

## 1980 ANNUAL MEETING PROGRAM



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**American Association for Thoracic Surgery  
60TH ANNUAL MEETING  
Scientific Program**

**MONDAY MORNING, APRIL 28, 1980**

**8:30 A.M. Business Session (Limited to Members)  
Continental Ballroom**

**8:45 A.M. Scientific Session  
Continental Ballroom**

**1. Myocardial Energy Replenishment and Reversal of Ischemic Damage by Substrate  
Enhancement with Amino Acids during Reperfusion**

HAROLD LAZAR\*, ANDREW M. MANGANARO\*,

HEINZ BECKER\* and GERALD D. BUCKBERG,

**Los Angeles, California**

We have shown previously that ischemia depletes myocardial energy stores and limits recovery by impairing the post-ischemic oxidative metabolism which repletes them. This study tests the hypothesis that near normal post-ischemic oxidative metabolism and functional recovery is possible by adding the amino acid L-glutamate to the blood cardioplegic solution used to lower oxygen demands during reperfusion.

Methods: Of 20 dogs undergoing 45 minutes of 37°C ischemic arrest, no heart could support the systemic circulation 15 minutes after unclamping. We prolonged bypass 30 minutes in all dogs; in 13 we lowered O<sub>2</sub> demands further by reoccluding the aorta for 10 minutes while continually infusing at 37° blood potassium cardioplegic solution. In 8 dogs, we added L-glutamate (0.026 M) to this solution. Coronary blood flow (micro-spheres), ATP, metabolism (O<sub>2</sub> content), and LV performance (Starling curves) were measured during control and at 15 and 45 minutes after unclamping.

Results: The lowest post-ischemic ATP (2.2 μM/gm) and least functional recovery (35% return of stroke work index) occurred in hearts not receiving cardioplegia during prolonged bypass. In

contrast, adding L-glutamate to the blood cardioplegic solution allowed better ATP repletion (4.2 vs 3.2  $\mu\text{M}/\text{gm}^*$ ), greater augmentation of LV subendocardial flow (85 vs 71%\*) and  $\text{O}_2$  uptake (108 vs 83%\*) during the working state, and higher stroke work indices (1.20 vs 0.74 g-m/kg, 85 vs 55%\* recovery) than with blood cardioplegia alone.

Conclusion: Adding L-glutamate to blood cardioplegia causes near complete reversal of ischemic damage, possibly through restoring ATP by stimulating oxidative metabolism and replenishing a vital Krebs cycle intermediate lost during ischemia. We believe L-glutamate will become an important component of future cardioplegic solutions.

\* $p < .05$  from hearts receiving L-glutamate

\*By invitation

## 2. Adenosine Metabolism and Myocardial Preservation

JOHN E. FOKER\*, STANLEY EINZIG\* and TING WANG\*,

**Minneapolis, Minnesota**

Sponsored by: Robert W. Anderson, Minneapolis, Minn.

The nature of the metabolic events leading to irreversible damage of the ischemic myocardium are not known. We tested the hypothesis that catabolism of ATP precursors limits the regeneration of this high energy compound following ischemic insult. Dogs on cardiopulmonary bypass (CPB) had their aortas cross-clamped (XC) for 20 min at normothermia and 30 min later CPB was discontinued. Rapidly frozen left ventricular biopsies were assayed and control levels ( $\mu\text{m}/\text{gm}$ ) found for ATP (5.30) and creatine phosphate (CP) (6.18) and the ATP precursors ADP (0.82) and AMP (0.43). Measurable AMP degradation did not occur in the control state and adenosine (Ad), inosine and hypoxanthine/xanthine (Hx/x) were not detectable. At the end of XC, ATP had fallen to 3.60 and CP to 0.57, however, the predicted rise in the levels of ADP, AMP and Ad was not found. The levels of inosine (1.23) and Hx/x (0.24) increased indicating ADP, AMP and Ad had been further catabolised and limited the potential for ATP recovery. Following XC release, ATP levels did not increase and even 60 min off CPB were only 3.27. CP levels rose from 0.57 during XC to 10.2 within 5 min after XC, indicating high energy bonds could be formed. To test the effect of blocking Ad catabolism, EHNA (10 mg/k), an inhibitor of adenosine deaminase was used in 5 dogs. At the end of XC, tissue Ad, previously unmeasurable, was 1.60  $\mu\text{m}/\text{gm}$ . Nevertheless, ATP levels did not rebound after XC release; the reason was found to be cellular loss and the coronary sinus blood contained high levels of Ad. Adenosine (20 mg/k) infusion alone was used to equilibrate intra- and extracellular levels during CPB in 5 dogs. This did not alter subsequent ATP levels so we combined EHNA treatment with Ad infusion (5 dogs). ATP levels showed good recovery and 30 min after XC were 4.68 and 60 min off CPB were 4.73. We conclude that ATP regeneration after ischemia is limited by the availability of ADP, AMP and Ad. Inhibition of Ad catabolism and infusion of Ad will enhance ATP return from ischemia. Current methods of myocardial preservation, chiefly hypothermia and cardioplegia, are designed to decrease ATP utilization. Our approach, by inhibiting an important biochemical consequence of ischemia, may improve myocardial preservation by providing precursors for ATP recovery.

\*By invitation

### 3. Relationship Between Atrio-Ventricular Arrhythmias and the Concentration of K<sup>(+)</sup> Ion in Cardioplegic Solution

ROBERT ELLIS\*, CONSTANTINE MAVROUDIS\*,  
 DANIEL ULLYOT, KEVIN TURLEY\* and PAUL EBERT,  
 San Francisco, California

Several centers have noted a high incidence of atrial arrhythmias following the use of potassium cardioplegia during cardiac surgery. A spectrum of arrhythmias such as atrio-ventricular dissociation, nodal rhythm, and either right or left hemiblock conduction defects have been observed at the termination of cardiopulmonary bypass. Although these are transient, they can have deleterious effects on the hemodynamic stability of the patient. The etiology of these arrhythmias is postulated to be due to one or a combination of the following factors: Anoxic myocardium, inadequate cooling of the right atrium, air in the A-V nodal artery or action potential changes in the conduction system induced by concentration of K<sup>(+)</sup> ion. We have recently compared the incidence of intraoperative arrhythmias using Holler monitoring in three groups of patients (20 each) where the concentration of K<sup>(+)</sup> ion was varied. Total revascularization was attempted with a mean of 3.2 grafts/pt and a mean cardioplegic arrest time of 55 ± 4 minutes. Patients had their cardiac rhythm taped using a Holter monitor throughout the cardiac procedure. Group I used 20meq K<sup>(+)</sup>/L/4°C as the perfusate with repeated infusions of this same solution. Group II used only 5meq K<sup>(+)</sup>/L/4°C as perfusate both initially and for subsequent infusions. Group III used 20meq K<sup>(+)</sup>/L/4°C as the initial perfusate and 5meq/L for subsequent infusions. All patients had an initial infusion of 800cc of cardioplegic fluid followed by a 400cc infusion after each distal anastomoses.

	High Energy Phosphates* (CP + ATP)	p Value	Arrhythmia
Group I	48.73		60% (12/20)
Group II	44.39	ns	5% (1/20)
Group III	47.45	ns	5% (1/20)

\*µm/G dry weight

These results indicate that myocardial HEP was preserved in all three groups. The incidence of arrhythmias in the immediate post arrest period in Group I was 60% whereas it was only 5% in Groups II and III. These data suggest that after the initial infusion of high K<sup>(+)</sup> solution, subsequent infusions could be with low potassium cardioplegia to avoid the arrhythmias noted with the repeated use of high potassium cardioplegic solution.

\*By invitation

### 4. Comparison of the Effect of Blood Cardioplegia to Crystalloid Cardioplegia on Human Myocardial Contractility

NA  
 MARVIN KIRSH, and KENNETH JOCHIM\*,  
 Ann Arbor, Michigan

Cardioplegic solutions (CP) of various compositions are advocated for myocardial protection during aortic clamping. However, no controlled quantitative measurements have been made on their comparative effect on human myocardial function.

Thirty-three patients (pts) undergoing coronary bypass grafting were randomly assigned to 1 of 3 groups: Group KCP (n = 9) received crystalloid CP (Ringers, 24 meqK VI, 12.5 gm mannitol/l, 4 meqNaHCO<sub>3</sub>/l). Group MgKCP (n = 9) received a different crystalloid CP (Mg<sup>++</sup> 30 meq/l, K<sup>+</sup> + 20 meq/l). Group BCP (n= 15) received blood CP (Hct 20%, K<sup>+</sup> = 30 meq/l). In each patient, 1 liter of 4°C CP sol. was given followed by 500 ml every 30 min. All distal anastomosis were completed during one continuous aortic cross-clamping. In each pt cardiac output (CO), LVEDP and contractile element velocity (Vpm) were recorded intraoperatively before and after aortic crossclamp period. Vpm was recorded from a Millar catheter placed in the LV through apical stab wound. Heart rate was held constant by atrial pacing during recordings. The 3 groups were comparable in regards to age, sex, ejection fraction, symptoms, propranolol use, number of bypasses performed (av. 2.6) and duration of ischemic arrest (av. 52 min).

Preoperative LV function assessed by CO, LVEDP and Vpm was similar in all 3 groups. No significant differences in myocardial function were observed after ischemia with BCP or KCP. However, pts receiving MgKCP had significant (P = 0.02) depression in Vpm (from 28.6±7.8 sec<sup>-1</sup> to 20.4±3.6 sec<sup>-1</sup>) and increase (P<0.05) in LVEDP (from 9.4 ± 2.2 mmHg to 13.4 ± 5.2 mmHg). Postoperative Vpm of MgKCP group was also significantly depressed (P <0.05) by comparison with BCP group. 89% of pts receiving KCP or MgKCP but only 40% of BCP pts required electrical defibrillation. There were no deaths, and only one patient sustained peri-operative infarction (in KCP group).

We conclude that blood Cardioplegia offers no advantage in degree of myocardial protection compared to simple high K<sup>+</sup> solution, although fewer patients required electrical defibrillation. MgKCP solution yielded inferior results.

\*By invitation

## **5. Heart and Lung Transplantation: Auto and Allotransplantation in Primates with Extended Survival**

**BRUCE A. REITZ,\* NELSON A. BURTON,\*  
STUART W. JAMIESON,\* JOHN L. PENNOCK,\***

EDWARD B. STINSON,\* and NORMAN E. SHUMWAY

### **Stanford, California**

A number of severely ill patients with congenital or acquired disease could be treated by transplantation of the heart and both lungs. In order to study this possibility, heart and lung transplantation (HLT<sub>x</sub>) was performed in 25 monkeys (rhesus or cynomolgous) divided into two groups.

