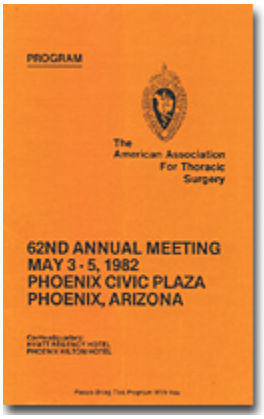
1982 ANNUAL MEETING PROGRAM



THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
1981 -1982

President Thomas B. Ferguson, *St. Louis, MO*
Vice-President Frank C. Spencer, *New York, NY*
Secretary John L. Ochsner, *New Orleans, LA*
Treasurer Paul A. Ebert, *San Francisco, CA*
Editor Dwight C. McGoon, *Rochester, MN*

Council
Donald L. Paulson, (1982), *Dallas, TX*
George J. Magovern, (1982), *Pittsburgh, PA*
Albert Starr, (1983), *Portland, OR*
F. Griffith Pearson, (1984), *Toronto, Ontario*
David B. Skinner, (1985), *Chicago, IL*

Membership Committee
Mortimer J. Buckley, *Chairman, Boston, MA*
Tom R. DeMeester, *Palos Heights, IL*
Donald B. Doty, *Iowa City, Iowa*
Rodney H. Herr, *Boise, Idaho*
Wilbert J. Keon, *Ottawa, Ontario*
William S. Stoney, *Nashville, TN*
Joe R. Utley, *San Diego, CA*

Association Representatives,
Herbert Sloan, *Ann Arbor, MI*

The American Board of Thoracic Surgery
Benson B. Roe, *San Francisco, CA*
Richard J. Cleveland, *Boston, MA*
Philip E. Bernatz, *Rochester, MI*

Board of Governors, American College of Surgeons
John A. Waldhausen, (1982), *Hershey, PA*
Myron W. Wheat, Jr., (1984), *St. Petersburg, FL*

THE AMERICAN ASSOCIATION
FOR THORACIC SURGERY
May 1982 Meeting
COMMITTEES

LOCAL ARRANGEMENTS

William R. Nelson, Chairman
Lee B. Brown
William P. Cornell
Dermont W. Melick
C. Thomas Read

PRESS COMMITTEE

C. Thomas Read

LADIES' COMMITTEE

Mrs. Betty Nelson, Chairman
Mrs. Kay Brown
Mrs. Traci Cornell
Mrs. Dorothy Melick
Mrs. Carol Read

PROGRAM COMMITTEE

Thomas B. Ferguson..... St. Louis, Missouri
Frank C. Spencer..... New York, New York
John L. Ochsner..... New Orleans, Louisiana
Dwight C. McGoon..... Rochester, Minnesota
Martin F. McKneally..... Albany, New York
Ashby C. Moncure..... Boston, Massachusetts
Andrew S. Wechsler..... Durham, North Carolina

Graham Memorial Traveling Fellowship Committee

Chairman, Robert B. Wallace..... Washington, D.C. (1982)
Thomas B. Ferguson..... St. Louis, Missouri (1982)
John L. Ochsner..... New Orleans, Louisiana (1982)
Paul A. Ebert..... San Francisco, California (1982)
Richard K. Hughes..... Los Angeles, California (1983)
Vincent L. Gott..... Baltimore, Maryland (1984)

Ethics Committee

Chairman, Nicholas Kouchoukos..... Birmingham, Alabama
L. Henry Edmunds, Jr..... Philadelphia, Pennsylvania
C. Frederick Kittle..... Chicago, Illinois
W. Spencer Payne..... Rochester, Minnesota
Richad M. Peters..... San Diego, California

American Association for Thoracic Surgery Representatives to: Committee for Coordinating Continuing Education in Thoracic Surgery (CCCETS)

Richard J. Cleveland..... Boston, Massachusetts (1982)
Benson R. Wilcox..... Chapel Hill, North Carolina (1984)
Robert W. Jamplis..... Palo Alto, California (1986)

House of Delegates, American Medical Association
Gilbert S. Campbell..... Little Rock, Arkansas
Thomas G. Baffes..... Skokie, Illinois (Alternate)

National Society for Medical Research
Walter L. Barker..... Chicago, Illinois

Cardiopulmonary Council, American Heart Association
Melvin R. Platt..... Dallas, Texas (AATS Liaison)

Association for the Advancement of Medical Instrumentation
John J. Collins..... Boston, Massachusetts
W. Gerald Rainer..... Denver, Colorado (Alternate)

Extracorporeal Perfusion (AmSect. ABCPT)
Richard E. Clark..... St. Louis, Missouri
George J. Magovern..... Pittsburgh, Pennsylvania
Richard P. Anderson..... Seattle, Washington
Stanton P. Nolan..... Charlottesville, Virginia

Council of Academic Societies (CAS),
Association of American Medical Colleges (AAMC)
Clarence S. Weldon..... St. Louis, Missouri
Judson G. Randolph..... Washington, D.C. (Alternate)

Advisory Council for Cardiothoracic Surgery,
American College of Surgeons
Chairman, Herbert Sloan..... Ann Arbor, Michigan (1983)
Jay L. Ankeney..... Cleveland, Ohio (1983)
F. Henry Ellis, Jr..... Burlington, Massachusetts (1982)
Charles R. Hatcher, Jr..... Atlanta, Georgia (1982)
Quentin R. Stiles..... Los Angeles, California (1982)
John A. Waldhausen..... Hershey, Pennsylvania (1983)
Clarence S. Weldon..... St. Louis, Missouri (1982)
Myron W. Wheat..... St. Petersburg, Florida (1984)
James DeWeese..... Rochester, New York (ex officio)
W. Gerald Austen..... Boston, Massachusetts (1984)

American Association for Thoracic Surgery
Administrative Office
Mr. William T. Maloney, Executive Secretary
13 Elm Street
Manchester, Massachusetts 01944
(617) 927-8330

American Association for Thoracic Surgery
62ND ANNUAL MEETING
Scientific Program

MONDAY MORNING, MAY 3, 1982

8:30 a.m. Business Session (Limited to Members)
Assembly Hall

8:45 a.m. Scientific Session
Assembly Hall

1. Surgical Resection in the Management of Small Cell Carcinoma of the Lung

THOMAS W. SHIELDS, GEORGE A. HIGGINS, JR.,*

MARY J. MATTHEWS and ROBERT J. KEEHN*,*

Chicago, Illinois and Washington, D. C.

To define the role of initial surgical resection in patients with undifferentiated small cell carcinoma of the lung (SCLC), the experience of a cooperative surgical group has been reviewed. One hundred forty-eight patients with SCLS have undergone a microscopically curative resection. This represents 4.7% of "curative" resections carried out in four major prospective adjuvant chemotherapy trials. In the early trials (101 patients), 16 patients (15.8%) died within the first 30 postoperative days. In the last trial, 8 of the patients still alive have been so less than 5 years. These two groups of patients have been excluded from the determinate group. Of the 124 determinate patients, 101 have died of their disease prior to the fifth anniversary of the operation and 23 had survived beyond this point (18.5%). The tumor of each was classified pathologically by the TNM system. The 5-year survival of each category was: T,N_0M_0, 45.8%; T,N,M_0, 28.5%; $T_2N_0M_0$, 20.0%; T;N,Mo, 7.8%; any T, or N_2, 3.5%.

The effect of postoperative adjuvant chemotherapy was evaluated in each of the trials. No beneficial effect of the adjuvant therapy was noted with a one course regimen of a single drug but possible benefit, although not significant, was noted in a prolonged intermittent chemotherapy trial. In the most recent trial of prolonged intermittent courses of two drugs, a two year survival of 80.8% was noted in those receiving adjuvant chemotherapy as compared to a 58.7% in the control group.

It is concluded that resection is indicated in the patients with $T_1N_0M_0$ lesions. Patients with $T_1N_0M_0$ lesions or $T_2N_0M_0$ lesions also probably should be resected. Intensive postoperative chemotherapy appears indicated. Whether or not prophylactic cranial irradiation is indicated has not been addressed. Primary surgical resection is contraindicated in patients with any oilier TNM classification.

*By Invitation

2. Influence of Histology on Survival After Curative Resection for Undifferentiated Lung Cancer

*JOHN E. MAYER, JR. *, STEPHEN L. EWING*,*

JAN OPHOVAN. HATTON W. SUMNER* and*

EDWARD W. HUMPHREY, Keeler AFB,

Mississippi; Minneapolis and St. Paul, Minnesota

Undifferentiated carcinoma of the lung carries a worse prognosis overall than other cell types, but it is unclear whether these tumors represent a homogenous group with uniformly poor survival. This study identifies certain histologic sub-types of large cell (LCC) and small cell (SCC) undifferen-tiated carcinoma which have a better prognosis after curative resection than other similarly treated Undifferentiated carcinomas. From 1950 thru 1975, 2352 patients with lung cancer were admitted to one hospital. Follow-up to death was available in all but 66 patients. Pathologic material was reviewed from 1979 cases by a team of three pathologists during a single six-month period without knowledge of clinical outcome. Curative resection was carried out in 632 with 170 (27%) 5 year survivors. SC cancer occurred in 551 patients and 7 (1.3%) survived 5 years. Curative resections were performed in 33 with polygonal SCC, 19 with negative lymph nodes (LNN) and 14 with positive lymph nodes (LNP). Seven survived 5 years (21%), 6/19 LNN and 1/14 LNP. Twelve with non-polygonal SCC (9 oatcell, 3 fusiform) (6 LNN, 6 LNP) underwent curative resection with no survivors. ($X^2 = 2.94$ $.05 < p < .10$) LCC occurred in 151 and 16 survived 5 years. Curative resection was performed in 22 having LCC with stratification (14 LNN, 8 LNP) and 11 (50%) survived 5 years. In 26 with non-stratified LCC undergoing curative resection (19 LNN, 7 LNP) 5 (19%) survived 5 years ($X^2 = 5.08$ $p<.05$). Thus, patients with resectable polygonal SCC have a better prognosis than those with non-polygonal SCC, and their prognosis approaches that of all patients with resectable lung cancer. Resectable patients having LCC with stratification have a significantly better prognosis than those with non-stratified LCC. Patients with these sub types should therefore not be denied an attempt at curative resection because of the diagnosis of Undifferentiated lung cancer.

*By invitation

3. Survival in Patients Undergoing Tracheal Sleeve Pneumonectomy for Bronchogenic Carcinoma

ROBERT J. JENSIK, L. PENFIELD FABER,

ROBERT W. MILEY, W. CRAIG THATCHER**

and NABIL M.I. ELBAZ. Chicago, Illinois*

and Fort Worth, Texas

Thirty-four patients (1964 through 1981) have undergone tracheal sleeve pneumonectomy for carcinoma of the lung in whom the malignancy has extended proximally to involve the orifice of the stem bronchus or the lateral aspect of the lower trachea.

Simultaneous lung, carinal, and lower tracheal resection with anastomosis between trachea and left main bronchus was performed in 30 patients. In 4 patients pneumonectomy had been done previously and because of recurrence in the stump, (2 on the right and 2 on the left) carinal resection and tracheo-bronchial anastomosis was carried out.

Radiation therapy was administered preoperatively to 28 patients in the series and was considered to be of major benefit in reducing the volume of the tumor to permit the surgical procedure.

Three patients have survived over 10 years and one lived approximately 9 years and 11 months. One is alive over 6 years, and of those dying before the 5-year period, 3 survived between 2 and 3'/2 years and 4 between one and 2 years.

The most common complication was fistula occurring at the anastomosis, developing in 5 of the 11 patients who died in the post-operative period (32% mortality). In all of these radiation had been given preoperatively.

In the last 4 cases, high frequency jet anesthesia was utilized which greatly simplified the anastomosis while maintaining ventilation through a 2 mm. catheter. Although one patient developed a fistula, it was small and successfully controlled by re-operation and application of an intercostal muscle pedicle.

The long-term survival achieved in 5 patients (15%) in whom the future is practically hopeless is justification in carrying out tracheal sleeve resection.

*By invitation

4. Mini-Thoracotomy with Chest Tube Insertion for Children With Empyema

*JOHN G. RAFFENSPERGER, SUSAN R. LUCK**

and RICHARD R. RICKETTS, Chicago, Illinois*

and Atlanta, Georgia

Empyema in children has become a rare disease since the staphylococcal epidemic during the 1950s. However, empyema continues to be an unusual complication of pneumococcal, staphylococcal and hemophilus pneumonia. When the initial pneumonia is inadequately treated with antibiotics, a pleural effusion forms which, if unrecognized, becomes thickened with fibrinous material. An early empyema may be treated with thoracentesis or the closed chest insertion of a chest tube. Either of these procedures are useless if the pus is thick and loculated. Furthermore, the insertion of a chest tube into a crying, stubborn child can be frustrating for the surgeon and painful for the patient.

We now perform a "mini-Thoracotomy" under general anesthesia. The area of suspected empyema is marked on the surface of the chest. We then make a small incision and resect a short segment of rib. The incision is only large enough to insert one or two fingers and a suction tip in order to break up loculations and to remove thick, fibrinous material. The pleural cavity is lavaged with an antibiotic solution and the chest tube is placed under direct vision in a suitable location. The wound is then closed and the tube is placed on suction. With this procedure complete drainage is quickly and easily obtained, the lung expands rapidly, and hospitalization is shortened. We have used this procedure in 15 infants and children during the past 10 years.

10:15 a.m. Intermission - Visit Exhibits

*By invitation

11:00 a.m. Scientific Session
Assembly Hall

5. The Hemodynamic Effects of Protamine Infusion Following Cardiopulmonary Bypass

NA

JEFFREY M. PIEHLER, JOHN C. SILL*,*

ROGER H. WHITE and JAMES R. PLUTH,*

Rochester, Minnesota

Systemic hypotension is commonly observed in association with the administration of Protamine (P) after cardiopulmonary bypass (CPB). Previous studies are inconclusive as to whether P produces its effect on myocardial performance or by changing systemic vascular resistance.

To elucidate the hemodynamic effects of P (150 mg x body surface area), 19 patients were studied following CPB. In one group (n =7) P was infused over 30 sec while in a second group (n = 6) over 60 sec. A third group (n = 6) received $CaCl_2$ (20 mg/kg over 10 min) just prior to P administration. The hemodynamic response was assessed by continuous recording of myocardial contractile element velocity (maximal value = Vpm, Millar transducer), aortic blood flow (electromagnetic flowmeter), systemic and pulmonary arterial and right atrial pressures, and EKO. The response in each group was analyzed by t-test for paired data and the groups were compared by two sample t-tests. Results (mean ± SD) are summarized below:

	30 sec		60 sec/no Ca		60 sec with Ca	
	Control	Response	Control	Response	Control	Response
Vpm (sec^{-1})	13.6 ± 3.6	11.9 ± 2.6	12.2 ± 3.0	11.5 ± 2.9	11.9 ± 2.1	11.1 ± 2.6
Mean BP (mmHg)	82 ± 9	54 ± 18*	77 ± 9	63 ± 11	90 ± 18	79 ± 23*
Cardiac Index ($L/min/m^2$)	3.2 ± .52	3.8 ± .61*	3.4 ± .96	3.7 ± .96*	2.6 ± 1.0	2.9 ± .97*
Systemic Vascular Resistance Index (U/m^2)	6.8 ± 2.7	4. 8 ± 2. 9*	5.5 ± 1.6	4.0 ± 1.5*	11.0 ± 6.1	7.9 ± 4.2

(*P < .05 vs control)

The response to P among the three groups was not different.

These results demonstrate that the P induced hypotension is the result of peripheral vasodilation only partially compensated by an increase in CI, whereas myocardial contractile state remained unchanged. These changes are transient (less than 4 min), unrelated to the rate of administration and not prevented by preinfusion of $CaCl_2$.

**By invitation*

6. Experience with Profound Hypothermia and Circulatory Arrest In the Treatment of Aneurysms of the Aortic Arch

M. ARISAN ERGIN, JAMES O'CONNOR*,*

ROY GUINTO and RANDALL B. CRIEPP,*

Brooklyn, New York

Resection and replacement of the aortic arch represents a demanding technical and tactical challenge to the surgeon. Circulatory arrest during the period of arch exclusion with preservation of central nervous system integrity by profound total body hypothermia renders this procedure simple and safe.

In a series of 19 consecutive patients, the aortic arch and varying portions of ascending and descending aorta, and in some, the aortic valve were replaced utilizing a standard method of profound total body hypothermia. Thirteen patients underwent elective and 6 patients emergency arch replacement. A combination of surface cooling and cardiopulmonary bypass was used to produce total body hypothermia. Replacement of the aortic arch was carried out during a single period of circulatory arrest. Cardiopulmonary bypass was utilized for core rewarming. The average cerebral ischemia time was 39 minutes (range 15-59 minutes) at an average core temperature of 13°C (range 11-18°C). The average myocardial ischemia time was 73 minutes (range 40-110 minutes) with an average duration of cardiopulmonary bypass of 124 minutes (range 85-201 minutes).

Of the 13 patients undergoing elective operation, 2 died; of the 6 patients undergoing emergency operations, 3 died (2 with ruptured aneurysms, 1 with acute arch dissection). Fourteen patients are alive and well 2 months to 7 years following surgery. All are free of neurological sequalae. One has an asymptomatic residual dissection in the descending aorta.

This experience indicates that profound total body hypothermia with cirulatory arrest is a safe and effective method for elective surgical treatment of enlarging aneurysms of the aortic arch and for emergency treatment of acute dissections where the intimal tear is located in the aortic arch. The technique is simple and produces results superior to those reported for methods which involve selective cerebral perfusion during arch replacement.

