Hospital, Shinjuku, Tokyo, JAPAN
24th	1973-74	Marc Roger deLeval, M.D.
		8 Thornton Way, Hampstead Garden Suburb, London NW11, ENGLAND
25th	1974-75	J. J. DeWet Lubbe, M.D.
		1406 City Park Medical Center, 181 Longmarket St., Cape Town 8001, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner, M.D.
		Institute of Surgery, 80-211 Ul, Deinsky 7, Gdansk, POLAND
27th	1976-77	Bum Koo Cho, M.D.
		Yonsei University, P.O. Box 71Severance Hospital, Seoul, KOREA
28th	1977-78	Alan William Gale, M.D., FRACP, FRACS

		171 Sutherland, Paddington 2021 Sydney, AUSTRALIA
29th	1978-79	Eduardo Otero Coto, M.D.
		Servicio de Cirugia Cardiovascular, Ciudad Sanitaria "Le Fe"
		Valencia, SPAIN
30th	1980-81	Richard K. Firmin, M.D.
		"Moss Grove", 5 Knighton Grange Road, Stoneygate, Leicester LE2 2LF, ENGLAND
31st	1981-82	Claudio A. Salles, M.D.
		Av Celso Porfirio Machado, 370, Bairro Belvedere
		Belo Horizonte MO, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki, M.D.
		First Dept. of Surgery, Osaka Univ. Medical School
		Fukushima-ku, Osaka 553, JAPAN
33rd	1983-84	Georg S. Kobinia, M.D.
		LKH Klagenfurt, Dept. of Cardiac Surgery, Klagenfurt,9020, AUSTRIA
34th	1984-85	Aram Smolinsky, M.D.
		Department of Cardiac Surgery, The Sheba Medical Center
		Tel Hashomer, 52621, ISRAEL
35th	1985-86	Florentino J. Vargas, M.D.
36th	1986-87	Ari L. J. Harjula, M.D.
		Mitalitte 2 A, 4 02680 Espoo68, SF, Finland
37th	1987-88	Byung-Chul Chang, M.D.
		Dept. of Thoracic and Cardiovascular Surgery, Yonsei University College of Medicine, CPO Box 8044, Seoul, Korea
38th	1988-89	Wang Cheng, M.D.
		Department of Cardiac Surgry, Beijing Heart, Lung, Blood Vessel Medical Center & Anzhen Hospital, Andingmenwai, Beijing,PEOPLE'S REPUBLIC OF CHINA
39th	1989-90	Christopher John Knott-Craig, M.D.
		Univ. of Stellenbosch, P.O. Box 65, Tygerberg, South Africa7505
40th	1991-92	Ko Bando, M.D., Ph.D.
		Division of Cardiac Surgery, Okayama University Medical School, 5-1 Shikatcho 2 Chome, Okayama City 700 Japan
41st	1992-93	Timothy E. Oaks, M.D.
		Department of Surgery, The Milton S. Hershey Medical Center Room #6314, Box 850, Hershey, Pennsylvania 17033

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY RESEARCH SCHOLARSHIP

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

EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

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

University of Chicago, Department of Surgery, Box 255

5841 South Maryland Avenue, Chicago, Illinois 60637

ALFRED BLALOCK RESEARCH SCHOLARSHIP

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

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

Massachusetts General Hospital and Harvard Medical School

Department of Surgery, Boston, Massachusetts 02114

JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

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

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

Duke University Medical Center, Box 31064

ALTON OCHSNER RESEARCH SCHOLARSHIP

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

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

Department of Surgery

Brigham and Women's Hospital

75 Francis Street

Boston, Massachusetts 02115