Group I (17 animals weighing 2.6-6.5 kg) underwent HLT<sub>x</sub> with surface-induced deep hypothermia (17-20°C), circulatory arrest (58-94 min), and surface rewarming. The phrenic nerves were preserved on pedicles and anastomoses were performed to the trachea, ascending aorta, and both vena cavae. Group IA (5 animals) had auto-HLT<sub>x</sub> with one long-term survivor (now 213 days). Group IB (12 animals) had allo-HLT<sub>x</sub> with 9 resuming spontaneous respiration, 6 surviving greater than 24 hours, and one surviving 5 days before dying of rejection. Group II (8 animals weighing 4.9-8.8 kg) had HLT<sub>x</sub> with cardiopulmonary bypass. A similar operative technique was

used with all cannulations performed in the thorax. Group IIA (2 animals) had auto-HLTx with both surviving at 49 and 157 days. Group IIB (6 animals) had allo-HLTx with all surviving more than 24 hours with spontaneous and normal respiratory patterns. The 3 animals surviving more than 48 hours were started on cyclosporin A (25 mg/kg/day) and azathioprine (2 mg/kg/day for 14 days) with 2 currently surviving at 110 and 41 days without rejection. These experiments demonstrate extended survival of primates after complete heart and lung transplantation. The allografts on cyclosporin and azathioprine are the longest reported survivors after this procedure.

#### INTERMISSION - 45 MINUTES

#### VISIT EXHIBITS

\*By invitation

### **6. Comparison of Standard Aneurysmectomy and Aneurysmectomy with Endocardial Resection for the Treatment of Recurrent Sustained Ventricular Tachycardia**

ALDEN H. HARKEN\*, LEONARD N. HOROWITZ\*,  
and MARK E. JOSEPHSON\*, Philadelphia, Pennsylvania

Sponsored by Dwight E. Harken, Boston, Massachusetts

Failure of aneurysmectomy to ablate ventricular tachycardia may be due to incomplete removal of the tachycardia site. We have developed an in-traoperative catheter mapping technique coupled with endocardial resection \* of sites demonstrated to be the origin of ventricular tachycardia. The value of intraoperative mapping and endocardial resection in addition to standard aneurysmectomy was compared to standard aneurysmectomy alone in 36 patients with recurrent sustained, medically refractory, ventricular tachycardia. Sixteen patients (group 1) ranging in age from 42 to 66 underwent standard aneurysmectomy alone (1971-1975) and 20 patients (group 2) ranging in age from 23 to 68 years underwent mapping and endocardial resection with or without aneurysmectomy (1976-1979). All 36 patients had coronary artery disease and prior myocardial infarction but only 85% of group 2 patients had left ventricular aneurysms. In group 1, mean ejection fraction was 32% (range 10-42%) and cardiac index was 2.2 L/Min/M<sup>2</sup>. Group 2 patients had an ejection fraction of 28% (5-39%) and cardiac index of 2.0 L/Min/M<sup>2</sup>. In group 1, there were 5 operative deaths (31%), 3 of recurrent ventricular tachycardia. Six survivors had recurrent ventricular tachycardia, 4 (25%) of whom subsequently died of their arrhythmia. The remaining 2 are controlled by anti-arrhythmic therapy. Five patients in group 1 have had no ventricular tachycardia in the absence of anti-arrhythmic medication. In group 2, intraoperative catheter mapping localized the origin of the tachycardia to a border of the infarction or aneurysm in all instances. At surgery, ventricular tachycardia persisted following resection of the ventricular aneurysm or ventriculotomy (prior to the endocardial resection) in 18 out of 20 patients. Following endocardial resection, 16 patients remain free of their ventricular tachycardia, 2 patients' tachycardia are now controllable on anti-arrhythmic therapy, and there were 2 (10%) operative deaths. We conclude that surgical therapy of recurrent sustained ventricular tachycardia is feasible by identification of the site of origin of the arrhythmia by intraoperative mapping and appropriately guided endocardial resection.

\*By invitation



## **7. Left Atrial Isolation: A New Technique for The Treatment of Supraventricular Arrhythmias**

J. MARK WILLIAMS\*, ROSS M. UNGERLEIDER\*,

GARY K. LOFLAND\* and JAMES L. COX\*, Durham, N.C.

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

Surgical therapy has been successful in the treatment of ectopic and re-entrant ventricular tachycardia and in re-entrant Supraventricular tachycardia. However, surgical ablation of ectopic Supraventricular arrhythmias, particularly those arising in the left atrium, has been unsuccessful. As a result, it has been necessary to cryoablate the bundle of His and insert a permanent ventricular pacemaker for control of these arrhythmias. It was the purpose of this study to develop a technique to isolate the left atrium electrically from the remainder of the heart, thereby precluding the necessity for an artificial pacemaking system.

Right atrial, left atrial, and ventricular epicardial electrograms were recorded in ten adult dogs prior to institution of cardiopulmonary bypass (CPB) and potassium cardioplegic arrest of the heart. During CPB, a standard left atriotomy was performed and was extended anteriorly across the mitral valve annulus between the right and left fibrous trigones. Posteriorly, the atriotomy was extended across the mitral valve annulus just to the left of the posterior crux and interatrial septum. The muscular interatrial fibers accompanying the coronary sinus were cryoablated at minus 60°C for two minutes. The atriotomy was closed and the animals were weaned from CPB.

Postoperatively, all animals remained in normal sinus rhythm. Neither rapid left atrial pacing nor left atrial fibrillation affected the rate or rhythm of the remainder of the heart. Preliminary hemodynamic measurements suggest that loss of the synchronous left atrial "kick" does not significantly affect left ventricular preload, afterload, or cardiac output.

This technique offers an alternative to the current surgical approach for treatment of refractory ectopic Supraventricular tachycardia arising in the left atrium. Moreover, it may ultimately become useful as an adjunctive treatment for chronic atrial fibrillation associated with mitral valve disease in patients requiring mitral valve replacement.

**11:30 A.M.**

**Presidential Address**

**LET US NOW PRAISE FAMOUS MEN**

**Herbert Sloan**

\*By invitation

## MONDAY AFTERNOON, April 28, 1980

2:00 P.M. Scientific Session

Continental Ballroom

### 8. A Prospective Study of 461 Lung Carcinomas with Positive Mediastinal Lymph Nodes

*NAEL MARTINI, MUHAMMAD B. ZAMAN\*and*

*EDWARD J. BEATTIE, JR., New York, N. Y.*

From 1974 to 1978, 989 patients with carcinoma of the lung were treated at our institution. All were staged according to the A.J.C. staging system. Of these, 461 patients had clinically evident mediastinal lymph node metastases. 208 were considered inoperable at diagnosis, based on histology of oat cell carcinoma, known distant metastases, or malignant effusion. The remaining 253 patients were surgically explored and treated by resection and/or implantation. 176 (70%) of this latter group had either palliative resection or implantation of their disease. The median survival in this group of patients was 9 months.

There were 77 patients who had complete, potentially curative resection of their primary tumor and all accessible mediastinal lymph nodes. The vast majority of these were also treated by postoperative external radiation therapy to the mediastinum. In this group, there were 28 epidermoid carcinomas, 29 adenocarcinomas, 13 bronchiolar carcinomas, and 7 large cell carcinomas. At one year of follow-up 53 of 77 patients (69%) were alive and well. 56 patients were at risk for 2 years, and 32 of these (57%) are alive, and 41 patients were at risk for 3 years and 18 (44%) are alive. Survival at 3 years was slightly better in glandular carcinoma (adenocarcinoma or bronchiolar) than in epidermoid carcinoma (56% versus 40%). We conclude that there is a select group of patients with mediastinal lymph node metastases who can be effectively treated by combined resection and radiation therapy with prolonged survival.

\*By invitation

### 9. Pathologic Stage Grouping of the Patients with Resected Carcinoma of the Lung

*THOMAS W. SHIELDS, EDWARD W. HUMPHREY,*

*CHARLES E. EASTERIDGE, MARY MATTHEWS\*,*

*ROBERT J. KEEHN\*, Chicago, Illinois; Minneapolis, Minnesota;*

*Memphis, Tennessee and Washington, D. C.*

The data from a new series of 569 patients with "curative" resection of non-oat cell tumors were analyzed by the life table method to evaluate the validity of the postresection pathologic staging classification suggested by the American Joint Committee. The cell types were: squamous: 305, adenocarcinoma: 172, large cell: 73 and mixed: 19. Each patient was assigned a pathologic TN classification on examination of the resected specimen (all patients were judged clinically to have no distant metastases-M<sub>0</sub>). There were 173 lesions classified as T<sub>1</sub>N<sub>0</sub>; 37 as T<sub>1</sub>N<sub>1</sub>; 212 as T<sub>2</sub>N<sub>0</sub>; 115 as T<sub>2</sub>N<sub>1</sub> and 32 with either T<sub>3</sub> with any N, or N<sub>2</sub> with any T. In analyzing the data, a subset of lesions (25) initially staged as T<sub>2</sub>N<sub>0</sub> was identified which should be pathologically staged as T<sub>1</sub>N<sub>0</sub>. These are small central lesions, 3 cm or less in size, located distal to a lobar take-off. Regardless of the presence of atelectasis or pneumonitis to the hilar area, these behave as do the more peripherally located lesions of similar size. Three year survival of 66.5 and 68.5 percent respectively were noted in these patients as compared to 53.6 percent for the patients with lesions greater than 3 cm in size

which could be classified as T<sub>2</sub>N<sub>0</sub> regardless of their location. When lymph nodes were positive (N<sub>1</sub>) such small central lesions (20) had a better survival than either of the other T<sub>1</sub>N<sub>1</sub> or T<sub>2</sub>N<sub>1</sub> lesions. It is therefore suggested that all small central lesion, 3 cm or less in size, distal to a lobar take-off be considered a T<sub>1</sub> lesion. In addition it was noted that patients with T<sub>1</sub>N<sub>1</sub> lesions had a three year survival of only 36.7 percent, which is similar to that of those with T<sub>2</sub>N<sub>1</sub> lesions (39.8%). Therefore, it would appear more appropriate to classify these patients as having Stage II rather than Stage I disease. The other patients in Stage I have a much better survival experience: T<sub>1</sub>N<sub>0</sub> lesions have three and five year survivals of 68.5 and 54.4 percent, and those with T<sub>2</sub>N<sub>0</sub> lesions 53.6 and 40.0 percent respectively.