11:30 a.m. Presidential Address

The Crisis of Excellence
Thomas B. Ferguson

**By invitation*

MONDAY AFTERNOON, May 3, 1982

2:00 p.m. Forum Session
Assembly Hall

7. Improved Myocardial Protection with Decreasing Temperature During Cardioplegic Arrest

F.L. ROSENFELDT and G.R. STIRLING*,*

Victoria, Australia

Sponsored by: David C. Sabiston, Jr., Durham,

North Carolina

The relationship between recovery and temperature during cardioplegic arrest was studied in canine hearts perfused by a support dog. Isolated hearts underwent 2 hours of arrest between -3°C and 37°C using the St. Thomas' Hospital (K+, Mg++, procaine) solution. Isovolumic left ventricular (LV) developed pressure (DP) was measured before and after arrest. LV biopsies were taken for lactate assay before and for ATP assay and electron microscopy after arrest. The results showed ultrastructural damage only at -3°C and a decrease in protection around 15-20°C:

Temp. °C	-3	4	15	20	25	30	37
n	4	6	7	5	8	6	2
DP % control	0	100	98	95	89	60	4
ATP<% control	24	96	106	88	86	60	12
Lactate µM/a	1.5	1.8	3.1	6.6	9.2	13.8	33.3

In a further study arrest was prolonged for 6 hours to bring out differences in protection in the lower temperature range:

Temperature	°C	4 (n = 6)	12 (n = 6)	20 (n = 5)
DP (mmHg):	Reperfusion	103 ± 5	96 ± 5	56 ± 5
ATP µM/g:	Control	3.8 ± 0.2	3.9 ± 0.2	3.6 ± 0.2
	2 hours	3.7 ± 0.2	3.9 ± 0.1	3.2 ± 0.1
	4 hours	3.4 ± 0.2	3.3 ± 0.2	2.4 ± 0.2
	6 hours	2.6 ± 0.2	2.1 ± 0.2	0.9 ± 0.3
	Reperfusion	2.6 ± 0.1	2.1 ± 0.1	1.1 ± 0.2

Values given are mean ± SEM. Analysis of variance was used for statistical comparisons.

ATP was depleted progressively with time in all groups but most rapidly at 20°C. After reperfusion, the 20°C group showed significantly lower biochemical and functional recovery than the other 2 groups ($P<0.001$) and the 12°C group showed lower biochemical recovery than the 4°C group ($P<0.01$).

Conclusion: Protection varies inversely with myocardial temperature and although this effect is most marked down to 20°C there are further increases in protection with cooling to 4°C.

*By invitation

8. Critical Importance of Topical Hypothermia in Cooling Myocardial Regions Supplied by Collateral Flow

JOHN C. LASCHINGER, FRANK P. CATINELLA*,*

JOSEPH N. CUNNINGHAM, JR., PETER X. ADAMS,*

IRA M. NATHAN and FRANK C. SPENCER,*

New York, New York

The importance of profound hypothermia in protecting the myocardium during cardioplegic arrest is well known. However, as is often the case in pts. with severe coronary artery disease, regions of the myocardium distal to critical stenoses supplied solely by small collateral vessels are underperfused and inadequately cooled. The following study was undertaken in a canine model of chronic progressive coronary occlusion to assess the importance of added topical hypothermia in augmenting myocardial cooling in regions of collateral flow.

Dogs (12) had hydroscopic ameroid constrictors placed at the origin of the circumflex coronary artery and mid-left anterior descending coronary artery. Eight weeks later after angiographic demonstration of vessel occlusion and collateral development, myocardial ischemia was confirmed by atrial pacing (180 BPM) and noting ST changes. Animals underwent 60 min. of aortic crossclamping (AXC). Hearts were protected with topical hypothermia (normal saline, temp. 4°C) and blood cardioplegia (temp. 10°C, pH 8.0, KC1 30 mEqs/L, 1 initially and then 500 cc every 30 min.). Subendocardial temperatures in both normal and collateralized myocardial regions were measured with thermistor probes after each injection and following the addition of 1 liter of topical hypothermia (TH).

The results are as follows (all values expressed as mean ± SEM):

		DISTAL LAD		RIGHT
	SEPTUM	REGION	CX REGION	VENTRICLE
Post Injection	12 ± .6°C	15 ± .9°C	13 ± .6°C	13 ± .7°C
Post TH	12 ± .7°C	11 ± .6°C	12 ± .6°C	9 ± .4°C
P Value	NS	<.001	<.025	<.001

These data demonstrate the importance of adjunctive topical hypothermia in cooling regions of the myocardium distal to critical coronary stenoses with poor distribution of cardioplegia solution. Furthermore, cooling those areas of the myocardium with the greatest amount of surface area exposed to ambient temperatures (e.g. anterior right ventricle) will also be improved.
*By invitation

9. Benefits of Normothermic Blood Cardioplegic Induction of Prolonged, Multidose Cold Blood Cardioplegia in Energy Depleted Hearts

ELIOT R. ROSENKRANZ,*

JAKOB VINTEN-JOHANSEN,*

*GERALD D. BUCKBERG and HANNIBAL EDWARDS**

Los Angeles, California

High risk cardiac patients arc more vulnerable to intraopcrative damage because myocardial ATP depletion may be present before aortic clamping and therefore limit myocardial recovery following cardioplegia. To simulate this experimentally, we produced 47%* ATP depletion in dogs by 45 minutes of normothermic ischemic arrest, followed immediately thereafter by 2 more hours of aortic clamping with 4°C multidose blood cardioplegia. We compared the effects of starting the blood cardioplegic infusion at 37 °C (for 5 minutes before cooling it 10 4°C) with those of immediate 4°C blood cardioplegic infusion. The study was designed lo determine if the initial 37 °C blood cardioplegic infusion would enhance energy replenishment by allowing more oxidative metabolism than that possible if the Q_{10} effect of 4°C hypothermia was immediate.

During the *5* minute period of cardioplegic induction, dogs receiving 37°C initial blood cardioplegia had a 3.3cc/100g/min* (421%)* greater oxygen uptake than when 4°C blood cardioplegia was used. Post-ischemic ventricular function recovered only 33%* in 10 control dogs undergoing only 45 minutes of aortic clamping (no cardioplegia). Better functional recovery, to 74%* of control, occurred in 5 dogs undergoing an additional 2 hours of aortic clamping when 4°C multidoes blood cardioplegia was used. In contrast, complete recovery (100%)* at a 42% lower left atrial pressure was seen in the four dogs receiving the same cardioplegia dose, but where the first 5 minutes of cardioplegic infusion was warmed to 37°C before inducing myocardial hypothermia.

We conclude that, in energy depleted hearts, enhancing myocardial oxidative metabolism by giving the initial blood cardioplegia dose at 37°C (before inducing hypothermia) improves the heart's tolerance to the subsequent aortic clamping required for cardiac repair.

*$p < 0.05$ compared with control

*By invitation

10. The Control of Myocardial Ca^{++} Sequestration with Nifedipine/Potassium Cardioplegia

STUART L. BOE, CHARLES M. DIXON*,*

TAMARA A. SAKERT and GEORGE J. MAGOVERN*

Pittsburgh, Pennsylvania

AIM: To determine (a) when and to what extent calcium ion (Ca^{++}) accumulation occurs in the ischemic and reperfused myocardium; (b) Can Ca^{++} uptake be inhibited by a specific Ca^{++} antagonist, Nifedipine? METHOD: Mongrel dogs were placed on total cardiopulmonary bypass at 32°C. Aortic crossclamp (AXC) was applied for 1 hour and 300 cc. of cardio-plegic solution at 4°C was infused al 30 minute intervals. Fifteen minutes after AXC,$^{45}CaCl$ was injected into the femoral artery, and a steady state of $_4$5Ca ‘ ‘ distribution maintained for 130 minutes. Myocardial biopsies were taken after 1 hour of AXC (T_0) and T_5, $T_{,0}$, $T_{,5}$, T_{20}, T_{30} minutes of reperfusion. The source of the $^{45}Ca^{++}$ available to the myocardium was the Serum Ca^{++}expressed as the Specific Activity (SA = [(cpm X 10^4)/1.0 gm]). and myocardial uptake expressed as Relative Specific Activity (RSA ={(SA tissue)/ (SA serum)]). Group 1 (N = 10) received standard cardioplegic solution (SCS). Group II (N = 10) received SCS plus Nifedipine, 20 me/kg. T_0 values represent sequestration of $^{45}Ca^{++}$ in the myocardium via collaterals despite one hour AXC. During all time frames of reperfusion, Nifedipine significantly ($p < .01$) inhibited myocardial uptake. The results show Nifedipine 1) aids maintenance of calcium ion homeostasis during AXC and reperfusion; and 2) blocks calcium influx into the ischemic myocardium during reperfusion. The delay of RSA returning to unity in Group II implies a beneficial effect by inhibiting Ca^{++} transport in early reperfusion, preventing excessive energy consumption by mitochondrial Ca' ‘

sequestration, best illustrated in Group I at T_{20} when Ca^{++} uptake was 5 times normal and confirmed by reduced ATP levels in previous studies. This histochemical sludy provides a rationale for the previously shown improved experimental and clinical hemodynamic function when Nifedipine is added to standard hyperkalemic cardioplegia.

RELATIVE SPECIFIC ACTIVITY

TIME	GROUP I	GROUP II
T_0	0.92 ± .09	0.63 ± .03
T_5	1.74 ± .07	0.67 ± .02
T_{10}	1.92 ± .06	0.69 ± .02
T_{15}	2.30 ± .18	0.75 ± .02
T_{20}	5.15 ± .56	0.78 ± .02
T_{30}	2.17 ± .06	1.31 ± .03

*By invitation

11. Experimental Evaluation of Magnesium Cardioplegia

*AKIO WAKABAYASHI, TSUNEHIRO NISHI**
*AND EDWARD J. GUILMETTE**

Irvine, California

Magnesium is a major active ingredient in all three representative European cardioplegic solutions bul its effectiveness has not been well defined because their data were based on experiments involving multiple components. Therefore, the present study was undertaken to determine the protective effect of magnesium per sc using it as a sole active ingredient. **Methods.** Thirty-six excised rabbits hearts were perfused with oxygenated blood, while the maximal net developed tension (T_{Nmax}) of the isometrically contracting posterior papillary muscle was determined. When T_{Nmax} became stabilized, anoxic normothermic cardioplegia was induced by infusing 10 ml of either control solution (Na+ 135, K+ 5, Cl^-140 mEq/L) or test solution (Mg+ + 140, SO_4^{--} 140 mEq/L) into the aortic cannula. The heart was immersed in a waterbath (37 C) for 30, 45, or 60 min., six experiments in each group. Percent recovery of $T_{N|1},_{.IX}$ was detemined after a full recovery. **Results:** Magnesium solution induced cardiac arrest within one minute which was significantly faster than the control (10.9 ± 1.9 min). The time required to restart the contractions with reperfusion was not significantly different between the control and test groups. After 30 minutes of magnesium anoxia, % recovery was 91.3 ± 8.3, which was significantly ($p<0.01$) better than the controls 77.7 ± 4.1. When the anoxic time was more than 45 min., however, this protective effect of magnesium could not be demonstrated; after 45 min., 65.1 ± 16 (control) vs. 55.1 ± 27.1 (magnesium), after 60 min. anoxia, 4.5 ± 5.1 (control) vs 18.6 ± 11.1 (magnesium). **Conclusion:** Magnesium per se has a protective effect on ischemic myocardium. Percent recovery of myocardial contractility following 30 minutes of normothermic anoxia observed in the present study was very similar to the result achieved by potassium (40 mEq/1) or procaine (0.02%) in our earlier studies. As with these two other active cardioplegic agents, however, the protective effect of magnesium rapidly diminishes as an anoxic time exceeds 30 minutes at normothermia.

*By invitation

12. Adequacy of the Reperfusate: Its Influence in Successful Myocardial Protection

IGNACIO Y. CHRISTLIEB and RICHARD E. CLARK*

St. Louis, Missouri

Previous experiments have indicated that the characteristics of the perfusate at the initiation of myocardial reperfusion after long ischemic inervals (180 min) and long hypothermic (22°C) perfusion times (220 ± 10 min) influenced post ischemic myocardial function despite adequate protection during ischemia. 6 dogs (Group A) were perfused with 5% glucose in Ringer's solution (290 mOsmol) and 6 dogs (Group B) were perfused with 5% glucose-hetastarch solution containing mannitol (osm = 360 ± 20 mOsmol). Priming volumes and oxygenators were identical in the two groups. Perfusion times, and base excess (+ 6 - 8 mEq/L) at the initiation of reperfusion after 3 hr. of global ischemia were equal. Both groups had had identical number and composition of cardioplegic infusions. Complete hemodynamic studies demonstrated that the two groups were equal in terms of cardiac index (CI), left ventricular stroke work index (LVSWI), LVEDP, LV dP/dt and response to volume loading prior to perfusion. 120 min. after cessation of perfusion, the group A dogs had 50% of normal LVSWI (10-12 gm-m/m^2) versus 20-22 gm-m/m^2 for group B dogs. CI's were 1.9 ± 2 vs. 3.6 ± 3 for the A and B groups respectively. Group A dogs had a flat response to volume loading 100 min. after initiation of reperfusion in contrast to group B which had a normal response. Group A required more crystalloid and buffer addition during perfusion. It is concluded that low sodium concentrations, hyperosmolarity and onconicity of the perfusate particularly at the onset of reperfusion significantly influences the myocardial performance immediately and for 2 hrs. after cessation of cardiopulmonary bypass in dogs. These data suggest that the reperfusion phase of cardiac operations is as important as the composition and administration of cardioplegic solutions and may acccount for differences in reports between investigators.

*By invitation

13. Platelet Deposition After Surgically Induced Myocardial Ischemia: An Etiologic Factor for Reperfusion Injury

HAROLD FEINBERG, DAVID ROSENBAUM*,*
*SIDNEY LEVITSKY and NORMAN A. SILVERMAN**
Chicago, Illinois

Despite meticulous adherence to presently known principles of myocardial preservation, reperfusion after aortic cross-clamping results in a unique injury manifested by decreasing high energy phosphate levels and increased coronary resistance. We hypothesize that platelet deposition (PD) into the coronary microvasculature is a major factor in reperfusion injury. To differentiate PD due to subendocardial hemorrhage from deposition to vascular entrapment, ^{111}In-labeled platelets together with ^{51}Cr-labeled erythrocytes were infused in 15 dogs on normothermic bypass and subjected to 60 minutes of global ischemia followed by 30 minutes of reperfusion. PD is indicated only when the proportion of platelets to erythrocytes in tissue exceeds that measured in peripheral blood. Myocardial biopsies were obtained after 120 minutes of continuous bypass (Group I) and at the end of reperfusion after global ischemia (Group II). In 5 dogs (Group III), dipyridamole (1

mg/kg), a potent anti-platelet activation agent, was administered in the pre-ischemic period. PD was expressed as a ratio of ^{111}In before and after ischemia.

Bypass for 120 min. resulted in only a minimal increase in PD. However, normothermic ischemia followed by reperfusion resulted in over a twofold increase in PD compared to controls.. Pre-treatment with dipyridamole appeared to avoid PD. These data indicate that platelet deposition in the coronary microcirculation following surgically induced myocardial ischemia may be associated with reperfusion injury and that anti-platelet drugs alter this sequence.

3:15 p.m. Intermission - Visit Exhibits

*By invitation

4:00 p.m. Scientific Sessions - Assembly Hall

14. A Critical Reassessment of the Performance Characteristics of the Starr-Edwards 6120 Mitral Valve Prosthesis Ten Years Later

D. CRAIG MILLER, PHILIP E. OYER*,*

EDWARD B. STINSON, BRUCE A. REITZ*,*

STUART W. JAMIESON, WILLIAM A. BAUMGARTNER**

and NORMAN E. SHUMWAY Stanford, California

Hesitancy regarding the long-term (10 yr) durability of porcine bioprostheses and the thrombotic complictions associated with tilting-disc mechanical prostheses have prompted resurgence in use of the Starr-Edwards (S-E) metal strut, silastic poppet (Model 6120) mitral valve. Despite 16 years of clinical availability, comprehensive data pertaining to its 10-15 year performance characteristics are unavailable. Therefore, we assessed the long-term results attained with this valve in 509 patients undergoing isolated MVR between 1965-1974. Folow-up totalled 3,157 patient-years (pt-yrs), extended to 15.5 yr maximum, and averaged (±SD) 9.8 ± 3.7 yrs. 124 pts were followed >10 yrs. Specific emphasis was focused on valve-related morbidity and mortality. Anticoagulant-related hemorrhage (ACH) occurred at a linearized rate of 3.8%/pt-yr, thromboembolism (TE) at 5.7%/pt-yr, MVR reoperation (RE-OP) at 1.6%/pt-yr, valve failure (VF; defined by one or more of the following criteria: new murmur of mitral regurgitation, thrombotic or tissue valvular occlusion or multiple TEs leading to reoperation or death, or prosthetic valve endocarditis causing reoperation or death) at 3.5%/pt-yr, and late death at 7.2%/pt-yr. Actuarial rates (± SEM) of being free of these complications were:

	ACH	TE	RE-OP	VF	Late Death
5 years	81 ± 2%	65 ± 2%	93 ± 1%	79 ± 2%	71 ± 2%
10 years	67 ± 3%	55 ± 3%	84 ± 2%	73 ± 2%	47 ± 3%
14 years	52 ± 5%	48 ± 3%	80 ± 3%	69 ± 3%	38 ± 4%

54% (60/111) were caused by multiple TE episodes. Although the intrinsic structural integrity of the S-E 6120 prosthesis is sound for 10-15 yrs, its overall "durability" is clearly suboptimal due to tissue and thrombotic valvular obstruction, thromboemboli, and other factors. Furthermore, when anticoagulant-related hemorrhage is added to each patient's calculated risk equation, the overall incidence of S-E valve-related mobidity at 10 yrs appears to favor tissue bioprostheses even

if 20-30% or more of xenografts were to require replacement by 10 yrs. Only similar large scale comparative analyses of the performance characteristics of xenografts and tilting-dise valves in this time frame will resolve these questions.
*By invitation

15. Early Valve Replacement in Active Infective Endocarditis: Results and Late Survival

RAMON A CUKINGNAN, JOSEPH S. CAREY,*

JOHN H. WITTIG and GEORGE E. CIMOCHOWSKI*,*

Inglewood and Los Angeles, California

In the last 14 years, 42 patients with active infective endocarditis underwent early valve replacements because of severe congestive heart failure, major prosthetic dehiscence, intramyocardial abscesses, or major embolization. Forty patients had positive blood cultures, and in 2 others, the valve tissues were positive. All patients were on antimicrobials for greater than 1 week and under 4 weeks. Drug addiction was noted in 24%, urinary tract manipulation in 7%, dental work in 5%, and unknown cause in 64%. Organisms were predominantly Staphylococcal (43%) and Streptococcal (41%) and 16% were gram negative (9%) or fungal (7%). The aortic valve was involved in 72%, mitral in 14%, tricuspid in 7%, and both aortic and mitral in 7%. By NYHA class, 90% (38/42) were in Class III or IV.