\*By invitation

#### **10. Diagnosis, Stage and Prognosis of Lung Carcinoma by Preoperative Assay of Lung Tumor Antigen-Sensitive T-Lymphocytes**

*WILLIAM G. RAMEY\*, GEORGE A. HASHIM\*,  
ANTOINE S. MUNTHER\*, ALEXANDER J. SWISTEL\*,  
WARREN B. BURROWS\*, and HUGH F. FITZPATRICK,  
New York, New York*

Levels of circulating T-lymphocytes sensitized to human lung tumor-associated antigens (LTA) were correlated with lung carcinoma detection, pathological tumor stage, and patient survival in preoperative studies of 62 patients by the antigen-stimulated active rosette-forming T-cell (AgARFC) assay. Incubation with LTA of peripheral blood lymphocytes (PEL) from patients found to have lung cancer produced increased AgARFC compared to incubation without LTA or with breast tumor extracts. Significant levels of LTA-sensitive T-cells were found in preoperative PEL of 80% of Stage I (8/10; p<0.005), 67% of Stage II (4/6; p<0.05), and 50% of Stage III (8/16) primary lung cancer patients, compared with 15.4% of patients with either benign lung lesions (2/9) or lung metastases (0/4) of nonpulmonary malignancies (by chi-square analysis). Postoperative survival correlated significantly with preoperative levels of LTA-sensitive T-cells by AgARFC assay within Stage I (r = 0.865; p<0.005), Stage I-II (r = 0.676; p<0.005), and Stage III (r = 0.550; p<0.05) groups of lung cancer patients. Preoperative PEL from patients with Stage I-II lung cancer were more frequently sensitized to LTA in the AgARFC assay than PBL of patients with nonpulmonary carcinomas (0/11; p< 0.005) or of smokers without pulmonary lesions (1/6; p<0.025). These findings demonstrate a high rate of detection of early, resectable lung carcinomas by preoperative AgARFC assay of PBL sensitization to LTA, and a significant correlation of LTA-sensitive T-cell level with tumor stage and patient survival. The AgARFC assay may be of prognostic as well as diagnostic value in the evaluation of patients with lung carcinoma.

\*By invitation

#### **11. Follow-up Report on Regional BCG Treatment of Lung Cancer**

*MARTIN F. McKNEALLY, JOSEPH B. McILDUFF\*,  
HARVEY W. KAUSEL, RALPH D. ALLEY, CAROLE MAVER\*,  
and LLOYD LININGER\*, Albany, New York*

Four years ago, our group reported the preliminary results of a trial of adjuvant intrapleural BCG following surgical resection of lung cancer. A total of 161 resected patients have entered this randomized trial. We now report the long term follow-up of this trial, including our early

experience with the use of cutaneous BCG restimulation after intrapleural treatment in Stage I patients.

These studies lead us to conclude that: 1) Intrapleural BCG prolonged the time to recurrence for patients with Stage I lung cancer in the study interval 1973-1977. The fact that cancer has recurred in 9 of the 30 BCG treated Stage I patients suggests that the benefit described in our initial report probably was not an artifact related to maldistribution of surgically curable cases in this group. 2) No benefit was demonstrable in Stage II, III patients. 3) The addition of cutaneous restimulation may increase the efficacy of intrapleural BCG.

\*By invitation

## **12. The Clinical Value of Quantitative Ventilation Perfusion Lung Scans in the Surgical Management of Bronchogenic Carcinoma**

*JORGE A. WERNLY\*, P. DAVID MYEROWITZ\*,*

*PETER T. KIRCHNER\*, DAVID E. OXFORD\*,*

*CORINNE A. SOVIK\*, HARVEY M. GOLOMB\*,*

*TOM R. DEMEESTER, Madison, Wisconsin and Chicago, Illinois*

Quantitative ventilation (Xe 133) and Perfusion (Tc99) lung scans were done in 22 patients requiring Pneumonectomy in order to predict post-operatives FEV<sub>1</sub>. The contribution of the lung to be resected and therefore the expected reduction in postoperative pulmonary function, was calculated from both the ventilation and perfusion scans using two different multiple regression equations. The predicted post-operative FEV<sub>1</sub> were derived by subtracting the calculated values from the pre-operative FEV<sub>1</sub>, and compared to the actual post-operative FEV<sub>1</sub> measured four weeks after pneumonectomy. Post-operative FEV<sub>1</sub> could be better predicted using the perfusion scan (coefficient of correlation = 0.85, standard error = 0.19) than using the ventilation scan (coefficient of correlation = 0.82, standard error = 0.22). There was a mean percent error of  $9 \pm 8\%$  for the FEV<sub>1</sub> values predicted on the basis of the perfusion and  $11.6 \pm 9\%$  for the FEV<sub>1</sub> values calculated on the basis of the ventilation.

Based on these findings, all patients requiring pneumonectomy had pre-operative pulmonary function test and quantitative perfusion lung scans in an effort to predict post-operative pulmonary function. Patients with a predicted FEV<sub>1</sub> value of less than one liter/sec, were considered medically inoperable. Thirty-four patients with a predicted post-operative FEV<sub>1</sub> greater than one liter/sec underwent pneumonectomy. Three operative deaths occurred in this group, one of which was related to pulmonary insufficiency. Seventeen of these patients had a pre-operative FEV<sub>1</sub> of less than two liter/sec and would not have been considered candidates for pneumonectomy if an equal split of pulmonary function was assumed for each lung. There were no deaths or post-operative respiratory insufficiency in these patients.

Similar calculations were used in 36 patients requiring lobectomy. The contribution of the to be resected lobe to the total pulmonary function was estimated in a semiquantitative fashion using the quantitative perfusion scans in order to identify which patients with marginal pulmonary function could tolerate surgical reduction in lung volume.

It is concluded that perfusion scan is a reliable means of predicting postoperative function after pneumonectomy. It allows the surgeon to safely determine which patients with marginal

pulmonary function can receive the benefits of resectional therapy with minimal risk of developing chronic respiratory failure.

### INTERMISSION - 45 MINUTES

#### VISIT EXHIBITS

\*By invitation

### 13. Total Anomalous Pulmonary Venous Connection; Ten Years Experience Including Postoperative Ventricular Function Studies

*JOHN W. HAMMON\**, *HARVEY W. BENDER*,  
*THOMAS P. GRAHAM\** and *HAROLD G. ERATH\**,  
*Nashville, Tennessee*

The surgical experience with total anomalous pulmonary venous connection (TAPVC) since 1969 was reviewed. Twenty-five patients with TAPVC were studied. The average age at operation was 4 months with 17 children less than 3 months of age. All patients had marked pulmonary hypertension and had been treated for congestive heart failure.

Operative mortality was 5/25 (20%). 4 of 5 deaths were in critically ill neonates requiring preoperative ventilatory support. All operative survivors have been followed for a mean 4.5 years (6 mos.-10 years). There have been no late deaths or reoperations. All children have had normal growth patterns. Ten patients have been recatheterized. Pulmonary artery systolic pressure fell from 79 mm Hg  $\pm$  6.9 before operation, 32 mm Hg  $\pm$  2.6\* post surgery. Pulmonary capillary wedge pressure was slightly elevated at 12 mm Hg at the postoperative evaluation. Ventricular function has been evaluated in eight patients before and after surgery using quantitative angiocardiology. Left ventricular end diastolic volume index (LVEDVI) was markedly depressed at 27.3  $\pm$  ml/m<sup>2</sup> preoperatively, an average 75% of normal values. Postoperatively LVEDVI rose to 58.4  $\pm$  4 ml\*/m<sup>2</sup> which averaged 110% of normal values. Left ventricular ejection fraction was depressed at 51  $\pm$  .04% before operation was within normal limits at 65  $\pm$  .02%\* postoperatively. Left atrial maximal volume index measured postoperatively was found to be 27.9  $\pm$  4.4 ml/m<sup>2</sup> which averages 93% of normal values. Thus, in postoperative survivors left ventricular function is normal and left atrial size is adequate.

\*P < .05 from preoperative

\*By invitation

### 14. Current Results with Mustard's Operation in Isolated Transposition of the Great Arteries

*GEORGE A. TRUSLER*, *WILLIAM G. WILLIAMS\**,  
*TERUO IZUKAWA\** and *PETER M. OLLEY\**, *Toronto, Ontario*

The current wave of enthusiasm for the Senning operation prompted a review of our results with the Mustard procedure. Two hundred and five infants and children with isolated transposition

of the great arteries (TGA) were operated on from 1963 to October 1979. To identify trends, the results in the last 100 patients, were compared with the earlier group of 105 children.

Early mortality was less with only 2 deaths in the recent group compared to 11 deaths in the first group. There were five late deaths in the recent group due to: cardiac failure (2), dysrhythmias, pulmonary venous obstruction and congenital respiratory tract anomaly. Complete pulmonary venous obstruction only occurred once in the last 100 cases but left pulmonary venous obstruction, also a technical problem, occurred twice. Inferior vena caval obstruction was rarely seen at any time but technical changes designed to reduce the incidence of dysrhythmias have increased the incidence of partial superior vena caval obstruction which was identified 9 times in the last 100 patients. The incidence of dysrhythmia is lower and a current prospective study indicates that all of the last 15 infants were in sinus rhythm when discharged from hospital. The most serious late problems are right ventricular failure and tricuspid incompetence.

This review suggests that current results with the Mustard repair are similar to those reported for the Senning repair. The choice of operation would appear to depend on the preference of the surgeon and possibly the age of the infant.

\*By invitation

#### **15. Management of Pulmonary Atresia with Hypoplastic Pulmonary Arteries by Right Ventricular to Pulmonary Arterial Reconstruction**

*J. M. PIEHLER\*, G. K. DANIELSON, R. B. WALLACE,  
D. C. McGOON, R. E. FULTON\* and D. D. MAIR\*,  
Rochester, Minnesota*

The presence of hypoplastic pulmonary arteries in the symptomatic patient with pulmonary atresia and ventricular septal defect (VSD) precludes one-stage complete correction and mandates some type of preliminary palliative procedure. Since 1976, we have palliated 38 patients (pts) by establishment of right ventricular to pulmonary arterial (RV-PA) continuity without closure of the VSD. The ages ranged from 2-54 years (mean 13.3); 12 pts had 15 prior systemic-pulmonary arterial shunts. The mean hemoglobin was 18.1 gm/dl and the mean arterial oxygen saturation was 72%. There were three hospital deaths (8%), all in pts with prior shunts; no deaths occurred in the 26 pts without shunts ( $p = 0.05$ ). All survivors manifested clinical improvement; there was a mean decrease in hemoglobin of 3.6 gm/dl ( $p < 0.001$ ) and a mean increase in arterial oxygen saturation of 15.2% ( $p < 0.001$ ).

Cardiac catheterization, performed in 12 pts 10-26 months (mean 17.2) after operation, revealed a 2.20 fold increase in mean pulmonary artery size ( $p = 0.0001$ ), defined as the ratio of the sum of the diameters of the right and left pulmonary arteries to the diameter of the descending aorta. There was no significant difference between right and left pulmonary arterial enlargement (2.35 and 2.04 fold increase, respectively,  $p > 0.1$ ). Three pts have now undergone completion of correction by placement of a valved RV-PA conduit with patch closure of VSD.