Operative mortality was 10% (4/42), and all had pre-existing renal failure. No predominant organism correlated with early deaths. In aortic valve replacement (30 patients), operative mortality was 7%. Postoperatively, 94% (35/37) are Class I or II with 1 lost to follow-up. Subsequent reoperation was required in 5 patients (13%) for recurrent endocarditis and major valve dehiscence with an operative mortality of 20% (1/5). Late death occurred in 45% (17/38), being 43% for aortic valves, 67% for mitral, 30% for tricuspid, and none for the combined. Major causes of death were sudden death, recurrent endocarditis, and unknown cause. On analyzing early and late mortality, all patients with fungal endocarditis were dead on follow-up. Similarly, Ser-ratia and Pseudomonas infections resulted in a 100% mortality. Staphylococcal endocarditis had a follow-up mortality of 44% compared to 41% for Streptococcal species. Overall survival by life-table method was 0.71 at 1 year and 0.52 at 5 years. Aortic valve endocarditis has a 5-year survival of 0.58.

This study shows that early operation after at least 1 week of antibiotics in patients with active endocarditis has an operative mortality comparable to non-infected valve replacements. Clinical improvement is excellent in 94%. Poor overall late survival may be due to the more virulent fungal, Staphylococcus epidermidis, and gram-negative organisms.

*By invitation

16. Left Ventricular Outflow Enlargement Using the Konno Procedure

GREGORY A. MISBACH, KEVIN TURLEY*,*

DANIEL J. ULLYOT and PAUL A. EBERT

San Francisco, California

The management of patients with small aortic annulus or left ventricular outflow tract obstruction remains unclear. Between 1976 and 1981, 14 patients have undergone enlargement of

their left ventricular outflow tract, using the Konno or a modification of the Konno type procedure. Ten of these 14 patients had previous operations for aortic stenosis or tunnel left ventricular outflow tract and two patients had undergone three previous operations. All 14 patients had symptoms of either heart failure or chest pain or had electrocardiograph evidence of strain. They ranged in age from 5 years to 57 years, with 12 of the 14 patients being less than 25 years of age.

A dacron patch was used to enlarge the left ventricular outflow tract after incising down the ventricular septum. In all patients, at least a 23mm valve was able to be placed with between 50 and 65 percent of the valve annulus being made up of natural tissue. The remaining portion of the valve annulus was constructed from the dacron patch. The patch was extended up the aorta to enlarge the ascending aorta and a pericardial patch was used to close the defect in the right ventricular outflow tract. In all 14 patients the gradient was obliterated at the time of operation. There were no operative deaths, but there has been one late death due to bacterial endocarditis in a child who also had a parachute mitral valve and evidence of pulmonary hypertension. The remaining 13 patients are functioning well after the Konno procedure. Two are receiving Coumadin, and 11 are receiving aspirin. These results suggest that this is an acceptable method of treating patients with small aortic annulus or left ventricular outflow tract obstructions and would appear to have advantages over a left ventricular apical aortic conduit.

*By invitation

17. The Surgical Treatment of Ventricular Tachycardias (Simple Aneurysmectomy Versus Electrophysiologically Guided Surgery)

JORG OSTERMEYER, RALF KOLVENBACK*,*

GUNTER BREITHARDT, LUDGER SEIPEL*,*

HAGEN D. SCHULTE and WOLFGANG BIRCKS*

Dusseldorf, West Germany

Sponsored by: JOHN W. KIRKLIN,

Birmingham, Alabama

Between 1975 and 1981 35 patients had surgery for life threatening recurrent ventricular tachycardia (VT) resistant to antiarrhythmic drug therapy. All except of 3 had severe coronary artery disease with a history of myocardial infarction. In 10 patients (group I) a simple aneurysmectomy was done, 25 (group II) had an electrophysiologically guided (epicardial/endocardial mapping) encircling endocardia! ventriculotomy at the earliest electrical activity during VT. There were no significant differences between the groups in age, sex ratio, NYHA class, coronary disease, aneurysm location, concom-mittant bypass grafting and left ventricular function.

All patients have been restudied by means of 24h-ECG-monitoring, programmed stimulation and/or the ECG-signal-averaging-technique 2-4weeks after surgery. The mean follow-up period in group I is 3,8 years, in group II 22,8 weeks. In group I we had 1 early and 4 late deaths after surgery (50%mortality) mainly due to persistent arrhythmias. 1 patient is free of VT (10%), 3 require still antiarrhythmics, 1 patient's post-operative history is unknown. Out of group II we lost 1 patient intraoperatively, there were no late deaths (4% mortality). 19 patients (76%) are free of VT clinically, they have no persisting late potentials or signs of ventricular irritability at programmed stimulation. 5 patients are under antiarrhythmic drugs, 1 patient has an antiarrhythmic pacemaker.

We conclude that an electrophysiologically guided approach improves the efficacy of the surgical treatment of VT significantly in comparison to simple, "blind" aneurysmectomy.

*By invitation

18. Changes in Indications for Heart Transplantation, An Additional Argument For the Preservation of the Recipient's Diseased Heart

JACQUES G. LOSMAN, HARRY D. LEVINE*,*

CHARLES D. CAMPBELL, ROBERT L. REPLOGLE*

*and CHRISTIAAN N. BARNARD**

Chicago, Illinois and Cape Town, South Africa

Heart transplantation (HT) one year (y) survival improved since 1967 from ±30% to ±70%, 5 y survival is now ±50%. This brought renewed interest in HT, now done in about 20 centers in 5 countries, and increased confidence has widened the indication to patients (pt) less than terminally ill, to restore quality of life. This trend is illustrated by the Cape Town series, when 1967-74 pt [CT_1 = 10 orthotopic HT (OHT)] are compared to 1974-78 pt [CT_2= 20 heterotopic HT (HHT)]. CT_2 pt are younger, m = 38.2±2.2 y than CT_1, pt, m = 51.1±2.7 y [$p<0.001$], CT_2 illness is shorter, m = 3.6 ± 0.7 y, versus CT1 m = 6.6±1.4 y [$p<0.019$], and CT_2 NYHA Class is less, m = 3.45 ± 0.11 versus CT_1, m = 3.9 ± 1.0, [$p<0.006$]. The improved survival is linked to pt selection, progress in management and switch to HHT, but not to progress in matching between donor (d) and recipient (r). Since there is no means to predict tolerance of the d heart (dh), HHT limits the risks from unforseeable mismatch. The retained r heart (rh) is a *build-in assist device,* life saving when dh fails acutely at surgery, during acute [3 pt] or chronic [2 pt] rejection. Had these pt been OHT r, they would have died. Comparing the 10 oldest HHT r with CT_1, no difference in pre-HT parameters was found. However, HHT r survival was longer during the critical post-HT period: at 3 months $p<0.011$, at 6 months $p<0.05$. Larger series will separate effects of management progress from HHT intrinsic advantages. Retaining the rh is logical, and brought few complications. Survival of 38 HHT r was 74% at 6, 67% at 12 and 51% at 36 months, 85% of survivors are in NYHA Class 1. In pt less than desperate, but who refuse to remain cripples, HHT eliminates the growing ethical problem of removing a rh that may still support its owner.

*By invitation

TUESDAY MORNING, MAY 4, 1982

8:30 a.m. Scientific Session
Assembly Hall

19. High Frequency Ventilation with A Single Lumen Tube For Intrathoracic Surgery

NABIL EL-BAZ, WAYNEH. WELSHER*,*

ABDEL EL-GANZOURI. ANTHONY IVANKOVICH*,*

C. FREDERICK KITTLE, L. PENFIELD FABER

and ROBERT J. JENSIK

Chicago, Illinois

One lung anesthesia has proved a valuable adjunct for many intrathoracic operations. The use of a single lumen (8 mm ID) long (35 cm) cuffed endo-bronchial tube placed in the main bronchus of the dependent lung with high frequency ventilation (HFV-60 to 600/minute) has been studied in 29 patients. These have had a variety of procedures (segmentectomy, lob-ectomy, pneumonectomy, and esophagectomy). The tube was placed using the fiberoptic bronchoscope. HFV at a frequency of 250/minute with an inspriatory time percentage of 40%, a driving gas pressure of 20 psi, and 100% O^2 were used. Arterial blood gases were measured at 10 minute intervals.

All patients maintained a PaO_2 of 150-500 mm Hg and a $PaCO_2$ of 20-50 mm Hg without appreciable pH changes.

The single lumen smaller tube avoids the occasional traumatic complications associated with a double lumen tube and is more easily placed. HFV maintains full expansion of the dependent lung without mediastinal shift and with adequate oxygenation. The collapsed atelectatic lung facilitates dissection for various procedures.

*By invitation

20. Exsanguinating Hemoptysis

*ANTONIO A. GARZON, MARCIAL M. CERRUTI**

*and MICHAEL R. GOLDING**

Stolen Island and Brooklyn, New York

Massive hemoptysis (600 ml/24h) treated conservatively carries a mortality of more than 50%. We have performed 74 pulmonary resections in patients with massive hemoptysis in the last fifteen years with a mortality of less than 15%. The mortality rate correlated with the speed and the amount of recorded blood lost before the operation. Those patients that required single lung anesthesia to control aspiration during operation had a mortality that was almost double the total group (25%).

From our experience, we have identified a sub-group of patients with such large hemoptysis that life was threatened by exsanguination. Twenty-four of our patients bled more than 1000 ml. and at a rate of at least 150 ml/h. before the pulmonary resection was performed. The bleeding site was always identified by bronchoscopy. Several methods were used to avoid the patient drowning during the operation. In four patients, double lumen endotracheal tube was used; two died of suffocation during the procedure. In eight patients, a single lung ventilation with endotracheal tube in the nonbleeding lung was used; two of them died of hypoxia and respiratory failure due to aspiration. In another ten patients, a bronchial blocker (#10 Fogarty balloon catheter) was used to stop the bleeding; two died of renal failure and G.I. bleeding but none had aspiration problems.

Our experience indicates that blocking of the bleeding bronchus under direct vision is the safer and more effective procedure to control aspiration and bleeding in massive hemoptysis. Thoracic surgeons managing massive hemoptysis must be familiar with the techniques to control bleeding under these difficult circumstances. Since massive pulmonary bleeding is not common, we believe the presentation of our experience and technique will be valuable.

*By invitation

21. Surgical Treatment of Pleural Mesothelioma

PATRICIA M. McCORMACK, FUMINO NAGASAKI*,*

BASIL S. HILARIS and NAEL MARTINI*

New York, New York

From 1939 through 1980, 155 patients were seen and treated for pleural mesothelioma. 19 of these were benign, and were treated by resection without recurrence. 136 were malignant tumors, 20% (27/136) were fibrosar-comatous and were resected with clear surgical margins where possible and with additional postoperative radiation therapy where adequate margins could not be attained. 80% (109/136) were diffuse malignant epithelial mesothelioma with involvement of parietal and visceral pleura as well as pericardia! and diaphragmatic surfaces.

Surgery, radiation therapy and chemotherapy have been used singly or in combination in the treatment of malignant mesothelioma. In fibro-sarcomatous mesothelioma, complete resection is possible, and offers significant chance of long survival. In epithelial mesothelioma where the disease is diffuse and often associated with effusion, complete resection is not possible. In an earlier report by us on 39 patients, better survival was obtained where pleurectomy, with resection of bulky disease, was combined with external radiation therapy and chemotherapy. No benefit was noted from sacrificing pulmonary tissue.

We have since seen and treated in a similar manner 70 additional patients with malignant epithelial mesothelioma. We have continued to preserve functioning lung tissue by performing a subtotal pleurectomy, but have since 1977 modified our technique of delivering radiation therapy by combining interstitial and external irradiation.

All patients with disease confined to one hemithorax are first surgically explored. A pleurectomy and pericardiectomy are carried out to remove as much tumor as possible. Bulky, non-resectable masses are implanted with radio-iodine sources. The superior mediastinum and the diaphragm, areas of high risk for recurrence, are treated with a removable Iridium-192 implant. Postoperatively, external radiation therapy is given in a technique that allows treatment of the entire pleural surface while sparing underlying lung and spinal cord. 32 patients have been treated in this manner from January 1977 to September 1981. 25 patients had malignant epithelial mesotheliomas, 4 had fibrosarcomatous generalized tumors and 3 were of the mixed type. There was no operative mortality. All the patients were able to complete the radiation therapy with minimal morbidity. Chemotherapy was initiated upon completion of the radiotherapy. The median survival of this group of patients is currently 17.3 months with a range of follow-up of 2-34 months. Survival with radiation therapy and chemotherapy without surgery is considerably less in most reported series.

Details of the surgical and radiation techniques involved in the treatment of these patients will be presented.

9:30 a.m. Intermission - Visit Exhibits

*By invitation

10:15 a.m. Scientific Session - Assembly Hall

22. Use of Operative Transluminal Coronary Angioplasty as an Adjunct to Coronary Artery Bypass

EUGENE WALLSH, ANDREW J. FRANZONE,*

ANDRE CLAVEL, GERALD S. WEINSTEIN**

*and SIMON H. STERTZER**

New York, New York

Intraoperative transluminal coronary angioplasly (OTCA) was used lo improve cornonary ariery bypass graft (CABG) runoff in patients having complex segmcntal and diffuse coronary artery obstructions.

OTCA was performed during CABG through the bypass arteriotomy on 63 arteries in 58 patients, employing an angioplasty system specifically designed for intraoperative use.

Elective restudy was performed on 29 dilated arteries in 27 patients: 21 between 8 and 21 days (mean 16) and 8 between 4 and 31 months (mean 21.4). Overall patency was 83% (16/21 studied early, 8/8 studied late).

Three perforations (4.8%) occurred and were repaired without sequelae. One operative death (1.7%) occurred in a patient with preoperativc refractory cardiogenic shock. There were 7 pcrio-operative infarctions (12.1%), of which 3 were in the distribution of the coronary artery undergoing OTCA.

We conclude that OTCA is a useful adjunct in the operative treatment of patients with complex proximal and diffuse distal coronary obstruction.

*By invitation

23. Coronary Athero-Embolism Causes Peri-Operative Myocardial Infaction - A Hazard at Reoperation

*WILBERT J. KEON and H. ALEXANDER HEGGTVEIT**

Ottawa, Ontario

We have demonstrated a phenomenon occurring during coronary artery bypass grafting (CABG) which has not been previously described. Thirteen instances of fatal peri-operative myocardial infarction following CABG were associated with intra-operative atheromatous embolization in the coronary microcirculation. In five cases the emboli originated from ulcerative atherosclerotic lesions in the aortic root at the site of the vein graft ostia, two cases likely emanated from coronary endarterectomy sites and two cases from mechanical disruption of plaques in the major epicardial coronary arteries during surgery. The above 9 cases occurred during initial revascularization procedures. We have performed 4,095 initial cases of CABG and this represents a risk of 0.22%. A further 4 cases occurred during repeat CABG procedures and resulted from manipulative disruption of atheroma in old vein grafts. Our total number of CABG's is 175; a 2.29% risk at reoperation representing a tenfold increase in risk for this complication at reoperation. Inadequate histological sampling of the myocardium at autopsy will necessarily result in underestimation of the incidence of this phenomenon.

Analysis of angiograms prior to repeat CABG can identify patients at increased risk who have severe graft atherosclerosis as opposed to myointimal hyperplasia. To reduce the incidence of atheroembolism at reoperation we strongly advocate ligation of the vein graft at the level of the distal anastomosis as early as possible during dissection reopening the chest.

*By invitation

24. Idiopathic Hypertrophic Subaortic Stenosis and Coronary Atherosclerosis; Results of Coronary Artery Bypass Alone and Myomectomy Combined with Coronary Artery Bypass Surgery

CARL C. GILL, ANDREW M. DUDA*,*

HIDEMASA KITAZUME, JOHN R. KRAMER**

and FLOYD D. LOOP Cleveland, Ohio

Twenty-one patients with combined coronary artery disease (CAD) and idiopathic hypertrophic subaortic stenoses (1HSS) have had coronary artery bypass grafting (CABG) alone (group I, n = 7) or combined with ventricular septal myomectomy (LVM) (group II, n = 14). There were no operative deaths. Follow-up is current for all patients. Patient data for both groups is summarized below:

	Mean age	Sex M	Sex F	Multivessel CAD (%)	Peak pre-op subvalvular gradient (mmHg.)	Grafts/ patient	Mean follow-up (months)
Group							
1	56.1	5	2	64	69.1	2.3	82
(n = 7)	± 6.1				± 19	± 76	
Group							
11	60.6	9	5	85	83.9	2.0	21
(n = 14)	± 8.3				± 39.6	± 1.2	

In group I there has been one sudden death 16 months postoperatively; five other patients in group I have undergone recatheterization. The subvalvular gradient was unchanged in all except one, who experienced graft occlusion and inferior myocardial infarction; he now has no subvalvular gradient 7 years after initial surgery. Another patient in group 1 had progressive angina and mitral insufficiency requiring mitral valve replacement 25 months after initial CABG. The functional class of the 5 other group 1 patients is unchanged or worse after CABG alone. In contrast group 11 patients are markedly improved (12 NYHA class 1? 1 NYHA class 11) except one patient who experienced recurrent angina pectoris 16 months postoperatively. CABG alone is ineffective in relieving symptoms in patients with IHSS and CAD. LVM combined with CABG provides safe and effective relief of symptoms for these difficult patients.

11:30 a.m. Address of Honored Speaker

Achievements in The Study and Control of Cancer of The Esophagus - 1940 - 1980

WU YING-KAI Beijing, China

12:15 p.m. Cardiothoracic Residents Luncheon - Flagstaff Room

*By invitation

TUESDAY AFTERNOON, MAY 4, 1982

2:00 p.m. Scientific Session
Assembly Hall

25. Pulmonary Outflow Tract Reconstruction Without Prosthetic Conduit

YVES LECOMPTE, JEAN-YVES NEVEUX*,*

FRANCINE LECA, LUCIO ZANNINI**

*and YVETTE DUBOYS**

Paris, France

Sponsored by: ALBERT STARR, Portland, Oregon

The use of prosthetic conduits in the reconstruction of the pulmonary outflow tract in infants or small children is a palliative procedure associated with well known drawbacks.

Our previously reported experience of 9 cases of anatomical correction of transposition of the great arteries (T.G.A.) by positioning the pulmonary bifurcation anterior to the ascending aorta stimulated us to extend this concept to correct other complex congenital anomalies: T.G.A. with ventricular septal defect (V.S.D.) and pulmonary stenosis (P.S.), truncus arteriosus, and pulmonary atresia with V.S.D.

In T.G.A. with V.S.D. and P.S., the technique comprised the resection of the conal seplum, the suturing of an interventricular patch establishing continuity between the left ventricle and the aorta, and the direct implantation of the pulmonary artery on the right ventricle after positioning the pulmonary bifurcation anterior to the aorta. This technique was feasible even in small infants and cases of small V.S.D. Eleven patients, from four months to six years of age were corrected using this technique, with four deaths and seven good short term results (3 months to 1 year).

In truncus arlcriosus, anatomical correction was achieved by direct implantation of the pulmonary artery on the right ventricle after positioning the pulmonary bifurcation anterior 10 the ascending aorta. 4 children aged from 4 months to 5 years were operated upon using this technique with 2 deaths probably due to severe pulmonary regurgitation, a complication which should be prevented in future cases by systematic implantation of a mono-cusp valve.

In pulmonary atresia with V.S.D. and absent pulmonary trunk, the continuity between the right ventricle and the pulmonary branches was established by using an arterial tube resected from the ascending aorta. This technique was successfully used in one child with extremely small pulmonary branches.

These preliminary results led us to conclude that many complex congenital cardiac anomalies can be effectively treated without prosthetic conduit.

*By invitation

26. Use of Sub-Pulmonary "Ventricular" Chamber in the Fontan Operation

MARC ROGER DE LEVAL, CATHERINE BULL*,*
JAROSLAV STARK and FERGUS MACARTNEY**
London, England

Sponsored by: DWIGHT C. McGOON, Rochester, Minnesota

In order to assess the value of incorporation of a sub-pulmonary "ventricular" chamber into a Fontan type repair, the haemodynamics of 13 patients (pts) with this type of repair were compared with 17 pts who had an atrio-pulmonary connection. The methods included analysis of pressure tracings (all pts) and relation of stroke work to filling pressures at completion of the repair (9 pts) detection of valve closure and opening by two-dimensional and pulsed Doppler echocardiography (10 pts) and postoperative cardiac catheterization (8 pts). Immediately post-operatively, mean right atrial (RA pressure was equal to or higher than mean pulmonary artery (PA) pressure in all pts, regardless of the incorporation of a sub-pulmonary "ventricular" chamber. The pressure difference increased as filling pressure was increased. Thus while the Starling curve for the left ventricle had a positive slope, that for the right heart had a negative one. There was an ‘a' wave in the PA pressure trace indicating an atrial dependent circulation in all pts but one. The exception was in a pt with an atrio-ventricular conduit but residual pulmonary stenosis who died in low cardiac output. In all pts with an atrio-ventricular connection, ventricular systole opened the pulmonary valve, produced a V wave in the PA pressure, and closed the conduit valve. By contrast, the conduit valve was observed to close in only one patient with an atrio-pulmonary conduit.

In one pt with an atrio-ventricular connection re-studied a year after repair, the RA pressure was normal and lower than PA pressure. The ‘a' wave in his PA pressure had disappeared indicating a ventricular dependent circulation, resulting from ventricular growth.

We conclude that immediately post-operatively the right heart is functioning more as a conduit for a circulation mainly driven by the left ventricle than as a pumping chamber assisting it. The main benefit of an atrio-ventricular connection is that it introduces a functioning conduit valve into the right heart though this benefit is outweighed if its insertion results in an obstructed pathway from RA to PA and compromises the necessary RA contribution to pulmonary blood flow. Post-operative right ventricular growth can convert an atrial dependent circulation into a ‘normal' ventricular dependent one.

*By invitation

27. Repair of Tricuspid Atresia in 100 Patients

FRANCIS M. FONTAN, CLAUDE DEVILLE,*
JAN QUAEGEBEUR, JAAP OTTENKAMP*,*
ALAIN CHOUSSAT and GERARD A. BROM*
Bordeaux, France and Leiden, The Netherlands

From April 1968 to September 1981, 100 patients (pts) had surgical repair of tricuspid atresia (T.A.), at a mean age of 9 years 5 months (extreme 20 months to 36 years). 72 procedures of palliative surgery had been previously performed in 62 pts. In pts with ventriculo-arterial concordance (VAC), a non valved Dacron conduit or an aortic valve homograft (AVH) was interposed between right atrium (R.A.) and right ventricular outlet chamber; in 32 pts with ventriculo-arterial discordance (VAD) AVH established the continuity between R.A. and pulmonary arteries (P.A.); in addition to closure of atrial septal defect in all pts, and of ventricular

septal defect when necessary, in VAC, other procedures were performed occasionally: pulmonary valve commissurotomy (2 pts), P.A. trunk enlargement (2 pts) mitral valve replacement (1 pt), inferior vena cava valvulation with A.V.H. (8 pts). The hospital mortality for the entire group was 12%, 7% in VAC (5 pts), 21.8% in VAD (7 pts), mainly due to P.A. hypertension (5 pts); from 1974 to 1981, hospital mortality was strikingly reduced in 92 pts (8 deaths, 8.7%, CL 6%-13%). There were 5 late deaths due to septicemia (1), heart failure (2), sudden death (1), reoperation for residual shunt (1). Reoperation was performed in 10 pts for residual shunts (6) dacron conduit obstruction (1) heart failure (2) chylothorax (1) with 2 deaths; there was no reoperation in the last 33 pts. Actuarial survival rate at 10 years is 85%. Late results are good in the majority of the pts. Control cardiac catheterization was performed in 46 pts from 1 month to 6 years postoperatively: mean R.A. pressure was 16 ± 4 mm Hg, no significant systolic pressure gradient was found between R.A. and P.A.; in pts with AVH between R.A. and outlet chamber, peak systolic pressure were higher in outlet chamber and P.A. than in R.A.; arterial oxygen saturation was 92.2% ± 5, mean left ventricular ejection fraction was 62%; enlargement of outlet chamber was noted in VAC. Exercise tests were performed in 20 pts; the performance was between 60% and 100% of normal in 13 pts, less than 50% in 2, and better in pts with AVH conduit. This review suggests that current immediate and long term results are satisfactory and permits to consider with confidence surgical repair of T.A.

3:00 p.m. Intermission - Visit Exhibits

*By invitation

3:45 p.m. Scientific Session - Assembly Hall

28. Repair of A-V Canal Malformation in the 1st Year of Life

HARVEY W. BENDER, JOHN W. MAMMON,*

STEVE G. HUBBARD, JAN MUIRHEAD**

*and THOMAS P. GRAHAM**

Nashville, Tennessee

Disappointing results with pulmonary artery banding and subsequent correction led to the decision in 1977 that all infants presenting to our hospital with A-V canal and evidence of severe heart failure, lack of growth, or of pulmonary hypertension should receive early operative correction. Since that time, twenty-five consecutive infants have been operated. All had refractory heart failure. Average age at operation was 18 (3-38) weeks and average weight was 4.4 (2.3-8.8) kg. Only two patients were greater than six months of age at operation. Preoperative peak pulmonary artery pressure was 81 ± 3.3 mm Hg which was equal to systemic arterial pressure in all cases. Mean pulmonary-systemic resistance ratio was .28 ± .05. Fifteen patients had greater than mild mitral regurgitation and five had a patent ductus arteriosus. Three had significant associated malformations. Profound hypothermia and circulatory arrest was utilized in all patients. Common atrioventricular valve tissue was divided and valvular integrity was insured by resuspension to a single dacron patch which closed both the atrial and ventricular defects. Operative mortality occurred in two patients (8%) both with associated defects (1-TAPVC, 1-coarctation). One patient required a permanent pacemaker for surgical heart block. Late mortality occurred in one patient with associated PAPVC, and one patient has had a pacemaker implanted. Survivors have been followed for an average 23 (7-47) months. All patients have returned to normal growth and development postoperatively. All cardiac medications have been discontinued in 14 of 22 patients.

Operative repair of complete A-V canal can be performed in infancy with low operative and late mortality and will relieve signs and symptoms of heart failure and allow infants to experience more normal growth and development. On the basis of this experience, it appears to be unnecessary to delay operative correction with the known increased risk of the development of pulmonary hypertension.

*By invitation

29. The A-V Canal Defects: Relation of Morphology, Function, and Surgical Technique To Early and Late Results

MARKUS R. STUDER, EUGENE H. BLACKSTONE*,*

JOHN W. KIRKLIN, ALBERTO. PACIFICO,

BENINGO SOTO, GEORGE K. T. CHUNG**
*and LIONEL M. BARGERON, JR.**

Birmingham, Alabama

For the 271 patients with A-V canal defects operated upon by us between 1967-1981, and while blinded to knowledge of the patient outcome, we have re-analyzed morphology with a modification of Anderson's concepts, applied this and an evaluation of ventricular dominance and left-sided A-V valve function to re-analysis of the cineangiograms, and related these and the evolving surgical technique to in-hospital death (47 patients, 17%) and followup results (15 late deaths, actuarial survival of hospital survivors 95.3"% at 12 months and 91% at 10 years; 16 reoperations, actuarial incidence 95% at 12 months, 92% at 10 years; and a minimum of 23 patients, 9%, with important P.O. left A-V valve incompetence; categories not mutually exclusive). Heart block developed after repair in 6 patients (2.2%) of whom 2 had no interventricular communication and had left A-V valve replacement and one had multiple VSD's. Variables relating to results are in the table.

Variable	Hospital Mortality (47)	Documented Valve Dehiscence (12)	Reoperation (16)	Important P.O. Left A-V Valve Incompetence (23)	Late Death (15)
Morphologically Unique malforma- tion (80)	26% >p=0.01†	-	-	-	-
Major associated defect (TF, DORV, TGA) (29)	44% >p=0.002	-	-	-	-
Interventricular communication (VC) (128)	31% >p<0.001	-	-	-	>p<0.05
Severe R or L ventricular dominace (23)	57% >p=0.0001	-	-	-	-
Preoperatively severe left A-V valve	-	>p=0.1	>p=0.03	.p=0.003	-

incompetence (LAVI) (48)					
Age (continuous variable)	>in very young p<=0.0001	>in very young p<=0.01	>in very young p<=0.05	>in very young P<=0.05	>in very young p<=0.0001
Clinical status (continuous variable)	> in higher NYHA class p<0.05†	- -	- -	> in higher NYHA class p<0.05†	> in higher NYHA class p<0.05

Note: Columns are not mutually exclusive. Numbers in ( ) refer to patients in the category

- No effect

† Dropped out of multivariate analysis

> increased risk compared with remainder of the 271 patients

TF=Tetralogy of Fallot

DORV=Double outlet right ventricle

TGA=Transposition of great arteries

Continuously evolving surgical technique (altered suture siting to more securely avoid A-V node and His bundle; "3-leaflet" concept of the left A-V valve; 2 patches when VC present, sandwiching A-V valve between them; functional valve analysis at operation and addition of annuloplasty) have decreased hospital mortality with time ($p<0.05$), decreased the incidence of valve dehiscence ($p<0.05$), but have not decreased the overall incidence of important postoperative LAVI in patients with important preoperative LAVI (the latter present in 21, 15%, of 143 patients without VC and in 27, 22%,of 125 with VC).

*By invitation

30. Double Outlet Right Ventricle - Surgical Results 1970-1980

JOHN P. JUDSON, GORDON K. DANIELSON,*

*FRANCISCO J. PUGA * and DWIGHT C. McGOON*

Lubbock, Texas and Rochester, Minnesota

Between January 1, 1970, and January 1, 1980, 62 consecutive patients who underwent repair of double outlet right ventricle were reviewed. Patients with subpulmonary ventricular septal defect (Taussig-Bing complex), complete atrioventricular canal, atrioventricular discordance, and univentricular heart were excluded. Associated defects in 54 patients included restrictive ventricular septal defect, multiple ventricular septal defects, two-chambered right ventricle, pulmonary atresia, atrial septal defect, coronary artery anomalies, bilateral superior venae cavae, juxtaposed atrial appendages, patent ductus arteriosus, dextrocardia, right aortic arch, subaortic stenosis, tricuspid regurgitation, and stradding of the mitral valve. There were 46 patients with pulmonary stenosis and 16 patients without pulmonary stenosis; of these, 36 were male and 26 female. Age at operation ranged from 8 months to 37 years (mean 9.0 years). The operative mortality was 5 (11%) in cases with pulmonary stenosis, 4 (25%) in cases without pulmonary stenosis, and 9 (14.5%) overall. Mortality was often related to associated anomalies. Causes of death included low cardiac output, infection, pulmonary hypertension, and technical problems (2

patients each), and right heart failure (1 patient). During the follow-up period of 6 months to 8 years, the late mortality was 17% in cases with pulmonary stenosis, 19% in cases without pulmonary stenosis, and 18% overall. Two late deaths were of noncardiac causes, but the remainder occurred 2 months to 6'/2 years postoperatively from arrhythmias. Late results in the majority of survivors are good to excellent. While the operative mortality for double outlet right ventricle continues to decrease, the late mortality is of concern. The problem of late arrhythmias requires further study and analysis.
*By invitation

31. Surgery for Congenital Valvular Aortic Stenosis: A Twenty-Three Year Experience

*JAY L. ANKENEY, THOMAS S. TZENG**

*and JEROME LIEBMAN**

Cleveland, Ohio

From 1958 through 1980, 70 consecutive patients, 2 to 20 years of age, were operated upon for congenital valvular aortic stenosis. The primary indication for surgery was development of symptoms in 53 patients - decreased exercise tolerance (22), chest pain (21), syncope (5) and SOB)5), ST and T abnormality in the electrocardiogram (ECG) in 13 and progressive LVH in 4. Pre-operative catheterization in 57 patients, in which the left ventricle was entered, showed an average left ventricular-aortic (LV-AO) systolic gradient of 85.3 mm. Hg. (± 36.3 S.D.). Only 9 patients had a gradient less than 50 mm. Hg.

Valvulotomy was performed in all patients, resulting in a bicuspid valve in 36 and a tricuspid valve in 33. In the 3-leaflet group, vestigial commissures were opened in 17 and a third leaflet was created in 16 by incising a common leaflet and using an everting suture near the ring to prevent aortic regurgitation. Twelve patients had insignificant diastolic murmurs in the immediate postoperative period. There were 2 operative deaths (2.8%); one due to technical failure of the valvulotomy, necessitating attempted valve insertion, and the other after sudden cardiac arrest on the third postoperative day.

All 68 surviving patients have been followed from 1 to 23 years (mean 10.5). Postoperative catheterization in 38 patients showed an average LV-AO systolic gradient of 38.7 mm. Hg. (± 27.2 S.D.). The patients' postoperative ECG showed improvement in 39 and deterioration in 7. There were 4 late deaths: one was a consequence of SEE and 3 were non-cardiac in origin - drowning, car accident, and neuroblastoma. Eleven patients underwent reoperation 2 to 22 years postoperatively (mean 10.5). Indications for re-operation were recurrent stenosis in 9, dissecting aneurysm in 1, and aortic regurgitation in 1. In 4, a second valvulotomy was done, and in 7 the valve was replaced. There were 2 operative deaths, both occurring in patients undergoing emergency reoperation: one for cardiogenic shock, and the other for acute dissecting aorta aneurysm. Another died suddenly 9years after the second operation. Of the 53 patients surviving the initial valvulotomy without requiring reoperation 49 are Class I and 4 are Class II. Only 2 patients receive digitalis; one for mild aortic regurgitation and the other for supraventricular arrhythmia.