These results were compared with those obtained in a comparable group of 13 pts with pulmonary atresia, VSD, and similarly hypoplastic pulmonary arteries treated initially with systemic-to-pulmonary arterial shunting. The mean pulmonary arterial enlargement of only 1.55 times initial size detected on late restudy (mean 33.4 months postoperatively),  $p = 0.005$ , was statistically less than that of the reconstruction group ( $p < 0.05$ ), was less predictable, and was associated with more pulmonary arterial and anastomotic distortion (46% vs. 8%). The results of RV-PA reconstruction suggest that this is a more satisfactory initial procedure for the management

of the symptomatic patient over 2 years of age who has pulmonary atresia, VSD, and hypoplastic pulmonary arteries.

\*By invitation

## 16. Hypertensive Mechanisms in Coarctation of the Thoracic Aorta

*FREDERICK S. PARKER, JR., BRUCE FARRELL\*,  
DAVID H. P. STREETEN\*, HENRY M. SONDHEIMER\*,  
GUNNAR H. ANDERSON\* and MARIE S. BLACKMAN\*,  
Syracuse, New York*

The renin angiotensin system continues to be implicated in the hypertension associated with coarctation of the thoracic aorta and in the paradoxical hypertension following its definitive repair. Available data, however, remain contradictory and unclear. Depletion of extracellular volume by sodium restriction, loop diuretics and use of specific antagonists of the renin-angiotensin system such as saralasin, an angiotensin II antagonist, have helped unmask abnormal plasma renin activity in other hypertensive states. In an attempt to more clearly define the role of this system in coarctation these methods were applied to a group of preoperative and postoperative patients.

Eight children, ages 5 to 16, with coarctation of the thoracic aorta were given a two day 200 mg sodium diet. Following furosemide administration 2 hour upright plasma renins were obtained, measured by radioimmuno-assay and compared to five normal controls. A standard saralasin (angiotensin II antagonist) test was then performed. Following surgical correction all patients developed paradoxical hypertension. Plasma renins were measured and a repeat saralasin infusion performed within 24 hours of surgery. Results: All preoperative patients developed elevated plasma renin levels ( $22.6 \pm 10$  ng/ml/hr.) compared to controls ( $4.5 \pm 2.0$  ng/ml/hr.)  $p < .01$ . Six of eight patients were saralasin responders preoperatively. (Systolic  $\uparrow 14 \pm 3.5$  mm Hg, diastolic  $\uparrow 11.8 \pm 6.7$  mm Hg.). The same six of eight patients were saralasin responders postoperatively. (Systolic  $\uparrow 16.7 \pm 5.6$  mm Hg, diastolic  $\uparrow 18 \pm 4.6$  mm Hg.). Postoperative plasma renins were also elevated in the same saralasin responders ( $13.9 \pm 6.8$  ng/ml/hr.).

This study strongly suggests that the renin angiotensin system plays an important role in the hypertension associated with coarctation and in the mechanism of paradoxical hypertension following coarctation repair. The combination of a low sodium diet and diuresis appears to unmask increased plasma renin activity in coarctation patients and the saralasin infusion test offers confirmatory evidence.

\*By invitation

## TUESDAY MORNING, APRIL 29, 1980

8:30 A.M. Scientific Session  
Continental Ballroom

### 17. Candida Esophagitis Following Heart Surgery and Short-term Antibiotic Prophylaxis

STEVEN R. GUNDRY\*, A. MICHAEL BORKON\*,

CHARLES L. McINTOSH\* and ANDREW G. MORROW,

*Bethesda, Maryland*

Dysphagia and retrosternal pain are common complaints in patients after heart surgery; most often they result from the median sternotomy and/or endotracheal intubation. While *Candida* esophagitis is a recognized cause of similar symptoms, it is usually not suspected except in immuno-logically compromised hosts. We present four patients, not immunosuppressed or cachectic, who develop persistent dysphagia during recovery from open heart operations; three patients received only four days of pre- and post-operative prophylactic antibiotic treatment with Kefzol® and Keflex®. A naso-gastric tube had been used for less than 24 hours in the post-operative period. The fourth patient developed symptoms following prolonged and varied antibiotic therapy. *Candida* esophagitis was diagnosed by a combination of coexisting oral candidiasis (3/4), roenteno-graphic appearance on barium swallow (4/4), endoscopy (4/4) and biopsy or culture (2/4). Initial therapy consisted of antireflux measures and antacids (4/4), Cimetidine (4/4), oral Nystatin in methylcellulose base (one million units every four hours 3/4), and termination of other antibiotic therapy (1/4). These measures were effective in clearing the infection in only one patient. A second patient required prolonged massive oral Nystatin therapy, while two patients required intravenous Amphotericin B to control their infections. Two patients subsequently developed strictures which necessitated multiple esophageal dilatations. One of these patients developed endocarditis during home dilatation therapy. All patients are currently free of disease.

Thus, *Candida* esophagitis can and does occur after short term antibiotic therapy in patients without immunosuppression. Because of the high morbidity in patients treated according to current recommendations, early diagnosis and aggressive treatment is essential. Due to the frequent occurrence of this post-operative symptomatology, a high index of suspicion is required to make the diagnosis.

\*By invitation

### 18. Extended Cervical Esophagomyotomy for Cricopharyngeal Dysfunction

MARK B. ORRINGER, *Ann Arbor, Michigan*

Forty adult patients have undergone a 7-10 cm cervical esophagomyotomy (from the upper edge of the left thyroid cartilage to behind the clavicle) for cricopharyngeal dysfunction. A Zenker's diverticulum was present in 12 patients (30%), and in 5 was recurrent. Preoperative symptoms included cervical dysphagia (95%), expectoration of saliva (40%), and intermittent hoarseness (30%). Four patients were being fed through feeding tubes because of total inability to swallow. "Heartburn" was experienced by one-half of the patients, but only 12 had acid or food regurgitation. The duration of symptoms ranged from 1 month to 11 years (average 3.9 years). Weight loss had occurred in 15 patients (38%) and ranged from 5.5-40.9 kg (average 16 kg). Barium swallows were "normal" in 10 patients. Abnormal findings included a Zenker's diverticulum (12), prominent cricopharyngeal sphincter (11), nasopharyngeal reflux and/or incoordinated initiation of deglutition (7), a sliding hiatal hernia (11) and abnormal esophageal motility (7). Esophageal manometry revealed abnormalities of upper esophageal sphincter function in only 16 patients. Of 36 patients undergoing standard acid reflux testing, one-third had moderate-to-severe gastroesophageal reflux. Seven patients underwent staple-resection of a Zenker's diverticulum at the time of cervical esophagomyotomy. Postoperative complications



included transient vocal cord paresis (4), vocal cord paralysis (1), and salivary fistula (1). There were no postoperative deaths. After 2-48 months (average 16 months) of follow-up, 33 patients (83%) have had a good to excellent result, and 7 (17%) have not been benefitted by operation.

Conclusions: The diagnosis of cricopharyngeal dysfunction must frequently be made on clinical grounds in a patient complaining of cervical dysphagia or a "lump in the throat", expectoration of saliva, and intermittent hoarseness with or without weight loss. There may be no objective radiographic, manometric, or endoscopic abnormalities. An extended cervical esophagomyotomy relieves symptoms in a substantial number of these individuals, ensures that all incoordinated pharyngoesophageal muscle is divided, and is a relatively low-risk procedure for which the gain may be great in appropriately selected patients.

\*By invitation

### **19. The Intercostal Pedicle Method for the Control of Gastroesophageal Reflux in the Presence of Esophagogastrostomy: 12-Year Clinical Results**

*N. J. DEMOS and R. M. BIBLE\*, Short Hills, New Jersey*

The experimental study of a new technique used for the prevention and treatment of reflux esophagitis was presented in this Society in 1967. During the last 12 years the intercostal pedicle has been used in 34 resected cancer patients, in 6 bypassed cancer patients, and in 3 patients with esophageal resection for benign disease.

The cancer patients were followed up to 6 years and the benign ones up to 12 years. The operative mortality was 1/34 in the resected cancer patients, 1/6 of the bypassed ones, and 0 in the benign ones. History, histology, fluoroscopy, esophagram, fiberoptic esophagoscopy, and motility and pH studies have been used to study the function of the new sphincter upon the esophagogastrostomy.

There has been uniform absence of symptomatic or observed reflux or esophagitis. The x-ray appearance is that of a sling-like antireflux mechanism. The immediate postoperative LES pressure has been up to 26 mm/HG settling to a 12-15 mm/HG in the long term follow-up. There is a sharp rise in pH from 1-2 to 6 at the LES. Competency has also been observed in the only 2 patients with postoperative leaks which closed spontaneously. Only 1 patient required occasional dilatation but has a competent sphincter.

The procedure is advocated for the patients with absent cardia. Its advantages are (1) ease of performance at any level in the chest, (2) easily and bilaterally available pedicles, (3) a living functional sphincter whose tightness can be adjusted at will, (4) interposition of intestinal segment is avoided, (5) only 15-20 minutes of operative time are required and (6) protection of the esophagogastrostomy against leak.

\*By invitation

### **20. The Contribution of Endoscopy to the Study of the Pathogenesis of Esophageal Adenocarcinoma**

*M. SAVARY\* and A. P. NAEF\*, Lausanne, Switzerland  
Sponsored by: F. G. Pearson, Toronto, Ontario, Canada*

The gastric mucosa of the cardia, the esophageal glands, and the presence of ectopic columnar mucosa have all been suspected to be at the origin of adenocarcinoma of the esophagus.

The endoscopic study of 387 cases of complicated peptic esophagitis (stage IV), carried out with serial endoscopic pictures and biopsies, showed that in 221 cases (60%) columnar epithelial clusters were also present.

Repeated endoscopic controls have confirmed the acquired character and progressive nature of this columnar epithelisation of the lower esophagus: this process represents therefore a metaplasia scarring of peptic ulcerations.

Metaplasia columnar epithelial linings usually stabilize after surgical cure of the gastro-esophageal reflux. Some endoscopic controls performed *more than ten years* after a Nissen-operation, have not shown any new changes in their pattern.

We have observed the association of adenocarcinoma of the esophagus in the presence of columnar epithelial metaplasia in 18 patients, all belonging to the groupe of 117 cases where the esophageal columnar metaplasia was diffuse and in continuity with the gastric mucosa (i.e. columnar epithelial lined lower esophagus). This represents a frequency of 15%.

Twice, repeated esophagoscopies have allowed us to witness the appearance of an adenocarcinoma at the level of a cluster of columnar metaplasia epithelium.

In conclusion, the frequency of the association of an adenocarcinoma with complicated peptic-esophagitis, increasingly mentioned in the literature, could be explained by a specific scarring process of chronic peptic ulcerations with re-epithelisation by metaplasia gastric columnar epithelium, instead of the usual squamous type.

#### **INTERMISSION - 45 MINUTES VISIT EXHIBITS**

\*By invitation

### **21. Pectus Excavatum: Late Results without Surgery and Following Operation**

*GEORGE H. HUMPHREYS II and ALFRED JARETZKI III,*

*New York, New York*

Decision on operations to correct Pectus Excavatum depend on knowledge of late results. Few surgical reports answer these questions:

1. What can be expected without surgical correction?
2. What are the late results of surgical procedures?
3. If operation is justified, what are the indications?

Records of all patients on whom the diagnosis was made in one medical center during thirty years were reviewed. Of 334 patients, 168 had no attempted correction; 174 operations were done on 166.

*A. In those not operated:*

1. Mortality, especially in infancy, was high.
2. Of the 52% with known late results, the deformity disappeared or improved in 36% up to age 6, after which it remained the same or worsened with age.
3. Mild deformities were compatible with long life and few or no symptoms.
4. Severe deformities were associated with chronic disabling symptoms.

*B. In those operated:*

1. There was no surgical mortality. The result meant little until more than five years after operation.
3. Late results were determined on 106 operations performed on 102 of the 122 patients done more than five years ago (84%).
4. Late results were satisfactory in 68 of the 102 patients (67%).
5. Late results tended to become worse with patient's age at follow-up.
6. The method of correction was more clearly related to the late result than sex, severity, age at operation or time after operation.

CONCLUSIONS

1. Surgical correction of Pectus Excavatum is justified in suitable cases.
2. Radical operations yielded better ultimate results than simpler ones.
3. The final result cannot be judged until the patient is fully grown.
4. Better objective methods of evaluation of all patients over a period of 15 to 20 years is necessary in order to judge the value of any corrective operation.

**22. Thymectomy for Myasthenia Gravis in the Young Adult -Long Term Results**

*RICHARD E. CLARK, JACK B. SHUMATE\*,*

*MICHAEL H. BROOKE\*, JOHN P. MARBARGER\*,*

*PHILIP N. WEST\*, CHARLES L. ROPER,*

*THOMAS B. FERGUSON, and CLARENCE S. WELDON,*

*St. Louis, Missouri*

This 7 year study involved 28 young adults ages 14-35 who had thymectomy as primary therapy for myasthenia gravis (MG) *prior* to failure of anticholinesterase and/or steroid medication. The diagnosis was established on both clinical and electromyograph bases. Most had preoperative muscle biopsies and more recently tomographic scans of the chest. Extensive objective testing of muscle strength and pulmonary function was performed pre and post operatively. All had a median sternotomy and wide dissection of the thymus. Special care was taken to obtain all thymic tissue and lymph nodes in the two tails of the gland into the neck. 93% received prostigmin in the immediate post operative period and 96% were then given mestinon. Prednisone 100 mg q.d. was given to all after the 7th post operative day and the mestinon discontinued over several months. Follow-up intervals range from 7 to 0.5 years (mean = 2.1 year). There have been no operative, perioperative or long term deaths. Careful follow-up by the Muscle Study Group with objective testing has shown 62% have excellent results without use of anticholinesterase drugs and minimal dosage of prednisone. 11 % have had a good response and 23% are improved but require mestinon. To date, no patient who has had an excellent or good response to thymectomy has an exacerbation of symptoms of MG. Only one patient who had congenital MG (4%) failed to respond to thymectomy. One patient in this series had a malignant thymoma. It is concluded that thymectomy early in the course of myasthenia gravis is efficacious and appears to have a lasting effect.

\*By invitation

### 23. Trends in Cardiac Surgery: A 5-Year Study of a Defined Population

ROBERT H. KENNEDY\*, MARGARET A. KENNEDY\*,

JAMES R. PLUTH and FRED T. NOBREGA\*,

*Rochester, Minnesota*

To provide population based information on cardiac surgery that would be useful in developing appropriate planning guidelines, the total number and types of cardiac operations performed on residents of Olmsted County, Minnesota, from 1973 through 1977 were studied through use of the medical records linkage system at the Mayo Clinic. During this time, 213 patients underwent 224 operations. A total of 104 patients (87 males and 17 females) had only coronary artery bypass operations, 94 (44 males and 50 females) had only other cardiac surgical procedures, and 15 (10 males and 5 females) had both. The overall number of cardiac operations per 10,000 population of all ages increased from 4.2 in 1973 to 6.2 in 1977. Non-coronary artery bypass operations showed no significant trends over time and the average rate was 2.7 per year. The number of coronary artery bypass operations per 10,000 population 25 years of age or older increased from 2.7 in 1973 to 7.0 in 1977 ( $P < 0.01$ ). Age-specific rates for patients who had coronary artery bypass operations were greatest in the 45 through 64 year age group. For those who had other cardiac operations the rates showed a peak in the less than 5 year age group, but were greatest among patients 65 years of age or older. The average annual number of open heart operations per 10,000 population less than 15 years of age was 2.1 and the 1977 rate for patients 15 years of age or older was 7.4. On the basis of these rates adjusted by age and sex to the 1970 United States white population, a population of approximately 350,000 less than 15 years of age would be required to assure use of a pediatric cardiovascular surgical facility at the minimum level of 75 cases requiring open heart operations per year and a population of approximately 225,000 greater than 14 years of age would provide 200 adult open heart cases per year. Thus, total populations of approximately 1,275,000 and 310,000 of all ages would be required to meet these minimum standards for pediatric and adult open heart operations.

**11:30 A.M.**

**Address of Honored Speaker**

**CARDIO-THORACIC METAMORPHOSIS**

*H. D'Arcy Sutherland*

*Melbourne, Australia*

\*By invitation

## TUESDAY AFTERNOON, APRIL 29, 1980

2:00 P.M. Scientific Sessions  
Continental Ballroom

### 24. Extending the Limits of Hemodilution on Cardiopulmonary Bypass Using Stroma-Free Hemoglobin Solution

WILLIAM Y. MOORES\*, FRANK DEVENUTO\*,  
WILLIAM H. HEYDORN\*, RICHARD B. WEISKOPF\*,  
MARK BA YSINGER and JOE R. UTLEY, San Diego,  
and San Francisco, California

Recent increased interest in blood conservation and tissue perfusion during cardiopulmonary bypass has heightened the importance of achieving an optimal level of hemodilution with the lowest hematocrit that adequately meets tissue oxygen requirements. To help meet this objective we utilized 10 swine to evaluate the ability of stroma-free hemoglobin solution (SFHS) to support myocardial performance at a hematocrit level of 5%. All animals were placed on normothermic, total and right-heart bypass (RHBP, controlled rate, pre-load and after-load) for evaluation of stroke volume (SV, ml/beat), coronary blood flow (CBF, ml/min/100g tissue), arterial-coronary sinus oxygen difference (A-CSO<sub>2</sub>A, Vols %) myocardial oxygen consumption (MVO<sub>2</sub>, ml/min/100g tissue), lactate extraction (LE, %) and mass spectrometrically measured myocardial tissue gases during a control period at a hematocrit level of 28%. We divided the 10 animals into 2 equal groups who underwent exchange transfusion to a hematocrit of 5% using either 1% SFHS (Gp I) or 1% bovine albumin solution (Gp II). All tests were repeated during these experimental conditions.

*Results (mean ± S.E.):* Myocardial performance following albumin solution exchange (Gp II) could not be sustained on RHBP and these animals had a SV of 0 at an LVEDP of 14 torr. SFHS animals (Gp I) had a significant drop in SV at 14 torr following exchange ( $20 \pm 3$  vs.  $10 \pm 4$ ,  $P < 0.025$ ), but this 50% performance level could be sustained. CBF rose and MVO<sub>2</sub> fell in both groups, although the statistically non-significant mean differences were less with SFHS. A-CSO<sub>2</sub>A fell significantly ( $P < 0.05$ ) with albumin solution ( $7.3 \pm 1.4$  vs.  $2.2 \pm 0.2$ ) and non-significantly with SFHS ( $5.6 \pm 0.7$  vs.  $4.1 \pm 1.4$ ). Lactate production occurred in both groups, but was greater with albumin ( $34 \pm 11\%$ ) than with SFHS ( $3 \pm 32\%$ ). No changes in myocardial tissue gases were noted in either group.

*Conclusion:* Although myocardial performance decreased and some lactate production occurred with SFHS, we believe these comparative results provide promise in the eventual utilization of an oxygen carrying agent such as SFHS to extend the limits of hemodilution to a hematocrit of 5% or less.

\*By invitation

### 25. Massive Air Embolism during Cardiopulmonary Bypass: Causes, Prevention and Management

NOEL L. MILLS and JOHN L. OCHSNER, New Orleans, La.

Massive air embolism during Cardiopulmonary bypass is a frightening complication requiring immediate response and carrying strong medico-legal implications. From July 1971 to July 1979 there were eight instances of massive air embolism during 3620 Cardiopulmonary bypass

operations. Five such accidents from other institutions are included in this report. Causes were (1) inattention to reservoir level, (2) reversal of pump head tubing or direction of pump head rotation, (3) unexpected resumption of heartbeat, (4) inadequate steps to remove air after cardiomy, (5) high flow suction deep in a pulmonary artery, (6) defective oxygenator, (7) use of a pressurized cardiomy reservoir, and (8) inadvertent detachment of oxygenator during bypass. Prevention includes a systematic check of pump suckers and perfusion lines before bypass, a sensing device on the oxygenator reservoir, secure fixation of the oxygenator and avoidance of traffic around pump equipment, immediate cessation of pump and inspection for abnormal noise, use of standard maneuvers to remove air from the heart and carotid compression with resumption of heartbeat.

*Immediate* management of massive air embolism consists of deep Trendelenburg position with a large stab wound in the ascending aorta for retrograde drainage from the cerebrovascular bed. Temporary retrograde perfusion through the superior vena cava may also be used. Subsequent steps are hypothermia with the resumption of Cardiopulmonary bypass, elevation of blood pressure, steroids, ventilation with 100% oxygen, and deep barbiturate anesthesia.

Of the 13 patients, there were 4 instantaneous deaths. Cerebral injury which resolved within a 2-month period occurred in 3 patients. The remainder had no neurologic sequelae. Nonfatal cerebral air injury may be associated with prolonged convalescence yet complete recovery, as compared to embolism from debris, clot, etc., which offers a poorer prognosis.

\*By invitation

## **26. Perioperative Myocardial Infarction and Shock: Successful Management with an LVAD**

*WILLIAM F. BERNHARD, SHUKRI F. KHURI\*,*

*ROBERT L. BERGER, ERNEST M. BARSAMIAN\*,*

*MICHAEL C. FISHBEIN\*, and JAMES G. CARR\*,*

*Boston, Massachusetts*

Postoperative circulatory support (CS) with a pneumatic, xenograft-valved left ventricular assist device (LVAD) was employed in 17 cardiopulmonary bypass (CPB) dependent patients unresponsive to catecholamines and an intra-aortic balloon pump. The device (stroke volume 75 ml) was interposed between the left ventricular apex and ascending aorta, and provided a mean (pulsatile) flow of  $2.3 \pm 0.2$  L/min/M<sup>2</sup> for periods of 2-180 hours (without anticoagulation). Cardiogenic shock secondary to a myocardial infarction was identified histologically in 10/17 patients (biopsy or autopsy) who survived for 72 hours or longer. Evidence of improvement in ventricular function was documented in seven of these after 48 hours, employing serial determinations of cardiac output (thermal dilution) or radionuclide (<sup>99m</sup>Tc) gated blood pool scans. Six patients recovered sufficiently to undergo pump removal, and three remain well 11, 17 and 18 months post-resuscitation. The other three expired prior to hospital discharge 5-30 days postoperatively.