Our experience indicates that aortic valvulotmy is an effective, palliative operation for congenital valvular aortic stenosis in children, and valve replacement, if necessary, can be delayed for many years.

4:45 p.m. Executive Session - Assembly Hall

7:00 p.m. President's Reception
Heard Museum

*By invitation

WEDNESDAY MORNING, MAY 5, 1982

8:30 a.m. Forum Session
Assembly Hall

32. What is the Relationship Between Plasma Heparin Concentration and (ACT) Activated Clotting Time?

RICK A. ESPOSITO, ALFRED T. CULLIFORD*,*

STEPHEN B. COLVIN, STEPHEN T. THOMAS*,*

SANFORD N. GITEL and FRANK C. SPENCER*

New York, New York

The ACT has gained popularity as a means of assessing the adequacy of anticoagulation during cardiopulmonary bypass (CPB). However, neither the precise correlation of the ACT to the plasma heparin level nor the desired therapeutic level are known. We investigated these relationships in 40 patients by measuring serial ACTs, plasma heparin levels, antithrombin III, and routine coagulation parameters. Before CPB dose-response curves were constructed with serial doses of 2 and 4 mg/kg of heparin. CPB was started when the ACT > 400 seconds, which occured in only 47% of patients after 4 mg/kg. The ACT-heparin relationhip varied to an astonishing degree (2 mg/kg: ACT 139.8 ± 43 sec, 4 mg/kg: 276 ± 91.5 sec). Heparin dosage compared to heparin concentration showed a separate wide variation, (2 mg/kg: 3.72 ± 0.96 u/ml, 4 mg/kg: 6.23 ± 1.08 u/ml). The ACT was not predictive of the heparin level at 2 mg/kg ($r = 0.447$ or at 4 mg/kg ($r = 0.33$). Heparin was neutralized with 2 mg protamine/kg, regardless of the total heparin dose with plasma heparin levels falling from 4.33 ± 1.47 to 0.20 ± .21 u/ml. If the ACT remained elevated after the 2 mg/kg dose, an additional protamine dose was calculated from the dose response curve. 49% of patients required additional protamine, but rarely as much as 3 mg/kg. Heparin levels were insignificant (<0.2 u/ml) for four hours after protamine. Total postoperative blood loss was not significantly different from a retrospective control group (528.9 ± 341.3 vs 516.7 ± 251.3cc $p>.05$). Hence a protamine dose of 30 to 50% less than that commonly used was effective. Heparin rebound occurred in two patients at one and two hours (heparin levels 0.57 u/ml and 0.89 u/ml), unassociated with additional blood loss. The sensitivity and specificity of the ACT to detect a plasma heparin level greater than 0.2 u/ml was 95% and 84% respectively in the postneutralization period. However the ACT did not correlate with the heparin levels at levels above 0.2 u/ml ($r = 0.354$).

These data show (1) ACT has no linear relationship to plasma heparin, (2) heparin sensitivity varied widely among patients, (3) 2-3 mg of protamine per kg effectively reversed heparin in almost all patients, (4) heparin rebound was not clinically significant. Whether the ACT level (greater than 400 sec) or heparin concentration (greater than 3 u/ml is the best measurement of safe anticoagulation during CPB remains uncertain.

*By invitation

33. Right Ventricular Function During Left Heart Bypass

ALFONSO T. MIYAMOTO, SHIGEO TANAKA**

and JACK M. MATLOFF Los Angeles, California

Right heart failure has been recognized as a complication of left heart support bypass (LHBP), particularly with the most effective left ventricular transapical cannulation (TaLV) using pulsatile systems. Eight dogs (26.9 ± 1.4 kgm) were subjected to roller pump LHBP using no reservoirs in the circuit. Two inlet cannulae were connected in parallel (LA septal suture cannulation with Litwak-Koffsky LHBP cannula of 6.3 mmID, and TaLV cannulation with No. 30 venous cannula of 6.7 mmID) to allow comparison of both techniques. The cardiac output (CO) was controlled (100 ml/kg/min) by a roller pump returning the entire venous blood to the RA after disconnecting both cavae from the RA. Right ventricular function was evaluated by peak RV pressure, its first derivative (dp/dt) and mean RAP mesurements. LAP, LVP, AoP and both roller pump flows (F) were determined. Results: mean ± SEM of duplicate determinations obtained after at least 5 minutes of hemodynamic stabilization at each particular condition. (* = p<0.05 vs control; ** = p<0.05 vs 60% LHBPF ratio).

	$\frac{\text{LHBPF}}{\text{CO}}$ x 100 %	LAP torr	Peak LVP torr	RV dp/dt Torr/sec	RAP torr	Peak RVP torr
Control	No LHBP	2.9 ± 1.2	126 ± 4.5	212 ± 17	3.3 ± 0.9	27 ± 2.9
LHBP-Scpuil(S)	60	0.7 ± 05*	109 ± 5.7*	192 ± 16*	2.3 ± 0.6	26 ± 3.1
LHBP-S	80	0.7 ± 04*	101 ± 6.3***	182 ± 17*	2.3 ± 0.5	26 ± 3.0
LHBP-S	90	0.3 ± 0.1*	99 ± 6.31***	178 ± 16***.	2.2 ± 0.5	27 ± 3. 3
LHBP-S Max.	101	0.1 ± 0.1*	90 ± 6.4***	174 ± 16***.	2.6 ± 0.6	26 ± 3.4
LHBP-Tal.V Max.	102	1.5 ± 0.7*	72 ± 11***0	200 ± 15	3. 4 ± 0.8	26 ± 3.0
LHBP-S + TaLV Max.	105	0.1 ± 0.1*	2 ± 1.7***	168 ± 13***	5.1 ± 1.1***	25 ± 3.0

The decrease of RV dp/dt and increase of RAP are related to degree of LV decompression, i.e., LHBP flow ratio. The more complete the LHBP, the more profound its detrimental effect on RV function. Practical implications: a) it is probably better to use the minimal LHBP flow ratio that is required to maintain an adequate body perfusion, rather than the highest LHBP flow ratio to provide maximal reduction of left ventricular myocardial oxygen consumption; b) if more than 80-90% LHBP flow ratio is required to maintain adequate body perfusion, cannulation for biventricular bypass or venoarterial bypass with extracoporeal membrane oxygenation support type bypass should be made available, and c) LA septal suture cannulation provides consistently any LHBP flow ratio up to almost the entire LV output without the disadvantages of the TaLV cannulation (LV injury, bleeding) for LHBP with standard roller pumps.

34. Mechanical Assistance of the Pulmonary Circulation After Right Ventricular Exclusion

AART BRUTEL DE LA RIVIERE, GEORGE HAASLER*,*

JAMES R. MALM and DAVID BREGMAN

New York, New York

The Fontan procedure is often associated with elevated right sided pressures and low cardiac output during the early postoperative period. A dog model was established to test the effect of pulmonary artery counter-pulsation after atriopulmonary anastomosis.

After exclusion of the right ventricle by a purse string at the right AV orifice placed during inflow occlusion, a valved conduit was inserted between the right atrial appendage and the pulmonary artery, thereby obtaining a ciculatory pattern comparable to a Fontan procedure. Counterpulsation was achieved by inserting a cannula into the conduit distal to the valve in eight dogs, while in four, alternatively, Counterpulsation could comparably be achieved through a 10 mm low porosity prosthetic graft also connected to the conduit distal to the graft.

Twenty-four observations were made. Counterpulsation resulted in a mean increase in cardiac output of 48% ($p<.0001$). Right atrial pressure fell significantly with a mean drop of 4 mm Hg ($p<.003$) allowing for a further increase in right sided filling pressue by transfusion with a subsequent further increase in cardiac output. Left atrial pressure did not change significantly unless altered by transfusion.

Pulmonary vascular resistance, which was elevated after the institution of "Fontan physiology," decreased with Counterpulsation (mean decree 35%; $p<.002$).

Counterpulsation instituted through the 10 mm side arm graft gave similar results. Without Counterpulsation the circulatory status of the dog deteriorated rapidly. The use of a side arm graft connected to the conduit after a Fontan procedure affords easy clinical application of this method of circulatory support without the need for additional surgical intervention for decannulation. Based upon these data it is shown that mechanical assistance of the failing right atrium after atriopulmonary anastomosis is simple and highly effective.

*By invitation

35. Growth of the Left Ventricle in Compensatory Right Ventricular Hypertrophy

DOUGLAS F. LARSON, JACQUE R. WOMBLE*,*
JACK G. COPELAND, ROBERTS. MAMMANA**
*and DIANE H. RUSSELL**

Tucson, Arizona

Sponsored by: NORMAN E. SHUMWA Y Stanford, California

Pressure load models of right ventricular hypertrophy (RVH) were produced by infusion of a toxin, monocrotaline, which causes pulmonary artery fibrosis and increased vascular resistance, or injection of silica into the pulmonary vasculature to mechanically increase pulmonary vascular resistance. Also, in a volume overload model, maximal RVH was developed by a pulmonary artery to right atrial vascular shunt. In these models of RVH a Cordis Introduced was surgically inserted into the external jugular vein to allow the introduction of 7 Fr. Swan Ganz ThermodilutionR catheter for hemodynamic measurements and chronic blood sampling. During the 30 day study period, the hypertrophy events were characterized by measurements of the hemodynamics, circulating hormones, and blood constituents.

A consistent finding in these dog models of RVH was a concomitant increase in left ventricular mass. Hypertrophy in this study was defined as an increased ventricular dry weight to body weight ratio. The monocrotaline toxin induced hypertrophy resulted in an increased RVH 141% of control with a simultaneous increase of left ventricular mass of 117% of control. The silica injected dogs were found to have RVH 132% of control with an increased left ventricular mass of 122% of

control. The volume overload model had RVH 166% of control with an increased left ventricular mass of 144% of control. The only consistent hormonal finding which occurred during the hypertrophy process in the above models was a marked elevation in circulating plasma epinephrine. In dogs with pressure overload and ablation of the epinephrine source by denervation of the adrenal medulla, the heart weight to body weight ratios were decreased 88% of control. Correlation of the RVH to plasma epinephrine levels resulted in r = 0.92. In the RVH models the degree of increase in left ventricular mass was correlated to the plasma epinephrine with an r = 0.88. These data implicate endogenous circulating epinephrine as a specific hormone regulating compensatory RVH. This hormone is promoting the growth of the left ventricle simultaneously with the compensatory right ventricular growth.

*By invitation

36. Fibrin Adhesive - An Important Hemostatic Adjunct in Cardiovascular Surgery

HANS GEORG BORST, AXEL HAVERICK*,*

GERD WALTERBUSCH and WINFRIED MAATZ*,*

Sponsored by: BRUNO MESSMER, Aachen, West Germany

Fibrin adhesive is a commercially available human fibrinogen cryoprecipi-tate activated by bovin thrombin and calcium. During the last 3 years this principle has been applied experimentally and clinically in our unit and was shown highly effective in controlling diffuse or localized hemorrhage from the heart and great vessels as well as in presealing prosthetic fabric. In 10 pigs bleeding from coronary arterial and venous anastomoses as well as from epicardial lacerations was effectively controlled. Water porosity of various woven and knitted vascular prostheses was reduced to zero by fibrin glue in ex vivo experiments and complete sealing was maintained in 12 grafts subsequently implanted into fully heparinized chronic dogs. Fibrin glue controlled or prevented hemorrhage in 94.5% of 236 clinical applications involving open heart surgery (190 pts) or systemic heparinization (46 pts) (Table). Neither recurrent bleeding nor complications of gluing were observed. Conclusions: Fibrin sealing of puncture holes, epi-/myocardial bleeding sites, fabric patches and high porosity vascular prostheses appears an expedient method for avoiding the hazards of continuing hemorrhage. Pregluing of prostheses eliminates the necessity of natural preclotting and knitted grafts may be used in situations otherwise requiring woven material.

	no. cases	**failures %**
High pressure suture lines, anastomoses, patches	107	9.3
Low pressure suture lines, lacerations; epicardial abrasions	55	2.0
Presealed woven grafts	28	3.6
Presealed knitted grafts*	46	-
	236	5.5

*heparinization only

*By invitation

37. Long Term Evaluation of Pericardia! Substitutes

PAUL J. MEUS, JORGE A. WERNLY*,*

CHARLES D. CAMPBELL, YOSHINORI TAKANASHI**

and ROBERT L. REPLOGLE

Chicago, Illinois and Tokyo, Japan

The development of postoperative pericardia! adhesions increases the risk of cardiac reoperations because of the danger of damaging the heart, vessels or grafts. Several pericardial substitutes have been tested in the past in an attempt to facilitate reoperation with inconclusive results. This study evaluated eight different materials as pericardial substitutes. In 32 mongrels a 10 x 5 cm piece of pericardium was excised through a right thoracotomy and the defect closed with a patch. Each material tested was implanted in 4 dogs that were sacrificed at 3, 6, 9 and 12 months. At autopsy the development of adhesions and the epicardial reaction were graded as none, minimal, moderate, and severe. Histological studies of the patch, the epicardium, and the suture line were performed. The Table below lists the materials evaluated in this study and summarizes the results obtained.

	Polytetrafluoroethylene (PTFE)						Bovine Pericardium Gluteraldehyde Fixed	
	Silicone Filled Film	Low Porosity Film	High Porosity Film	Flourinated Ethylene Propylene Film	Poly-ethylene Film	Silicone Coated Polyester Fabric	Formaldehyde Preserved	Ethanol Preserved
Pleural Adhesions	Minimal	Moderate	Minimal	Moderate	Moderate	None	Minimal	Minimal
Pericardial Adhesions	Minimal	Moderate	None	Severe	Severe	None	Minimal	Minimal
Epicardial Reaction	Severe	Moderate	Severe	Severe	Severe	Severe	None	None
Patch	Intact	Thickened	Incact	Torn	Torn	Incact	Incact	Intact

Our results suggest that both types of bovine pericardium were an excellent substitute. Although there was development of minimal adhesions, these were easily dissected. The underlying anatomy was clearly recognizable due to the lack of epicardial reaction. Silicone rubber coated polyester fabric was an acceptable material for the prevention of adhesions but a severe fibrous epicardial reaction impeded the recognition of the coronary arteries. Both silicone filled and high porosity PTFE films reduced adhesions but caused a severe epicardial reaction. The other synthetic materials were considered inferior due to severe epicardial reactions and/or structural deterioration.

*By invitation

38. Elective Prolongation of Atrioventricular Conduction by Multiple Discrete Cryolesions: A New Technique for the Treatment of Paroxysmal Supraventricular Tachycardia

WILLIAM L. HOLMAN, MASA TOSHIIKESHITA*,*

PETER K. SMITH, JAMES M. DOUGLAS, JR.*,*

T. BRUCE FERGUSON, JR. and JAMES L. COX**

Durham, North Carolina

Sponsored by: DAVID C. SABISTON, JR.,

Durham, North Carolina

The most common etiology of paroxysmal Supraventricular tachycardia (PSVT) is re-entry within the A-V node. Heretofore, the only surgical treatment employed for medically refractory PSVT has been His bundle (HB) interruption, necessitating a permanent ventricular pacemaking system. The present study was designed to develop a technique for altering the input pathways of the A-V node electively in hopes of achieving permanent prolongation of A-V conduction and ablation or modification of A-V node reentrant arrhythmias. Bipolar atrial and ventricular pacing and sensing electrodes and a tri-electrode catheter positioned in the non-coronary cusp of the aorta were used to measure the pace-artifact to atrial depolarization (PA), atrial-His (AH), and His-ventricular (HV) intervals in 27 dogs. In Group I animals (n = 17), during cardiopulmonary bypass (CPB), nine separate 4-mm cryolesions (-60°C for 2 minutes) were placed around the A-V node after the position of the HB had been identified by endocardia! mapping. PA, AH, & HV intervals were measured before and at 15, 30, 60, and 180 minutes following CPB. Group II animals (N = 10) underwent identical procedures, omitting the cryolesions. Conduction times in msec: (M ± SEM)

		Pre-CPB (Control)	**Post-CPB 15 Min**	**30 Min**	**1 Hour**	**3 Hours**
GROUP	PA	30 ± 2	28 ± 2	29 ± 2	31 ± 2	27 ± 2
I	AH	73 ± 3	117 ± 11*	108 ± 10*	102 ± 6*	101 ± 8*
	HV	29 ± 1	29 ± 1	32 ± 2	31 ± 2	28 ± 1
GROUP	PA	28 ± 2	30 ± 1	27 ± 2	28 ± 2	27 ± 2
II	AH	74 ± 5	66 ± 4 *	70 ± 5	71 ± 5	67 ± 4
	HV	29 ± 1	25 ± 1	31 ± 1	31 ± 1	28 ± 2

*p less than 0.05 compared to same interval at control

Three Group I animals survived 10 weeks and all demonstrated persistent prolongation of the A-H interval. Multiple, precisely placed small cryolesions can reliably produce permanent prolongation of normal A-V conduction by altering the input pathways and conduction characteristics of the A-V node. This results in either a modification of the ventricular response to PSVT or ablation of the A-V node reentry responsible for the PSVT without necessitating an artificial ventricular pacemaker.