Deaths were secondary to a hemorrhagic diathesis (HD) in 9/17 patients, renal failure (2), sepsis (2), and congestive failure (1). The HD (diffuse bleeding from all suture lines) was the result of prolonged CPB (mean  $5.6 \pm 0.4$  hours) utilized to complete the operation and attempt cardiac resuscitation. It was associated with erythrocyte trauma including elevated plasma hemoglobin concentrations and erythrocyte mechanical fragility values (60-200 mgm%). However, if renal function was not impaired, these values rapidly decreased to normal (12 hours). Thrombocytopenia ( $25-40 \times 10^3$ mm<sup>3</sup>) and abnormal platelet function were also evident and persisted until device

removal. The functional derangement was characterized by reduced platelet dense body content (consistent with lysis or release of granules), and marked impairment of  $^{14}\text{C}$ -serotonin release at thrombin-to-platelet concentrations of 0.013-0.02  $\mu\text{g}/10^8$  platelets. *Conclusion:* Effective management of severe left ventricular dysfunction could be accomplished with an LVAD, but earlier application of the device, to reduce CPB time, is essential if the associated HD is to be avoided.

\*By invitation

## 27. A New Approach to the Treatment of Aortic Dissection

*ALAIN CARPENTIER\*, ALAIN DELOCHE\*,*

*JEAN-NOEL FABIANI\*, SYLVAIN CHAUVAUD\*,*

*REMI NOTTIN\*, JOHN RELLAND\* and CHARLES DUBOST,*

*Paris, France*

In spite of continued progress in surgical technique and myocardial protection, aortic dissection remains associated with a high incidence of early and late mortality. Actuarial survival at 8 years varied between 33 to 50% depending of the type of dissection as reported by the Stanford Group at the last A.A.T.S. Meeting. This poor prognosis may be at least partially explained by 2 factors : 1) The Dacron graft anastomoses involve the dissected areas which may lead to excessive bleeding or disruption, 2) A large segment of the dissected aorta is left in place which may lead to recurrent dissection, rupture or ischemia.

The following approach has been developed to overcome these drawbacks: 1) The Dacron graft anastomoses are performed on the non-dissected zones of the aorta, 2) The dissected aorta is totally and progressively excluded from the systemic circulation by flow inversion and subsequent thrombosis.

In type III dissection involving the descending aorta, the operation is performed through a median sternotomy, which may be prolonged by a median laparotomy, without extracorporeal circulation. A Dacron graft is placed between the non dissected ascending aorta to a non dissected zone of the diaphragmatic or abdominal aorta. A permanent clamp is placed on the aorta distal to the left subclavian artery effectively reversing the flow in the descending aorta and the dissected areas which are progressively excluded from the circulation by an intravascular thrombosis. In type I dissections, involving the entire thoracic aorta, the Dacron graft is first anastomosed to the distal aorta. Revascularization of the cerebral and brachial arteries is then achieved without extracorporeal circulation by a bifurcated graft laterally implanted on the main Dacron graft. Finally, the proximal end of the Dacron graft is anastomosed to the aortic annulus contouring the coronary ostia using an extracorporeal circulation.

Experimentation in animal have been carried out to study the conditions and extent of the thrombosis after flow reversal in the descending aorta.

4 patients were operated on for either type III (2 cases) or type I dissection (2 cases). Post-operative investigations and clinical follow-up up to 16 months support the concept of flow reversal and thromboexclusion in the surgical treatment of type I and III aortic dissection.

\*By invitation

**28. Reduction of Operative Heat Loss and Pulmonary Secretions in Neonates Using Heated and Humidified Anesthetic Gases**

*ERIC W. FONKALSRUD, SELMA CALMES\* and*

*LARRY BARCLIFF\*, Los Angeles, California*

Humidified ventilation reduces the incidence of atelectasis and the difficulty removing tracheobronchial secretions in nonanesthetized infants requiring respiratory support; however, this technique has not been widely used for neonates receiving general anesthesia (GA). Hypothermia with consequent increased oxygen utilization and acidosis is a common sequella following GA in neonates despite the use of various external heating techniques.

During the past 3 years, 42 neonates undergoing surgical repair of major congenital malformations, including 28 thoracic operations, (18 full term, 24 premies) received heated (37°C) and humidified (100% saturated) anesthetic ventilation (HHV) for 1 to 3 hours with a mixture of O<sub>2</sub>, room air, N<sub>2</sub>O, and halothane via a Baby Bird Respirator with minimal external heating. During the past 4 years, a comparable group of 38 neonates with major anomalies underwent surgical repair under general anesthesia using the same anesthetic mixtures but with dry gases at room temperature (22 to 24°C) and with a heating mattress and an overhead infrared heating lamp (SV). The mean rectal T of infants given HHV decreased 0.38°C during the operation, whereas the mean T decreased 1.15°C in the SV infants. Twenty-three of the infants with HHV, but only 2 of the SV babies experienced heat gain by the end of the operation (p<0.05). Blood gas determinations during and shortly after operation were better in the HV infants compared to the SV babies. The mean period of post anesthetic intubation with ventilatory support was shorter in the HHV infants. Postoperative atelectasis or pneumonia occurred in only 9 of the SV group and in 2 of the HHV infants. Pulmonary secretions in the HHV infants were less voluminous and tenacious than in SV infants.

Since previous studies have shown that HHV does not interfere with anesthetic gas uptake it appears that this technique may be a useful adjunct to the currently used methods of neonatal anesthesia,

**INTERMISSION - 45 MINUTES**

**VISIT EXHIBITS**

\*By invitation

**TUESDAY - APRIL 29, 1980**

**4:00 P.M. Executive Session (Limited to Active and Senior Members) - Continental Ballroom**

**6:30 P.M. President's Reception - Continental Ballroom**



## WEDNESDAY MORNING, APRIL 30, 1980

8:30 A.M. Scientific Session  
Continental Ballroom

### 29. Clinical Durability of the (Hancock) Porcine Xenograft Valve

*PHILIP E. OVER\**, *D. CRAIG MILLER\**,  
*EDWARD B. STINSON\**, *BRUCE A REITZ\**,  
*RICARDO MORENO-CABRAL\**, and  
*NORMAN E. SHUMWAY*, *Stanford, California*

The principal feature which remains to be completely defined in regard to the Hancock xenograft bioprosthesis is long-term durability. As increasing numbers of bioprosthetic valves are implanted, this characteristic assumes greater importance, particularly since some investigators have recently advocated abandonment of xenografts because of impaired function after 5 years (yrs). This report provides extended data regarding valve durability derived from 1407 patients (707 aortic [AYR] and 700 mitral [MVR] replacements) who received Hancock bioprostheses between 1971 and 1979; cumulative duration of follow-up was 1732 patient-years (pt-yrs) for AYR and 1843 pt-yrs for MVR, and maximum followup was 8.4 yrs. One hundred seventy nine pts were followed for more than 5 yrs and 67 for more than 6 yrs. Valve failure was defined on the basis of the following criteria: 1) development of a new regurgitant murmur 2) thrombotic valvular occlusion 3) infective endocarditis resulting in reoperation or death 4) hemodynamic valvular dysfunction confirmed by catheterization and resulting in reoperation or death. Using these criteria 15 valve failures occurred in the AVR cohort and 23 in the MVR cohort, yielding linearized occurrence rates of 0.9%/pt-yr and 1.3%/pt-yr., respectively. Actuarial analysis revealed that  $96 \pm 1.2\%$  ( $\pm$  SEM) of AVR pts after 5 yrs and  $90 \pm 2.6\%$  of MVR pts after 6 yrs were free of valve failure. Primary tissue failure (defined by calcification or leaflet disruption resulting in stenosis and/or regurgitation without antecedent endocarditis) or valve thrombosis accounted for 5 of 15 AVR failures and 13 of 23 MVR failures, equivalent to linearized occurrence rates of 0.3%/ pt-yr and 0.7%/ pt-yr, respectively. Calculation by actuarial methods revealed that  $98 \pm 1\%$  of AVR (at 5 yrs) and  $92.5 \pm 2.4\%$  of MVR pts (at 6 yrs) were free of primary tissue failure or valve thrombosis. The actuarially-calculated rate of valve failure up to 5 yrs remains constant as a function of time for AVR pts; for MVR pts there appears to be a slight acceleration in the rate of valve failure after 5 yrs. In this extended experience the incidence of xenograft valve failure 5 to 6 yrs postoperatively remains acceptably low and supports the continued use of the xenograft bioprosthesis for cardiac valve replacement.

\*By invitation

### 30. Risk-Benefit Analysis of Warfarin Therapy in Hancock Mitral Valve Replacement

*J. DONALD HILL, LIZELLEN LA FOLLETTE\**

*ROBERT J. SZARNICKI\*, G. JAMES A VERY, II\*,*

*WILLIAM J. KERTH, FRANK GERBODE,*

*and ROBERT RODVIEN\*, San Francisco, California*

Porcine mitral valve bioprostheses have a low thromboembolic rate. Controversy persists concerning whether warfarin lowers this rate further without undue risk. In mid-1976 we changed from no systemic anticoagulation to routine systemic anticoagulation begun on day 3. The purpose, therefore, of this investigation was to analyze the thromboembolic and/or major bleeding complications of 124 consecutive but nonrandomized patients who had only Hancock mitral valve replacements between 9/74 and 6/79 treated with and without anticoagulants. Four basic study groups were created: Group 1, warfarin; Group 2, aspirin; Group 3, no anticoagulants; Group 4, warfarin and aspirin. Group 5 combines Groups 1 and 4 (warfarin and warfarin and aspirin) and Group 6 combines Groups 2 and 3 (aspirin and no anticoagulants).

<i>Group</i>	<i>1</i>	<i>2</i>	<i>3</i>	<i>4</i>	<i>5</i>	<i>6</i>
<i>Therapy</i>	<i>Warfarin</i>	<i>Aspirin</i>	<i>No anti-coagulants</i>	<i>Warfarin+ Aspirin</i>	<i>Combined 1 + 4</i>	<i>Combined 2 + 3</i>
No. of Patients	72	21	26	5	77	47
Mean follow-up (yr)	2.2	4.2	3.7	2.0	2.2	3.9
Patient years	158.2	87.8	96.7	9.9	168.1	184.4
Emboli	4	2	3	0	4	5
Emboli per 100 patient years	2.5	2.3	3.1	0.0	2.4	2.7
Major bleeding complications	9	1	0.0	0.0	9	1
Major bleeding per 100 patient yrs	5.7	1.1	0.0	0.0	5.4	0.5

The embolic rate was not significantly different in any group ( $P = NS$ ). There were no deaths from emboli in any group. Group 1 (warfarin) resulted in significant major bleeding complications ( $P < 0.05$ ), including one death. Bleeding was also increased in Group 5 (warfarin, and warfarin and aspirin) ( $P < 0.01$ ).

Conclusions: There is no difference in the thromboembolic rate with or without warfarin in patients with Hancock mitral valve prostheses. 2. Warfarin significantly increases major bleeding complications in this clinical setting.

\*By invitation

### 31. Year of Operation as a Risk Factor in the Late Results of Valve Replacement

*ALBERT STARR, GARY L. GRUNKEMEIER\*,  
JOSEPH F. TEPLY\* and QUENTIN MacMANUS\*,*

*Portland, Oregon*

The actuarial thromboembolic rates of aortic and mitral noncloth-covered caged-ball valves used during the second decade of cardiac valve replacement are significantly lower than for the same prostheses implanted during the first decade, as shown in the following table:

Implant years	<i>Mitral 6120</i>		<i>Aortic 1200/60</i>	
	1965-72	1973-79	1965-72	1973-79
Number of patients	83	67	133	216
Maximum/mean follow-up (years)	14/8.4	5/1.8	13/8.8	6/1.9
Five year embolus-free rate (± SE)	69(± 6)%	96(± 4)%	78(± 4)%	93(± 2)%
P-value	p=.017		p=.004	

Five year embolus-free rates for the composite-strut caged-ball, Bjork-Shiley tilting disc and porcine xenograft valves all fall in the range of from 88% to 91% for the mitral position and from 91% to 94% for the aortic. Thus the standard noncloth-covered prosthesis, used during the current era, has a thromboembolic risk as low as that reported with other concurrently utilized valve substitutes. This striking reduction in thrombogenicity demonstrates that the time frame of implantation must be considered when evaluating the results of cardiac valve replacement.

\*By invitation

### 32. Open Mitral Commissurotomy-A Modern Evaluation

*WILLIAM L. HALSETH\*, DONALD P. ELLIOTT\*,  
E. LANCE WALKER\* and ELEANOR A. SMITH\*,*

*Denver, Colorado*

*Sponsored by: Ben Eiseman, Denver, Colorado*

Familiarity with replacement of the mitral valve (MVR) with prosthetic and tissue valves has dimmed awareness of the usefulness of open mitral commissurotomy (OMC). This is a review of a 10-year experience ending in December 1978 of 259 consecutive patients operated upon with a clinical diagnosis of mitral stenosis. MVR was necessary in 62 patients (24%), primarily because of severe deformity of valvular and subvalvular structures. No closed commissurotomies were performed-an operation now considered passe.

Of the 197 OMC, 12 had additional cardiac procedures. Of the 3 patients who died (1.52%), two were operated upon as emergencies because of rapidly progressive cardiac failure.

Followup was obtained on 191/197 (91%) of the OMC patients. Late mortality was 9% (18 patients) of whom 14 were cardiac related. 76% of patients (146) had at least one category New York Heart Association Class improvement following OMC. Fourteen (7%) of the 191 OMC patients had subsequent MVR at times varying from 2-92 months (mean 41.6 months). Ten year survival for the 191 OMC patients was 81%.

#### Summary and Conclusions:

(1) Open mitral commissurotomy (OMC) was performed in 197 of 259 patients with the clinical diagnosis of mitral stenosis. Of these, 81% survived 10 years, and 14 (7%) subsequently required valve replacement. (2) OMC is the operation of first choice for patients with the clinical diagnosis of mitral stenosis. The main indication for proceeding to MVR at the first operation is severe valve deformity, which prevents successful commissurotomy.

(3) There will be no need for valve replacement in 93% of patients following OMC, 76% of whom will experience measurable and lasting functional benefit from open commissurotomy alone.

(4) This clinical experience emphasizes that open commissurotomy rather than valve replacement is the best initial treatment for most patients with mitral stenosis.

\*By invitation

### 33. Is Tricuspid Valve Repair Necessary?

*CARLOS M. G. DURAN, JOSEL. POMAR\**,

*THIERRY COLMAN\*, ALVARO FIGUEROA\**

#### **JOSE M. REVUELTA\*, and JOSE L. UBAGO\*, Santander, Spain**

In an attempt to clarify the still confusing indications for tricuspid surgery, a group of 150 patients (pts) has been studied. Selection was made according to the following criteria 1) All had preoperative significant tricuspid disease and 2) pre and postoperative right and left catheterization including biventriculography was available. It has been previously proven in our laboratory, by correlating the hemodynamic and surgical findings, that our technique of right ventriculography is highly reliable in the diagnosis of tricuspid insufficiency (TI) and whether it is an organic or functional lesion. 123 (82%) pts were recatheterized as part of a routine follow up study and 27 (18%) for clinical deterioration. 89 had an associated mitral and 61 mitral and aortic disease. After left side repair, 119 pts underwent tricuspid surgery (group A). In 31 pts the tricuspid valve was not repaired (group B).

In group A, 64 (54%) had one or more tricuspid commissures ("organic") and the remainder 55 were classified as "functional". Tricuspid valve repair includes 73 ring annuloplasties, 42 commissurotomies and annulo-plasties and 4 commissurotomies. Postoperative gradients averaging 2.8 mm Hg ( $\pm$  1.6) were found in 31% of the pts with functional and in 44% of those with organic TI. Those pts with tricuspid gradients averaged a cardiac index (CI) of 3.2 L/min/m<sup>2</sup> and those without gradients 2.8 (p<0.01). Significant residual TI was found in 10 functional and 26 organic valves. Clinically, 85% were in Functional Class I

despite their small residual gradients and TI. Further analysis of the hemodynamic data of this group of pts showed that the postoperative CI was directly related to the adequacy of the left side repair and to the residual pulmonary resistances independently of whether residual TI was present or not. These facts seem to imply that the associated tricuspid repair is at best unnecessary. However, when measuring right ventricular volumes it was observed a postoperative increase of 9.3% in those pts with persistent TI and a 37% decrease in those with tricuspid competence.

In group B, all 14 pts with organic disease showed residual TI. 8 pts out of the 17 with functional TI had no regurgitation concomitant with a significant decrease in pulmonary resistances. In the remainder 9 pts the TI persisted.

Based on these data that emphasize the need for a correct left side repair, our present attitude towards tricuspid repair is: 1) Functional TI can only be ignored in pts with predictable significant reduction in pulmonary resistances, 2) Organic disease must be repaired.

### **INTERMISSION - 45 MINUTES**

#### **VISIT EXHIBITS**

\*By invitation

### **34. Sternal Wound Complication Management And Results**

*CYRUS SERRY\*, PHYLLIS C. BLECK\*, HUSHANG JAVID,*

*JAMES A. HUNTER, MARSHALL D. GOLDIN\*,*

*GIACOMO A. DELARIA\*, and HASSAN NAJAFI,*

*Chicago, Illinois*

Records of 4,125 patients who underwent sternotomy for cardiac surgery were reviewed to determine the incidence of sternal wound complications (1.8%), including wound drainage, skin separation, unstable sternum, and sternal dehiscence with or without infection. Septicemia and mediastinal abscesses were found in all 19 patients who expired.

Based on findings, local treatment (incision and drainage of skin and subcutaneous tissue with frequent change of dressing or irrigation (Method A) is recommended for (1) serosanguineous drainage, (2) patients with stable sternums and superficial infection, but no systemic reaction. However, surgical debridement and closure followed by mediastinal irrigation via drainage tubes with 0.5% povidone iodine solution (Method B) is recommended for (1) draining, unstable sternums, (2) wound infections involving the retrosternal space and (3) wound infections causing systemic reactions unresponsive to Method A.

It was found that none of the eight patients in the latter group died when managed by Method B, and only one developed recurrent infection. In contrast, from 28 patients of the latter group not treated with Method B, 11 died of infection related causes and 13 returned with recurrent infection.

### **35. Fulminating Non-Cardiogenic Pulmonary Edema-A Newly Recognized Hazard During Cardiac Operations**

*ALFRED T. CULLIFORD,\* STEPHEN THOMAS\* and  
FRANK C. SPENCER, New York, New York*

In the past 24 months, four patients have been successfully treated for fulminating pulmonary edema following coronary bypass operations. In each patient, the fulminating edema began within 30-60 minutes after infusion of blood products, usually fresh-frozen plasma, suggesting an allergic mechanism. Copious frothy fluid poured from the endotracheal tube, with hypoxia and respiratory acidosis. Left atrial pressure was only 5.0-10 mm., excluding left ventricular failure; and the electrocardiogram and CPK isoenzymes excluded myocardial infarction. The albumin content of the fluid was significantly elevated, suggesting an exudate rather than a transudate.

Three postoperative patients were promptly reoperated upon, and in one of these, the sternum could not be closed for 72 hours because stiff, distended lungs compressed the heart. Treatment consisted of intravenous prednisolone 2.0-3.0 gms., positive pressure ventilation, diuretics and albumin (in the last two patients).

It was dramatic that within 72 hours this near-lethal condition subsided completely, with no further cardiac or pulmonary complications.

The allergic mechanism is still unclear, but the effectiveness of immediate treatment, as described, prompts this early report. Undoubtedly, patients have died in past years with the erroneous diagnosis of fulminating cardiac failure or "pump lung." This rare condition is readily recognized when massive pulmonary edema develops without signs of left ventricular failure-an occurrence which is easily discerned in a properly monitored patient.

\*By invitation

### **36. Immediate Coronary Artery Bypass for Acute Evolving Myocardial Infarction (AEMI)**

*RALPH BERG, JR., SAMUEL L. SELINGER\*,*

**RONALD P. GRUNWALD\* and**  
*WILLIAM P. O'GRADY\*, Spokane, Washington*

212 consecutive patients (March 1971-April 1979) all had infarction syndrome, electrocardiographic, coronary angiographic, ventriculographic and retrospective enzyme changes consistent with AEMI. In 212 AEMI patients, mean age 55.8 years (R = 28-79), onset of CAB surgery was less than six hours, yielding three surgical deaths for a 1.7% mortality. Conversely, medical therapy in 200 consecutive AEMI patients, mean age 53.2 years (R = 40-65) resulted in 23 deaths giving in-hospital mortality (IHM) of 11.5% plus first year mortality of 14%. Pre and post-operative enzyme and EKG values, age, sex and coronary artery involvement are presented. Angiograms done an average of 7.9 months post-op of 97 grafts to the infarcting areas revealed 95 patent (92%). Ejection fractions were normal, unchanged or improved in 86%. Two small ventricular aneurysms were noted. Follow-up of 200 patients

revealed three deaths within the first year (1.8%). 26 patients had mild angina. Quality of life was the same or improved in 196. In summary, AEMI patients must have CAB early in the syndrome. Surgical results are far superior to medical therapy: Medical IHM (11.5%) + first year mortality (14%) = 25.5%. Surgical IHM (1.7%) + first year mortality (1.8%) = 3.5%.