*By invitation

39. Prosthetic Replacements for the Thoracic Vena Cava: An Experimental Study

ANDREW C. FIORE, JOHN W. BROWN*,*

ROBERTS. CROMARTIE, LOUIS C. OFSTEIN*,*

PAMELA S. PEIGH, NICHOLAS S. SEARS*,*

WILLIAM P. DESCHNER* and HAROLD KING
Indianapolis, Indiana; Ormand Beach, Florida and

Sioux Falls, South Dakota

The ideal substitute for the thoracic vena cava continues to be a problem. Failure of an adequate prosthesis may be due in part to decreased flow, variable intrathoracic pressure and external compression by adjacent structures. Dacron (D) grafts in the venous system have a low patency rate, PTFE (Cortex), externally stented PTFE (1MPRA) and glutaraldehyde preserved porcine pericardium (GPPP) may offer alternatives, whose use in the thoracic vena cava has not been thoroughly evaluated. The purpose of the present study was to assess the short term patency of D, Cortex, IMPRA and GPPP when compared to autologous vein (AV) as a thoracic vena cava prosthesis.

Under general anesthesia, 40 adult mongrel dogs underwent right thoracotomies and the entire intrathoracic superior or inferior vena cava of each animal was replaced with a standard segment (4.5 cm (1) x 3.5 cm (w)) of knitted D (8 dogs), Cortex (8 dogs), IMPRA (12 dogs) and GPPP (12 dogs). An additional 6 animals had the same caval segment replaced with AV, fashioned as a panel graft using the external jugular vein and served as controls. After 30 days, patency was assessed by contrast venography and the implanted material removed for histological evaluation. The patency rate of each graft was compared to that of AV and the results are shown below:

Prosthesis	**Patency at 30 Days**	**vs. Autologous Vein**
AV	6/6	-
D	0/8	*P<.001
PTFE	6/8	NSD
IMPRA	12/12	NSD
GPPP	6/12	*P<.05

*Obtained by chi square analysis.

No D grafts and only 50% of GPPP grafts were patent at 30 days. Three-fourths of the Cortex grafts and all of the IMPRA prostheses remained patent.

This study demonstrated: (1) D grafts remain inadequate as venous conduits. (2) PTFE has been shown to offer higher expectations of patency when used as a thoracic venous prosthesis, than do grafts fashioned from porcine xenograft. (3) In the context of this experiment, the early patency of externally stented PTFE equals that of autologous vein in the thoracic vena cava.

9:45 a.m. Intermission - Visit Exhibits

*By invitation

10:30 a.m. Scientific Session - Assembly Hall

40. En Bloc Resection for Neoplasms of the Esophagus and Cardia

DAVID B. SKINNER
Chicago, Illinois

In 1963, Logan reported experiences with *en bloc* resection of carcinoma of the cardia. In 1965, a technique was developed for *en bloc* resection of carcinoma of the body of the esophagus with removal of the entire posterior mediastinum including thoracic duct and azygos vein system.

Beginning in 1969, a radical *en bloc* resection for carcinoma of the cardia and esophagus was adopted for all operable cases. In 1974, *en bloc* approach including radical neck dissection was adopted for carcinoma of the cevical esophagus.

From June, 1969 to July, 1981, 175 patients with neoplasms of the esophagus and cardia were referred to me for treatment. Among these, 80 were considered operable based upon preoperative and intraoperative evaluation indicating that all grossly detectable disease could be encompassed by radical resection. Another 15 had palliative resection, 19 had a bypass operation, 57 were radiated for palliation after exploration or positive node or liver biopsy, and 4 had a tube inserted for palliation.

Among the 80 radical resections, there were 9 (11%) hospital deaths within 30 days of resection. Abolute survival rate for all patients operated more than 3 years ago is 22%, and actuarial table 3 year survival for the entire series is 24%. There have been no deaths for recurrent cancer after 3 years. Results were significantly worse among 12 patients receiving preoperative radiation therapy than in those 68 in which surgery was the first therapy (1 year survival 18% vs. 66% with no difference in hospital mortality). Results were similar for 39 lower third and 27 middle third tumors and slightly better for 14 cancers of the cervical esophagus. There was no difference in results between squamous and adenocarcinoma, although 18 patients with adeno-carcinoma in Barrett's esophagus had poorer results than 17 with carcinoma of the cardia.

Operative techniques, complications, rationale, and detailed results are presented.

41. Transhiatal Esophagectomy Without Thoracotomy - A Dangerous Operation?

*MARK B. ORRINGER and JAY S. ORRINGER**

Ann Arbor, Michigan

In 1978, before this Association, a preliminary report describing trans-hiatal esophagectomy without thoracotomy in 26 patients was criticized for advocating a dangerous operation which violates the basic surgical principles of adequate exposure and hemostasis. This report describes our cumulative clinical experience with this operation in 134 patients: 40 with benign disease and 94 with carcinomas at various levels of the esophagus (10 pharyngeal, 20 cervicothoracic, 5 upper third, 32 middle third, and 27 distal third). Esophageal resection and reconstruction were performed in a single stage in 129 patients, and the esophageal substitute was positioned in the posterior mediastinum in the original esophageal bed in 124 patients. Continuity of the alimentary tract was restored by anastomosing the pharynx or cervical esophagus either to stomach (119 patients) or to a colonic graft (10 patients).

There have been 11 postoperative deaths (8.2%operative morality) due to myocardial infarction (3), pneumonia (3), innominate artery rupture (2), pulmonary embolus (1), and mediastinal (1) or retroperitoneal (1) infection. None was the direct result of the technique of esophagectomy. Complications included intraoperative pneumothorax (67), transient hoarseness (35), anastomotic leak (19), chylothorax (4), and tracheal laceration (2). Average intraoperative blood loss for the entire group was 1200 ml, 1100 ml for those with benign disease, 1800 ml for those with carcinoma requiring concom-mitant laryngectomy, and 900 ml for those with carcinoma undergoing esophagectomy without laryngectomy.

Of 61 patients with carcinoma who underwent esophagectomy without concommitant laryngectomy and were discharged from the hospital alive, 45 (74%) left within 14 days of operation, and another 7 (11%) left between 15 and 21 days; thus 52/61 (85%) were discharged within 3 weeks of their operation.

These data support (he contention that a thoracic incision is seldom required to resect the esophagus for either benign or malignant disease. Transhiatal esophagectomy without thoracotomy is a safe well-lolerated operation, the "hazards" of which can be minimized by careful technique and experience.

*By invitation

42. Total Fundoplication Gastroplasty (T.F.G.) - Long-Term Follow-Up in 500 Patients

*ROBERT D. HENDERSON and GARY MARRYATT**

Toronto, Ontario

Five hundred patients have been treated surgically by T.F.G. for reflux control. Patients were selected because of intractable symptoms; 182 had a previous surgery; 8 scleroderma; and 31 had a peptic stricture. Surgically, a *5* cm gastroplaty tube was made over a #60 Fr bougie and reflux control achieved using total fundoplication. In the last 3 years the completion wrap length was reduced from 3 to 1.5 cm.

There was no mortality and major morbidity was 9 (1.8%) including 5 fisculae (1%). Follow-up ranges from 1.25 to 6 years; clinical 98%; radiologic 91%; manometric and pH studies 70.8%. Four anatomic recurrences are present (0.8%), one asymptomatic and three treated surgically. Seven required revision surgery (1.4%) with wrap shortening. Revision has not been required following reduction of the completion wrap to 1.5 cm. Minor residual symptoms are present in 41 patients; 12 (2.4%) with minor gastroesophageal and 9 (1.8%) cricopharyngeal dysphagia; 5 with minor bloating and 15 (3%) with nonspecific indigestion.

Radiologically 1 (0.2%) had asymptomatic reflux. Manometrically the HPZ tone rose from 12.7 to 17.57 (38.3%). Percent DMA in the lower half of the esophagus decreased from 45.5% to 28.5%. Reflux was not demonstrated by pH studies. Asymptomatic results are present in 89.8%; 8.2% have minor residual symptoms and 2% required revision surgery and are now improved. Dysphagia was originally ocassionally produced by too long a wrap, and this problem has been minimized by reducing the wrap length.

T.F.G. has proved to be a safe operative approach capable of producing effective reflux control with minor risks of analomic recurrence.

*By invitation

43. Thymectomy in Multiple Sclerosis: Preliminary Trial

*THOMAS B. FERGUSON and JOHN L. TROTTER**

St. Louis, Missouri

Myasthcnia gravis (MG) is an auto-immune disease which is known lo be influenced by the thymus gland. During the past 9 years thymectomy, utilized as the initial treatment for MG and done early in the course of the disease, has produced a 90% remission rate in the young MG patient ai our institution.

Multiple sclerosis (MS) is also thought to be a disease involving the immune system. The experience with MG cited above suggests that ihymectomy may favorably influence MS. A

preliminary trial was initiated in 1976, and to date 36 patients have been entered. Proven MS patients with a clearly established pattern of progression were selected. All operations were done through a sternal-split by one surgeon. The patients have had a complete neurologic and immunologic survey before and at yearly intervals after thymeetomy. All patients are more than one year post-operative (fn 33.9 months).

Results: Comparing the trial patients one year after thymeclomy to a carefully matched group of control (no operation) MS patients: (1) pyramidal functions are significantly better ($P > .01$) in trial patients. Other neurologic functions show no significant difference. (2) Disability is decreased ($P > .05$) in trial patients with relapsing-remitting MS. (3) The number of exacerbations is significantly decreased ($P > .01$) in trial patients. (4) Immunologic profiles show no alteration in T- and B-cells, or in mitogen studies. Cerebrospinal fluid immunoglobulins are unchanged.

We are sufficiently encouraged by these results to continue the trial. In the future, a multi-institutional study will be required to reach definite conclusions in this unpredictable disease.

*By invitation

44. Management of Air Embolism in Blunt and Penetrating Trauma

EDWARDS. YEE, EDWARD D. VERRIER**

and ARTHUR N. THOMAS

San Francisco, California

The charts of 54 patients treated from 1970 lo 1981 were reviewed to determine the clinical outcome after treatment of air embolism from blunt (15 patients) and penetrating (18 gunshot and 21 stabbing) thoracic injuries. The diagnosis of air embolism was confirmed by the presence of air in coronary vessels, air aspirated from the heart or a major artery, or doppler findings.

Thirty-nine patients (72%) presented to the Emergency Room in profound shock (30 patients, 56%) or cardiac arrest (9 patients, 16%) and the other 15 patients (28%) deteriorated during the first twelve hours (shock 10 and arrest 5 patients). Six patients out of 40 in shock (15%) arrested unexpectedly after intubation and administration of positive ventilation. Hemoptysis or bronchial bleeding from endotracheal tube is an early sign for air embolus (8/54, 15%).

Successful management included: (1) early thoractotomy, (2) control of bronchovenous communication by hilar crossclamping, (3) maintaining normal systolic pressures with vasopressors (56%) or aortic crosscelamping (13%), and (4) prompt correction of embolic source (lung 85%, heart 11%, cava-liver 4%), which usually requires a major lung resection. The overall survival rate is (28/54, 52%). Survival correlates with mechanism of injury (blunt - 4/15, 27% vs penetrating - 24/39, 67%, $0.10 > p > 0.05$) and the presence of associated nonthoracic injuries (present 9/33, 27% vs absent 19/21, 90% - $p < .001$).

We conclude that: (1) air embolus can insidiously occur even in blunt trauma, (2) suspicion should be high with occurrence of hemoptysis or unexpected arrest during positive ventilation, (3) treatment should include early proximal control of hilum, maintaining coronary perfusion pressures, and prompt correction of embolic sources, and (4) successful results correlate with the outcome of associated injuries.

1:00 p.m. ADJOURNMENT

WEDNESDAY MORNING, MAY 5, 1982

Alternate Papers (To be presented in case of a cancellation)

Forum Session

A1 Ultrafiltration to Decrease the Accumulation of Lung Water During Cardiopulmonary Bypass

DONALD J. MAGILLIGAN, JR.

*and CHOKEN OYAMA**

Detroit, Michigan

Ultrafiltration is the removal of water and plasma concentration of electrolytes from blood by passage across a semi-permeable membrane. An Amicon Diafilter was placed between the arterial and venous lines of standard cardio-pulmonary bypass (CPB) in an attempt to determine the effect of ultrafiltra-tion on the accumulation of lung water during dilution perfusion CPB. Mongrel dogs were placed on CPB with non-blood prime and LRS added to maintain a flow of 100 ml/min/kg. Measurements made pre-bypass and 30 minutes post-bypass were: wedge pressure (Ppcw), colloid oncotic pressure (COP), COP-Ppcw, cardiac index (CI), and extravacular lung water (EVLW) determined by the thermal-dye indicator dilution technique wilh gravimetric correlation. Dogs were divided into two groups: Group 1 - two hours normothermic bypass, beating heart, left heart vented, and Group II - 90 minutes, cold, potassium, cardioplegic arrest and 30 minute recovery.
Results:

Group I:	**Control**	**Ultrafiltration**	**Difference**
Number	4	4	__
Fluid added	2350	2750	N.S.
Fluid out	180	2288	p = <.01
Î”CI	†“.20 L/min/m^2	†“1.65 L/min/m^2	N.S.
Î”EVLW	†‘2.83 ml/kg	†‘1.03 ml/kg	P = <.01
Group 11			
Number	2	4	-
Fluid added	2775	3425	P = <.01
Fluid out	110	2710	p = <.01
Î”CI	†“0.09L/min/m^2	†“0.65 L/min/m^2	N.S.
Î”EVLW	†‘3.40 ml/kg	†‘0.95 ml/kg	P = <.05

There were no significant changes in Ppcw, COP, or COP-Ppcw in either group. The change in CI was similar in control and Ultrafiltration animals. Ultrafiltration during CPB allowed significantly less water to accumulate in the lungs compared lo control in both groups.

When CPB is necessary in a patient with elevated EVLW, ultrafiltration may prevent further increases in EVLW and thereby decrease posl-operative pulmonary dysfunction.

*By invitation

Scientific Session

A1 Complications of Percutaneous Intraaortic Balloon Insertion

RAYMOND S. MARTIN, III, ASHBY C. MONCURE,*

MORTIMER J. BUCKLEY, W. GERALD AUSTEN,

GARY W. AKINS and ROBERT C. LEINBACH**

Boston, Massachusetts

Early studies reported that percutaneous intraaortic balloons can be placed quickly and easily with minimal complications. To assess our experience with this technique, the records of the first 100 consecutive percutaneous inlra-aortic balloon insertion attempts at our hospital were reviewed. In 17 insertion attempts, the ballooon could not be passed retrograde inio the thoracic aorta, usually due to tortuosity or obstructive disease of the iliac arterties. Of the 83 patients in whom the balloon could be passed, 63 had no subsequent complications. Five balloons were successfully inserted, but the patient died immediately after insertion. Four patients developed in ischemic lower c.v Iremity on the side of insertion, requiring only balloon removal for limb salvage. Eleven patients developed an ischemic limb or false ancurysm requiring surgery for correction; of these, amputation was required in two extremities, and one patient died as a result of iliac artery perforation by the balloon.

Specific vascular injuries included six instances of femoral artery laceration with inlimal plaque elevation and thrombosis. Other injuries were false aneurysm with or without thrombosis, thrombosis without significant arterial injury, and iliac perforation. Techniques of repair included Ihrombectomy with lateral repair, vein or prosthetic patch angioplasly, and simple suture repair of false aneurysms.

Excluding patients who died immediately after balloon insertion, and thus had no follow-up, complications requiring surgery occurred in 11 of 95 attempted insertions (11.6%) in this series. This complication rate prompted us to examine indications and risk factors which might predict complications. In addition we have reviewed potentially preventativc techniques, such as prei-nsertion aortography and the use of a wire guide or long sheath for insertion.

Though percutaneous intraaortic balloon insertion has proved to be a valuable technique, a significant number of complications have been seen. Careful selection of patients and insertion by experienced hands should minimize complications.

*By invitation

A2 Repair of Post-Infarction Ventricular Septal Defect in the Elderly: Early and Long Term Results

*RONALD W. WEINTRAUB, ROBERT L. THURER**

*and JULIAN M. AORESTY**

Boston, Massachusetts

We performed 12 operations upon 11 consecutive elderly patients (pts) having postinfarction ventricular septal defect (VSD). All patients were older than 65 years (range: 66-82 years) and six were over 70. Nine underwent repair, with counterpulsation support, within one week of onset of the VSD. Of 8 anteriorly located VSDs, there were four survivors. Of three inferior defects, all survived, for an overall acute survival of 64%.

Our experience with respect to acute management suggests that 1) unless medical management results in *continued improvement* rather than stability alone, hemodynamic deterioration is inevitable, and survival for delayed repair is unlikely; 2) secondary operation for recurrent VSD and/or aneurysm formation can succeed despite high risk; and 3) technical details of infartectomy and graft preparation and placement are critically important in repair of inferior VSD.

The seven long term survivors were followed from 5 months to 7.5 years (mean: 2.8 years) by personal interview with patient or referring physician. There was one sudden death at 7.5 years in a previously well man. Of the remaining six paitnets, 5 are NYHA Class 1, and 1 is Class II. One lady, now 84, lives independently two years after repair.

We conclude that 1) most patients with VSDrequire early operation, 2) advanced age is no bar to successful VSD repair, and 3) long term results and quality of life may be excellent following VSD repair in the aged.

*By invitation

American Association for Thoracic Surgery, 1981-1982

(Listed by Countries, States, Provinces and Cities)

Geographical - UNITED STATES

ALABAMA

BIRMINGHAM

Kahn, Donald R.

Karp, Robert B.

Kessler, Charles R.

Kirklin, John W.

Kouchoukos, Nicholas T.

Pacifico, Albert D.

MONTGOMERY

Simmons, Earl M.

OPELIKA

Le Beck, Martin

ALASKA

ANCHORAGE

Phillips, Francis J.

EL MACERO

Andrews, Neil C.

ESCONDIDO

Mannix, Edgar P., Jr.

FRESNO

Evans, Byron H.

HEMET

Hewlett, Thomas H.

INGLEWOOD

Carey, Joseph S.

IRVINE

Connolly, John E.

Miller, Don R.

Wakabayashi, Akio

LA CANADA

ARIZONA

PHOENIX

Brown, Lee B.

Cornell, William P.

Melick, Dermont W.

Nelson, Arthur R.

SUN CITY

Read, C. Thomas

TUCSON

Burbank, Benjamin

Sanderson, Richard G.

ARKANSAS

JASPER

Hudson, W. A.

LITTLE ROCK

Campbell, Gilbert S.

Read, Raymond C.

Williams, G. Doyne

CALIFORNIA

ANAHEIM

Main, F. Beachley

ARCADIA

Silver, Arthur W.

CARMEL

Daniels, Albert C.

COVINA

Carter, P. Richard

DAVIS

Andrews, Neil C.

DUARTE

Benfield, John R.

Aronstam, Elmore M.

LA JOLLA

Fosburg, Richard G.

Hutchin, Peter

LA MESA

Long, David M., Jr.

LOMA LINDA

Wareham, Ellsworth E.

LONG BEACH

Bloomer, William E.

Carlson, Herbert A.

Stemmer, Edward A.

LOS ANGELES

Baisch, Bruce F.

Buckberg, Gerald D.

Davis, Lowell L.

Fonkalsrud, Eric W.

Holmes, E. Carmack

Hughes, Richard K.

Kay, Jerome Harold

Lindesmith, George G.

Longmire, William, Jr.

Maloney, James V., Jr.

Matloff, Jack M.

Meyer, Bert W.

Morton, Donald L.

Mulder, Donald G.

Stiles, Quentin R.

MONTEBELLO

Lui, Alfred H. F.

OAKLAND

Dugan, David J.

Ecker, Roger R.

May, Ivan A.

ORANGE

Mason, G. Robert

Salyer, John M.

PACIFIC PALISADES

Weinberg, Joseph A.

PALM SPRINGS

Goldman, Alfred

PALM DESERT

Julian, Ormand C.

PALO ALTO

Cohn, Roy B.

Gonzalez-Lavin, Lorenzo

Jamplis, Robert W.

PASADENA

Cotton, Bert H.

Ingram, Ivan N.

Penido, John R. F.

S. LACUNA

Oatway, William H., Jr.

S. PASADENA

Brewer, Lyman A., III

SACRAMENTO

Harlan, Bradley J.

Hurley, Edward J.

Miller, George E., Jr.

Smeloff, Edward A.

Treasure, Robert L.

Tyson, Kenneth R. T.

SAN BERNADINO

Flynn, Pierce J.

SAN DIEGO

Baronofsky, Ivan D.

Chambers, John S., Jr.

Daily, Pat O.

SANTA BARBARA

Higginson, John F.

Jahnke, Edward J., Jr.

Lewis, F. John

Love, Jack W.

SANTA MONICA

Ramsay, Beatty H.

STANFORD

Mark, James B. D.

Shumway, Norman E.

Wilson, John L.

THOUSAND OAKS

Tsuji, Harold K.

TORRANCE

Nelson, Ronald J.

State, David

VENTURA

Dart, Charles H., Jr.

WALNUT CREEK

Stephens, H. Brodie

COLORADO

DENVER

Blair, Emil

Brown, Robert K.

Burrington, John D.

Condon, William B.

Eiseman, Ben

Grow, John B.

Harper, Frederick R.

Hopeman, Alan R.

Kovarik, Joseph L.

Newman, Melvin M.

Pappas, George

Paton, Bruce C.

Lamberti, John J., Jr.

Peters, Richard M.

Trummer, Max J.

Utley, Joe R.

SAN FRANCISCO

Culiner, Morris M.

Ebert, Paul A.

Ellis, Robert J.

Fishman, Noel H.

Gardner, Richard E.

Gerbode, Frank

Grimes, Orville F.

Hill, J. Donald

Leeds, Sanford E.

Richards, Victor

Roe, Benson B.

Rogers, W. L.

Thomas, Arthur N.

Ullyot, Daniel J.

SAN JOSE

Angell, William W.

SANTA ANA

Gazzaniga, Alan B.

Pratt, Lawrence A.

Pomerantz, Marvin

Rainer, W. Gerald

Waddell, William R.

Wright, George W.

LAKEWOOD

Swan, Henry, II

CONNECTICUT

HARTFORD

Kemler, R. Leonard

NEW HAVEN

Baue, Arthur E.

Carter, Max G.

Geha, Alexander S.

Glenn, William W. L.

Hammond, Graeme L.

Laks, Hillil

Lindskog, Gustaf E.

Stansel, Horace C., Jr.

Stern, Harold

NORTHFORD

Amberson, J. B.

NORWICH

Kelley, Winfield O.

SHARON

Wylic, Robert H.

WILTON

Pool, John L.

DELAWARE

WILMINGTON

Reis, Robert L.

Ripstein, Charles B.

Stanford, William

Thurer, Richard J.

MIAMI BEACH

Greenberg, Jack J.

Grondin, Pierre

Spear, Harold C.

NAPLES

Linberg, Eugene J.

Pecora, David V.

DISTRICT OF COLUMBIA

WASHINGTON

Aaron, Benjamin L.

Bowles, L. Thompson

Brotl, Walter H.

Hufnagel, Charles A.

Keshishian, John M.

Midgley, Frank M.

Mills, Mitchell

Peabody, Joseph, Jr.

Randolph, Judson G.

Simmons, Robert L.

Smyth, Nicholas P. D.

Wallace, Robert B.

FLORIDA

BELLEAIRE

Lasley, Charles H.

BOCA RATON

Seley, Gabriel P.

COCONUT CREEK

Selman, Morris W.

CORAL GABLES

Cooke, Francis N.

DELRAY BEACH

Geary, Paul

GAINESVILLE

Hartley, Thomas D.

La Brosse, Claude C.

JACKSONVILLE

Stephenson, Sam, Jr.

LAKELAND

Brown, Ivan W., Jr.

MIAMI

Bolooki, Hooshang

Center, Sol

NO. PALM BEACH

Dorsey, John M.

ORLANDO

Sherman, Paul H.

PONTE VEDRA BEACH

Gilbert, Joseph, Jr.

Stranahan, Allan

SOUTH MIAMI

Chesney, John G.

ST. PETERSBURG

Clerf, Louis H.

Daicoff, George R.

DeMatteis, Albert

Wheat, Myron W., Jr.

TALLAHASSEE

Kraeft, Nelson H.

TAMPA

Blank, Richard H.

Connar, Richard G.

Seiler, Hawley H.

WINTER HAVEN

Mauer, Elmer, P.R.

WINTER PARK

Bloodwell, Robert D.

GEORGIA

ATLANTA

Craver, Joseph M.

Hatcher, Charles, Jr.

Hopkins, William A.

Jones, Ellis L.

King, Richard

Logan, William D., Jr.

Mansour, Kamal A.

Miller, Joseph I.

Rivkin, Laurence M.

Symbas, Panagiotis

Daughtry, Dewitt C.

Gentsch, Thomas O.

Jude, James R.

Kaiser, Gerard A.

MacGregor, David C.

Papper, Emanuel M.

AUGUSTA

Ellison, Robert G.

Rubin, Joseph W.

MACON

Van De Water, Joseph

SAVANNAH

Yeh, Thomas J.

ST. SIMONS

Collins, Harold A.

HAWAII

HONOLULU

Ching, Nathaniel P.

Gebauer, Paul W.

McNamara, Joseph J.

KAILUA KONA

Fell, Egbert H.

IDAHO

BOISE

Ashbaugh, David G.

Herr, Rodney H.

ILLINOIS

CHICAGO

Anagnostopoulos, C.

Barker, Walter L.

Faber, L. Penfield

Hanlon, C. Rollins

Head, Louis R.

Hudson, Theodore R.

Hunter, James A.

Idriss, Farouk S.

Javid, Hushang

Jensik, Robert J.

Kittle, C. Frederick

Langston, Hiram T.

WINNETKA

Mackler, S. Allen

INDIANA

INDIANAPOLIS

Battersby, James S.

King, Harold

King, Robert D.

Mandelbaum, Isidore

Shumacker, Harris, Jr.

Siderys, Harry

SOUTH BEND

Van Fleit, William E.

IOWA

CEDAR RAPIDS

Lawrence, Montague S.

DES MOINES

Dorner, Ralph A.

Watkins, David H.

IOWA CITY

Doty, Donald B.

Ehrenhaft, Johann L.

Rossi, Nicholas P.

KANSAS

CUNNINGHAM

Allbritten, F. F., Jr.

Leininger, Bernard J.

Levitsky, Sidney

Michaelis, Lawrence

Midell, Allen I.

Moran, John M.

Najafi, Hassan

Raffensperger, John

Replogle, Robert L.

Shields, Thomas W.

Skinner, David B.

Thomas, Paul A., Jr.

Weinberg, Milton, Jr.

EVANSTON

Fry, Willard A.

Tatooles, Constantine

GLENCOE

Rubenstein, L. H.

MAYWOOD

Keeley, John L.

Pifarre, Roque

OAK BROOK

Nigro, Salvatore L.

PALO HEIGHTS

DeMeester, Tom R.

PEORIA

Collins, Harold A.

SKOKIE

Baffes, Thomas G.

KANSAS CITY

Barnhorst, Donald A.

Friesen, Stanley R.

WICHITA

Tocker, Alfred M.

WINFIELD

Snyder, Howard E.

KENTUCKY

LEXINGTON

Crutcher, Richard R.

Dillon, Marcus L.; Jr.

Todd, Edward P.J.

LOUISVILLE

Bryant, J. Ray

Harter, John S.

Mahaffey, Daniel E.

Ransdell, Herbert, Jr.

LOUISIANA

ALEXANDRIA

Knoepp, Louis F.

BATON ROUGE

Beskin, Charles A.

METAIRIE

Ochsner, Alton, Jr.

NEW ORLEANS

Blalock, John B.

DeCamp, Paul T.

Hewitt, Robert L.

Lindsey, Edward S.

Mills, Noel L.

Cleveland, Richard J.

Clowes, George, Jr.

Colin, Lawrence H.

Collins, John J.

Daggett, Willard M.

Deterling, Ralph, Jr.

Moulder, Peter V.

Ochsner, John L.

Pearce, Charles W.

Rosenberg, Dennis M.

Schramcl, Roberl J.

Strug, Lawrence H.

Webb, Watts R.

MAINE

KENNEBUNK

Hurwitz, Alfred

PORTLAND

Drake, Emerson H.

Hiebert, Clement

ROCKPORT

Swenson, Orvar

MARYLAND

BALTIMORE

Attar, Safuh M.A.

Baker, R. Robinson

Brawley, Robert K.

Cowley, R. Adam

Donahoo, James

Gott, Vincent L.

Haller, J. Alex, Jr.

Hankins, John R.

McLaughlin, Joseph S.

Michelson, Elliott

Turney, Stephen Z.

BETHESDA

Iovine, Vincent M.

Morrow, Andrew G.

POTOMAC

Zajtchuk, Rostik

WORTON

Walkup, Harry E.

MASSACHUSETTS

Frank, Howard A.

Gaensler, Edward A.

Grille, Hermes C.

Moncure, Ashby C.

Neptune, Wilford B.

Overholt, Richard H.

Rheinlander, Harold

Russell, Paul S.

Scannell, J. Gordon

Schuster, Samuel R.

Starkey, George W.

Weintraub, Ronald

Wilkins, Earle W., Jr.

BROOKLINE

Madoff, Irving M.

BURLINGTON

Boyd, David P.

Ellis, F. Henry, Jr.

Watkins, Elton, Jr.

CAMBRIDGE

Harken, Dwight E.

CONCORD

Soutter, Lamar

LYNNFIELD

Wesolow, Adam

MEDFORD

Boyd, Thomas F.

Desforges, Gerard

METHUEN

Wilson, Norman J.

N. ANDOVER

Cook, William A.

NANTUCKET

Mahoney, Earle B.

NEWTON

Norwood, William I.

BOSTON

Austen, W. Gerald

Barsamian, Ernest M.

Berger, Robert L.

Bernhard, William F.

Bougas, James A.

Braunwald, Nina S.

Buckley, Mortimer J.

Burke, John F.

Castaneda, Aldo R.

NEWTON LOWER FALLS

Laforet, Eugene G.

Strieder, John W.

S. WEYMOUTH

Malcolm, John A.

SPRINGFIELD

Engelman, Richard M.

STOUGHTON

Black, Harrison

WINCHESTER

Taylor, Warren J.

ST. PAUL

Leven, N. Logan

Lillehei, C. Walton

Miller, Fletcher A.

Perry, John F., Jr.

MICHIGAN

ANN ARBOR

Bartlett, Robert H.

Behrendt, Douglas M.

Gago, Otto

Kirsh, Marvin M.

Morris, Joe D.

Orringer, Mark B.

Sloan, Herbert

BIRMINGHAM

Dodrill, Forest D.

DETROIT

Arbulu, Augustin

Arcinicgas, Eduardo

Day, J. Claude

Lam, Conrad R.

Magilligan, D. J., Jr.

Wilson, Robert F.

GRAND RAPIDS

Harrison, Robert W.

Meade, Richard H.

Rasmussen, Richard A.

MISSISSIPPI

JACKSON

Hardy, James D.

Johnston, J. H., Jr.

Neely, William A.

Netterville, Rush E.

MISSOURI

COLUMBIA

Silver, Donald

KANSAS CITY

Adelman, Arthur

Ashcraft, Keith W.

Benoit, Hector W., Jr.

Holder, Thomas M.

Killen, Duncan A.

Mayer, John H., Jr.

Padula, Richard T.

GROSSE POINTE

Benson, Clifford D.

Gerbasi, Francis S.

Taber, Rodman E.

KALAMAZOO

Neerken, A. John

ROYAL OAK

Timmis, Hilary H.

MINNESOTA

CROOKSTON

Deniord, Richard N.

DULUTH

Fuller, Josiah

MINNEAPOLIS

Anderson, Robert W.

Arom, Kit V.

Garamella, Joseph J.

Helseth, Hovald K.

Humphrey, Edward W.

Johnson, Frank E.

Kiser, Joseph C.

Molina, J. Ernesto

Nicoloff, Demetre M.

ROCHESTER

Bernatz, Philip E.

Clagett, O. Theron

Danielson, G. K.

Kaye, Michael P.

McGoon, Dwight C.

Olsen, Arthur M.

Payne, W. Spencer

Pluth, James R.

NEW JERSEY

BELLVILLE

Reed, William A.

MT. VERNON

Campbell, Daniel C., Jr.

ST. LOUIS

Earner, Hendrick B.

Bergmann, Martin

Clark, Richard E.

Codd, John E.

Connors, John P.

Ferguson, Thomas B.

Kaiser, George C.

Lewis, J. Eugene, Jr.

Pennington, D. Glenn

Roper, Charles L.

Weldon, Clarence S.

Willman, V. L.

NEBRASKA

OMAHA

Fleming, William H.

Malette, William G.

Sellers, Robert D.

NEW HAMPSHIRE

HANOVER

Crandell, Walter, B.

PETERBOROUGH

Woods, Francis M.

WHITE RIVER JUNCTION

Tyson, M. Dawson

BRONX

Becker, Ronald M.

Gerard, Franklyn P.

BELMAR

Bailey, Charles P.

BROWNS MILL

Fernandez, Javier

CAMDEN

Camishion, Rudolph C.

EAST ORANGE

Auerbach, Oscar

MILLBURN

Parsonnet, Victor

MOORESTOWN

Morse, Dryden P.

N. CALDWELL

Wychulis, Adam R.

NEW BRUNSWICK

Kunderman, Philip J.

NEWARK

Abel, Ronald M.

Amato, Joseph J.

Neville, William E.

PENNSAUKEN

Pierucci, Louis, Jr.

PISCATAWAY

MacKenzie, James W.

SHORT HILLS

Demos, Nicholas J.

Timmes, Joseph L.

TENAFLY

Gerst, Paul H.

NEW MEXICO

ALBUQUERQUE

Edwards, W. Sterling

ARTESIA

Glass, Bertram A.

LAS VEGAS

Bloomberg, Allan E.

Fell, Stanley C.

Hirose, Teruo

Robinson, George

BRONXVILLE

Frater, Robert W. M.

BROOKLYN

Griepp, Randall B.

Levowitz, Bernard S.

Sawyer, Philip N.

BUFFALO

Adler, Richard H.

Andersen, Murray N.

Lajos, Thomas Z.

MacManus, Joseph E.

Subramanian, S.

COOPERSTOWN

Blumenstock, David A.

FAYETTEV1LLE

Bugden, Walter F.

FLORAL PARK

Crastnopol, Philip

GREAT NECK

Wisoff, B. George

IRVINGTON

Altai, Lari A.

NEW PALTZ

Johnson, Elgie K.

NEW YORK

Beattie, Edward, Jr.

Bowman, Frederick, Jr.

Boyd, Arthur D.

Bregman, David

Cahan, William G.

Clauss, Roy H.

Conklin, Edward F.

Thai, Alan P.

SANTA FE

Wilson, Julius L.

NEW YORK

ALBANY

Alley, Ralph D.

Kausel, Harvey W.

McKneally, Marlin F.

BAY SHORE

Ryan, Bernard J.

Cournand, Andre

Cracovaner, Arthur J.

Cunningham, J.N., Jr.

Davidson, Louis R.

Findlay, Charles W., Jr.

Ford, Joseph M.

Friedlander, Ralph

Gay, William A., Jr.

Giannelli, Stanley, Jr.

Green, George E.

Holman, Cranston W.

Holswade, George R.

Hood, R. Maurice

Humphreys, G. H., II

Hutchinson, John E., Ill

Isom, O. Wayne

Jarclzki, Alfred, III

King, Thomas C.

Kirschner, Paul A.

Lambert, Adrian

Litwak, Robert S.

Maier, Herbert C.

Malm, James R.

Martini, Nael

Nealon, Thomas F., Jr.

Okinaka, Arlhur J.

Redo, S. Frank

Reemtsma, Keith

Rubin, Morris

Spencer, Frank C.

Spotnitz, Henry M.

Steichen, Felicien M.

Subramanian, V. A.

Tice, David

VALHALLA

Reed, George E.

W. HAMPTON BEACH

Sarol, Irving A.

NORTH CAROLINA

ASHEVILLE

Belts, Reeve H.

Scott, Stewart M.

Sethi, Gulshan K.

Takaro, Timothy

CHAPEL HILL

Murray, Gordon F.

Starek, Peter J. K.

Wilcox, Benson R.

CHARLOTTE

Robicsek, Francis

Taylor, Frederick H.

DURHAM

Jones, Robert H.

Oldham, H. N., Jr.

Sabiston, David C.

Veith, Frank J.

Wallsh, Eugene

Wichern, Waller, Jr.

Wolff, William I.

OLEAN

Douglass, Richmond

PATCHOGUE

Finnerty, James

PLATTSBURG

Potter, Robert T.

ROCHESTER

DeWeese, James A.

Emerson, George L.

Schwartz, Seymour I.

Stewart, Scott

Zaroff, Lawrence I.

ROSLYN

Thomson, Norman, Jr.

SARANAC LAKE

Decker, Alfred M., Jr.

Merkel, Carl G.

SEAFORD

Mangiardi, Joseph L.

SOUTHAMPTON

Heroy, William W.

STATEN ISLAND

Garzon, Antonio A.

STONY BROOK

Dennis, Clarence

Soroff, Harry S.

SYRACUSE

Bredenberg, Carl E.

Effler, Donald B.

Meyer, John A.

Parker, Frederick, Jr.

Sealy, Will C.

Smith, David T.

Wechsler, Andrew S.

Wolfe, Walter G.

Young, W. Glenn, Jr.

GREENSBORO

Deaton, W. Ralph, Jr.

PINEHURST

Fischer, Walter W.

WINSTON-SALEM

Cordell, A. Robert

Hudspeth, Allen S.

Johnston, Frank R.

Meredith, Jesse H.

NORTH DAKOTA

GRAND FORKS

James, Edwin C.

OHIO

AKRON

Falor, William H.

CHARDON

Mautz, Frederick R.

CINCINNATI

Carter, B. Noland

Gonzalez, Luis L.

Helmsworth, James A.

Rosenkrantz, Jens G.

Wright, Creighton B.

CLEVELAND

Ankeney, Jay L.

Cosgrove, Delos M.

Cross, Frederick S.

Groves, Laurence K.

Kay, Earle B.

Loop, Floyd

COLUMBUS

Clatworthy, H. W., Jr.

Kilman, James W.

McEnany, M. Terry

Meckstroth, Charles

Vasko, John S.

Williams, Thomas, Jr.

DAYTON

Dewall, Richard A.

PEPPER PIKE

Mendelsohn, Harvey J.

TOLEDO

Blakemore, William S.

OKLAHOMA

OKLAHOMA CITY

Elkins, Ronald C.

Felton, Warren L., II

Fisher, R. Darryl

Greer, Allen E.

Munnell, Edward R.

Wilder, Robert J.

Williams, G. Rainey

Wilson III, Hugh E.

Zuhdi, M. Nazih

TULSA

Guernsey, James M.

McPhail, Jasper L.

DARBY

McKeown, John J., Jr.

FAIRFIELD

McClenathan, James E.

GLADWYNE

Johnson, Julian

HAMBURG

Judd, Archibald R.

HAVERTOWN

Chodoff, Richard J.

HERSHEY

Demuth, William, Jr.

Pierce, William S.

Waldhausen, John A.

LANCASTER

Witmer, Robert H.

LUMBERVILLE

O'Neill, Thomas J. E.

PHILADELPHIA

Brockman, Stanley K.

Edie, Richard N.

Edmunds, L. Henry, Jr.

Fineberg, Charles

Haupt, George J.

Lemmon, William M.

Lemole, Gerald M.

MacVaugh, Horace, III

Mendelssohn, Edwin

Mundth, Eldred D.

Nemir, Paul, Jr.

Rosemond, George P.

Templeton, John, III

Wallace, Herbert W.

PITTSBURGH

Tyras, Denis H.

OREGON

DAYS CREEK

Miller, Arthur C.

PORTLAND

Lawrence, G. Hugh

Okies, J. Edward

Poppe, J. Karl

Starr, Albert

PENNSYLVANIA

ABINGTON

Frobese, Alfred S.

BETHLEHEM

Snyder, John M.

BUCK HILL FALLS

Thompson, Samuel A.

Bahnson, Henry T.

Ford, William B.

Magovern, George J.

Pontius, Robert G.

Rams, James J.

Ravitch, Mark M.

SAYRE

Sewell, William H.

WYNNEWOOD

Harken, Alden H.

YARDLEY

Sommer, George N., Jr.

RHODE ISLAND

PROVIDENCE

Karlson, Karl E.

Simeone, Fiorindo A.

SOUTH CAROLINA

CHARLESTON

Bradham, Randolph R.

Hairston, Peter

Parker, Edward F.

Sade, Robert M.

COLUMBIA

Almond, Carl H.

LANDRUM

Stayman, Joseph W.

STATE PARK

Ryan, Thomas

TENNESSEE

CHATTANOOGA

Adams, Jesse E., Jr.

Hall, David P.

JACKSON

Chandler, John H.

BEAUMONT

Harrison, Albert W.

BURNET

Ross, Raleigh R.

DALLAS

Adam, Maurice

Davis, Milton V.

Holland, Robert H.

Kee, John L., Jr.

Lambert, Cary J.

Mills, Lawrence J.

Mitchel, Ben F., Jr.

Paulson, Donald L.

Platt, Melvin R.

Razzuk, Maruf A.

Seybold, William D.

Shaw, Robert R.

Sugg, Winfred L.

JOHNSON CITY

Bryant, Lester R.

Lefemine, Armand A.

KNOXVILLE

Blake, Hu Al

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.

NASHVILLE

Alford, William, Jr.

Bender, Harvey W., Jr.

Dale, W. Andrew

Foster, John H.

Gobbel, Walter G., Jr.

Johnson, Hollis E.

Sawyers, John L.

Scott, Henry W., Jr.

Stoney, William S.

Thomas, Clarence, Jr.

SEWANEE

Thrower, Wendell

TEXAS

AMARILLO

Sutherland, R. Duncan

Urschel, Harold, Jr

DILLEY

Hood, Richard H., Jr.

FORT SAM HOUSTON

Strevey, Tracy E., Jr.

GALVESTON

Derrick, John R.

HOUSTON

Beall, Arthur C, Jr.

Burdette, Walter J.

Cooley, Denton A.

Crawford, E. Stanley

De Bakey, Michael E.

Hallman, Grady L., Jr.

Henly, Walter S.

Lawrie, Gerald M.

Mattox, Kenneth L.

Morris, George C., Jr.

Mountain, Clifton F.

Norman, John C.

Overstreet, John W.

Reul, George J., Jr.

Wukasch, Don C.

LUBBOCK

Bricker, Donald L.

Dalton, Martin L., Jr.

SAN ANTONIO

Dooley, Byron N.

French, Sanford, III

Grover, Frederick L.

Heaney, John P.

Nixon, James W.

Proctor, Oscar S.

Trinkle, J. Kent

TEMPLE

Brindley, G. V., Jr.

UTAH

SALT LAKE CITY

Cutler, Preston R.

Johnson, Clive R.

Liddle, Harold V.

Mortensen, J. D.

Nelson, Russell M.

Wolcott, Mark W.

VERMONT

BRATTLEBORO

Gross, Robert E.

BURLINGTON

Coffin, Laurence H.

Miller, Donald B.

CHESTER DEPOT

Adams, Herbert D.

VIRGINIA

ARLINGTON

Conrad, Peter W.

Klepser, Roy G.

CHARLOTTESVILLE

Crosby, Ivan K.

Dammann, John F.

Minor, George R.

Muller, William, Jr.

Nolan, Stanton P.

Wellons, Harry A., Jr.

LYNCHBURG

Moore, Richmond L.

RICHMOND

Bosher, Lewis H., Jr.

Brooks, James W.

Cole, Dean B.

Greenfield, Lazar J.

Gwathmey, Owen

Mansfield, Peter B.

Miller, Donald W., Jr.

Pinkham, Roland D.

Rittenhouse, Edward

Sauvage, Lester

Thomas, George I.

SPOKANE

Berg, Ralph, Jr.

WEST VIRGINIA

E. CHARLESTON

Walker, James H.

HUNTINGTON

Littlefield, James B.

MORGANTOWN

Tarnay, Thomas J.

Warden, Herbert E.

WISCONSIN

LA CROSSE

Gundersen, Erik A.

MADISON

Chopra, Paramjeet S.

Young, William P.

MARSHFIELD

Myers, William O.

Ray, Jefferson F., Ill

Sautter, Richard D.

MILWAUKEE

Bonchek, Lawrence I.

Flemma, Robert J.

Hausmann, Paul F.

Johnson, W. Dudley

Lepley, Derward, Jr.

Litwin, S. Bert

Mullen, Donald C.

Narodick, Benjamin

Johns, Thomas N. P.

Lower, Richard R.

WASHINGTON

BELLINGHAM

Varco, Richard L.

FRIDAY HARBOR

Fox, Robert T.

MERCER ISLAND

Mills, Waldo O.

SEATTLE

Anderson, Richard P.

Cantrell, James R.

Dillard, David H.

Hill, Lucius D., III

Jarvis, Fred J.

Jones, Thomas W.

Manhas, Dev R.

Olinger, Gordon N.

Tector, Alfred J.

Weisel, Wilson

WAUSAU

Davila, Julio C.

WEST BEND

Gardner, Robert J.

WYOMING

TETON VILLAGE

Kaunitz, Victor H.

CANADA

ALBERTA

CALGARY

Miller, George E.

EDMONTON

Callaghan, John C.

Meltzer, Herbert

Sterns, Laurence P.

BRITISH COLUMBIA

KELOWNA

Couves, Cecil M.

VANCOUVER

Allen, Peter

Ashmore, Phillip G.

Harrison, Elliott

Tyers, G. Frank O.

VICTORIA

Stenstrom, John D.

W. VANCOUVER

Robertson, Ross

MANITOBA

OTTAWA

Keon, Wilbert J.

Key, James A.

SUDBURY

Field, Paul

Walker, George R.

TORONTO

Baird, Ronald J.

Bigelow, Wilfred G.

Cooper, Joel D.

Ginsberg, Robert J.

Goldman, Bernard S.

Henderson, Robert D.

Joynt, George H. C.

Lockwood, A. L.

Mclntosh, Clarence A.

Pearson, F. Griffith

WINNIPEG

Barwinsky, Jaroslaw

Cohen, Morley

NEWFOUNDLAND

ST. JOHNS

Brownrigg, Garrett M.

NOVA SCOTIA

HALIFAX

Murphy, David A.

KENTVILLE

Quinlan, John J.

MABOU

Thomas, Gordon W.

ONTARIO

DORSET

Mustard, William T.

HAMILTON

Sullivan, Herbert J.

LONDON

Heimbecker, Raymond

Scully, Hugh E.

Trimble, Alan S.

Trusler, George A.

WESTBROOK

Lynn, R. Beverley

WOODBRIDGE.

Laird, Robert C

QUEBEC

MONTREAL

Blundell, Peter E.

Bruneau, Jacques

Chiu, Chu-Jeng (Ray)

Dobell, Anthony R.

Grondin, Claude M.

MacLean, Lloyd D.

Morin, Jean E.

Mulder, David S.

Scott, Henry J.

OUTREMONT

Lepage, Gilles

QUEBEC CITY

Gravel, Joffre-Andre

WESTMOUNT

Vineberg, Arthur M.

OTHER COUNTRIES

ARGENTINA

BUENOS AIRES

Favaloro, Rene G.

AUSTRALIA

NO. ADELAIDE

Sutherland, H. D'Arcy

BANGLADESH

DACCA DISTRICT

McCord, Colin W.

BRAZIL

SAO PAULO

JAPAN

YAMAGUCHI

Mohri, Hitoshi

NETHERLANDS

LEIDEN

Brom, Gerard A.

NEW ZEALAND

AUCKLAND

Barratt-Boyes, Sir Brian

PORTUGAL

LISBON

Zerbini, E. J.

ENGLAND

BATH

Belsey, Ronald

BUCKINGHAMSHIRE

Sellors, Sir Thomas

CAMBRIDGE

Moore, Thomas C.

COVENTRY

Smith, Roger Abbey

ESSEX

Kennedy, John H.

HEREFORD

Thompson, Vernon C.

LONDON

Ross, Donald N.

FRANCE

BORDEAUX

Fontan, Francis M.

PARIS

Dubost, Charles

GUATEMALA

GUATEMALA CITY

Herrera, Rodolfo

INDIA

RAIPUTANA

Van Allen, Chester M.

IRELAND

DUBLIN

O'Malley, Eoin

Macedo, Manuel E. M.

SAUDI ARABIA

RIYADH

Kerth, William J.

Merendino, K. Alvin

SCOTLAND

EDINBURGH

Logan, Andrew

SPAIN

SANTANDER

Duran, Carlos Gomez

SWEDEN

STOCKHOLM

Bjork, Viking O.

Crafoord, Clarence

SWITZERLAND

ZURICH

Senning, Ake

GENOLIER

Hahn, Charles J.

VENEZUELA

CARACAS

Tricerri, Fernando E.

WEST GERMANY

AACHEN

Messmer, Bruno J.

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

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

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 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 mem-* bers 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 60 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 reelected to succeed himself. Any Councilor so elected shall take office upon the conclusion of the annual meeting at which he is elected.

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

ARTICLE V. Officers

Section 1. The officers of the Association shall be 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 reelected 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 reelected 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.

ARTICLE VII Finances

Section 1. The fiscal year of the Association shall begin on the first day of March and end on the last day of February 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 of Directors and Officers

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

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

ARTICLE X. Papers

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

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

ARTICLE XI. Initiation Fees, Dues and Assessments

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

Section 2. Annual dues for Active Members shall be $75.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Annual dues for Associate Members shall be $75.00 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.

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

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWS

1st	1951-52	L. L. Whytehead, M.D., F.R.C.S. 790 Sherbrooke St., Winnipeg 2, Manitoba, 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	TheodorM. Scheinin, M.D. Oulun Laaninsairaala, Oulu, FINLAND
11th	1963-64	Masahiro Saigusa, M.D. Department of Surgery, Tokyo University School of Medicine 1 Motofuji-cho, Bunkyo-Ku, Tokyo, 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. Brompton Hospital, London, S.W. 3, ENGLAND
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S. University Hospital A.H.E.P.A., Surgical Clinic Department Aristotelian University of Thessaloniki, Thessaloniki, GREECE
15th	1965-66	Gerhard Friehs, M.D. Chirugische University Klinik, Graz, AUSTRIA
16th	1965-66	Ary Blesovsky, M.D. London, ENGLAND
17th	1966-67	C. Peter Clarke, F.R.A.C.S. Cardiac Surgeon, The Royal Childrens Hospital, Flemington Road, Parkville, Vic. 3052 AUSTRALIA
18th	1966-67	G. B. Parulkar, M.D. Thoracic and Cardiovascular Center, K.E.M. Hospital, Parel, Bombay 12, INDIA
19th	1967-68	Claus Jessen, M.D. Surg. Dept. D, Rigshospitalet, Blegdamsvej 9, Copenhagen, DENMARK
20th	1969-70	Peter E. Bruecke, M.D. A-1090 Vienna, Alserstrasse 4, 1st Surgical Clinic, Vienna, AUSTRIA
21st	1970-71	Michel S. Slim, M.D. Department of Surgery, American University Hospital, Beirut, LEBANON
22nd	1971-72	Seven Pellervo Mattila, M.D. Department of Thoracic Surgery, Helsinki University Central Hospital, 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. 41 rue Louvrex, Liege B-4000, BELGIUM
25th	1974-75	J. J. DeWet Lubbe, M.D. Dept. of Cardio-Thoracic Surgery, University of Stellenbosch P. O. Box 53 Bellville, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner, M.D. Institute of Surgery Debinski, POLAND
27th	1976-77	Bum Koo Cho, M.D. Yonsei University P.O. Box 71 Severance Hospital Seoul, KOREA
28th	1977-78	Alan William Gale, M.D., FRACP, FRACS. St. Vincents Medical Centre 376-382 Victoria St. Darlinghurst 2010 AUSTRALIA
29th	1978-79	Eduardo Otero Coto, M.D. Servicio de Cirugia Cardiovascular Ciudad Sanitaria "Le Fe" Valencia. SPAIN
30th	1981-82	Richard Firmin, M.D. 20 Andrew Drive, Apt. 63 Tiburon, California 94920
31st	1981-82	Claudio A. Salles, M.D. 1489 Wisteria Ann Arbor, Michigan 47104
32nd	1982-83	Yasuhisa Shimazaki, M.D.

1st Department of Surgery
Osaka University Medical School
1-1-50, Fukushima, Fukushima-ku
Osaka 553 Japan