\*By invitation

### **37. Multivariate Discriminant Analysis of Clinical and Angiographic Predictor of Coronary Surgery Mortality**

*J. WARD KENNEDY\*, GEORGE KAISER, LLOYD FISHER\*,*

**WILLIAM O. MYERS, GERARD MUDD\* and**  
*THOMAS J. RYAN\*, Seattle, Washington; St. Louis, Missouri*

CASS is a large multi-institutional study of the medical and surgical treatment of coronary artery disease. Fifteen cooperating institutes have operated on 7,122 patients from August, 1975 through December, 1978. The in hospital operative mortality (OM) was 2.89%. In an effort to better understand the clinical and angiographic characteristics predictive of OM, we have done a multivariate discriminant analysis of variables associated with OM.

Numerous clinical and angiographic variables were selected from the CASS data file and evaluated in a univariate manner for their relationship to OM. Twenty-two of these variables were then selected for multivariate discriminant analysis. Clinical variables of most value were age, sex, heart size, and symptoms, signs, or treatment for congestive heart failure. Angiographic variables of importance included EF, left ventricular wall motion abnormalities, and the severity of left main coronary disease.

Multivariate discriminant analysis resulted in the selection of eight variables that contained the most predictive information as listed below.

<i>Variable</i>	<i>F Statistic</i>
Congestive Heart Failure Score	73.3
Age	37.5
Left Main Coronary Stenosis % $\geq$ 90%	24.8
Left Ventricular Wall Motion Score	22.9
Female Sex	21.4
Heart Enlargement by Chest X-Ray	17.9
Pulmonary Rales	12.0
Left Ventricular Dilatation by Angiography	9.4

The strong association of OM with advanced age, female sex and variables associated with left ventricular dysfunction is clear. The risk of OM for an individual patient may be estimated with the use of these eight clinical and angiographic characteristics.

\*By invitation

### **38. Doppler Velocity Measurements of Coronary Flow: A Noninvasive Intraoperative Guide to Hemodynamically Significant Lesions**

*CREIGHTON B. WRIGHT, DONALD B. DOTY,  
MELVIN L. MARCUS\*, and CHARLES L. EASTHAM\*,  
Iowa City, Iowa*

Although angiography is in general a reliable guide to the coronary vessels which should be bypassed in individuals with angina pectoris, some lesions escape accurate preoperative assessment. This leaves the surgeon with the dilemma of whether to bypass all visible vessels or to risk not bypassing significant disease.

Utilizing the miniaturized pulsed single crystal Doppler velocity probe and technique developed at the University of Iowa Hospitals and Clinics to measure phasic coronary velocity, the hemodynamic significance of experimental and clinical coronary stenoses can be assessed readily. No dissection of the vessels is required. The probe is held in place over the coronary vessel with a small suction cup. The velocity traces are linearly related to electromagnetic blood flow ( $R = 0.98$ ). Reactive hyperemia curves obtained with 20 second occlusions demonstrate reproducibly the vessels with hemodynamically significant stenoses.

This technique has been used in over 50 patients with a variety of anatomic lesions. In 6 recent patients in which the degree of stenosis of a vessel was disputed or thought to be less than 50%, the use of the Doppler probe and a 20 second reactive hyperemia curve allowed accurate and immediate assessment of the need for bypass.

It is the purpose of this presentation to describe this new unique technique, its benefits, limitations, and overall accuracy.

#### **Adjournment**

**1:00 P.M. Cardiothoracic Residents' Luncheon  
Continental Ballrooms 8 & 9**

\*By invitation

## **WEDNESDAY MORNING, APRIL 30, 1980**

### **A1 Objective Evaluation of the Efficacy of Various Venous Cannulae**

*KIT V. AROM, CHERYL ELLESTAD\*,  
FREDERICK L. GROVER and J. KENTTRINKLE,  
San Antonio, Texas*

Six venous cannulae (USCI #32, #40, #44; Sams #40, Sarns two-stage cavoatrial (CA); and Ferguson Argyle #40) were tested for efficiency of venous flow during cardiopulmonary bypass,



with and without aortic cross-clamping. Each cannula was tested six times in dog models and the data averaged.

The tip of the CA Sarns was positioned in the IVC as recommended. Two #32 USCI caval cannula were placed either with or without caval snaring. The other cannulae (AC) were placed in the right atrium. Arterial flow was constant at 80 ml/kg/min. and pressure averaged 84 mm Hg. The CVP and right atrial pressure (RAP) were recorded. A right ventricular vent (RVV) was placed and the RV excluded from the pulmonary artery. Blood from the right heart which was not picked up by the venous cannula was emptied via the RVV and measured.

	<i>Two H32 USCI</i>		<i>RVV FLOW ml/mm</i>				
	<i>SNARE</i>	<i>NOSNARE</i>	<i>#44 USCI</i>	<i>CA SARNS</i>	<i>#40 SARNS</i>	<i>#40 ARCYLE</i>	<i>H40 USCI</i>
BEATING	190 ± 18**	74 ± 7*	63 ± 6	85 ± 6*	68 ± 7	70 ± 9	62 ± 1
X-CLAMP	16 ± 4	19 ± 5	14 ± 3	18 ± 4	14 ± 2	16 ± 2	14 ± 3

\*p<0.005    \*\*p<0.001

Single AC emptied blood from the right heart (RAP = 0-1) better than two caval cannulae (RAP = 0-2). Caval snaring left more blood in the RA (RAP = 2-5) than any single cannula. The CA Sarns did not empty the RA as well (RAP = 2-4) but drained blood from the IVC (CVP = 0) better than other AC'S (CVP= 1-5). Each of the AC performed almost equally well. Aortic cross-clamping eliminated coronary sinus flow and decreased RVV flow.

Therefore, a single atrial cannula is more efficient in draining blood from the right heart when compared to two caval or a caval-atrial cannula. This advantage is negated by aortic cross-clamping.

\*By invitation

## A2 Special Problems in Management of Tracheostomy in Neonates and Infants

**J. ALEX HALLER, JR., J. J. TEPAS\*, JAMES D. HEROY\***  
*and DENNIS W. SHERMETA\*, Baltimore, Maryland and*

*Portsmouth, Virginia*

Newborn babies and small infants who require tracheostomy often follow a complicated clinical course characterized by frequent sepsis, altered ventilatory dynamics and eventual respiratory decompensation. Many of these problems are avoidable by using a properly placed endotracheal tube during tracheostomy, by using a special surgical technique in the performance of the tracheostomy and by using silastic tracheostomy tubes. Seventy four babies underwent tracheostomy at The Johns Hopkins Hospital and Baltimore City Hospitals between 1963 and 1976. A review of this experience demonstrates the benefits of unhurried, standardized technique and management. In the 48 newborns requiring tracheostomy, no technical difficulties were encountered and no complications occurred as a result of the tracheostomy. In the group of 26 older infants, however, there were significant complications, especially in children undergoing emergency tracheostomy without a previously placed endotracheal tube. Although there were no deaths directly related to tracheostomy, one case of purulent tracheitis and one case of interstitial thyroid hemorrhage were noted at autopsy. Among survivors there was one case of bilateral pneumothoraces, two cases of severe subcutaneous emphysema and three cases of postoperative bleeding. Review of the long term complications in this series demonstrates the benefits of the

silastic polymer tube. Since its routine use, problems with stomal granulation have almost disappeared. There have been no problems in extubating the very young babies. Our operative technique and intensive care management of these babies will be emphasized as the keys to the improved outcome.

\*By invitation

## The American Association for Thoracic Surgery, 1979-1980

*(Listed by Countries, States, Provinces and Cities)*

### Geographical - UNITED STATES

<i>ALABAMA</i>	Covina
Auburn	Carter, Paul R.
Leibovitz, Martin	Davis
Birmingham	Andrews, Neil C.
Karp, Robert B.	Duarte
Kessler, Charles R.	Benfield, John R.
Kirklin, John W.	Escondido
Kouchoukos, Nicholas	Mannix, Edgar P., Jr.
Labrosse, Claude C. L.	Flintridge
Pacífico, Albert D.	Hughes, Richard K.
Montgomery	Fresno
Simmons, Earl M.	Evans, Byron H.
<i>ALASKA</i>	Hemet
Anchorage	Hewlett, Thomas H.
Phillips, Francis J.	Hillsborough
<i>ARIZONA</i>	Ullyot, Daniel J.
Phoenix	Irvine
Brown, Lee B.	Connolly, John E.
Nelson, Arthur R.	Miller, Don R.
Cornell, William P.	Wakabayashi, Akio
McPhail, Jasper L.	La Canada
Melick, Dermont W.	Aronstam, Elmore M.
Sun City	La Jolla
Read, C. Thomas	Fosburg, Richard G.
Tucson	Hutchin, Peter
Burbank, Benjamin	La Mesa

Sanderson, Richard G.	Long, David M., Jr.
<i>ARKANSAS</i>	Pratt, Lawrence A.
Jasper	Laquinta
Hudson, W. A.	Cotton, Bert H.
Little Rock	Loma Linda
Campbell, Gilbert S.	Wareham, Ellsworth E.
Read, Raymond C.	Long Beach
Pine Bluff	Bloomer, William E.
Tillou, Donald J.	Carlson, Herbert A.
<i>CALIFORNIA</i>	Stemmer, Edward A.
Anaheim	Los Angeles
Main, F. Beachley	Baisch, Bruce F.
Beverly Hills	Buckberg, Gerald D.
Carey, Joseph S.	Cracovaner, Arthur J.
Carmel	Davis, Lowell L.
Daniels Albert C.	Fonkalsrud, Eric W.
	Kay, Jerome Harold
	Lindesmith, George G.
Longmire, Wm. P., Jr.	San Francisco
Maloney, James V., Jr.	Culiner, Morris M.
Matloff, Jack M.	Ebert, Paul A.
Meyer, Bert W.	Fishman, Noel H.
Morton, Donald L.	Gardner, Richard E.
Mulder, Donald G.	Gerbode, Frank
Ramsay, Beatty H.	Grimes, Orville F.
Rigler, Leo G.	Hill, J. Donald
Stiles, Quentin R.	Kerth, William J.
Martinez	Leeds, Sanford E.
Treasure, Robert L.	Richards, Victor
Montebello	Roe, Benson B.
Lui, Alfred H. F.	Rogers, W. L.
Oakland	Thomas, Arthur N.
Dugan, David J.	San Jose
Ecker, Roger R.	Angell, William W.

May, Ivan A.	Santa Ana
Orange	Gazzaniga, Alan B.
Bartlett, Robert H.	Santa Barbara
Salyer, John M.	Higginson, John F.
Orinda	Jahnke, Edward J., Jr.
Stephens, H. Brodie	Lewis, F. John
Pacific Palisades	Love, Jack W.
Weinberg, Joseph A.	Stanford
Palm Desert	Mark, James B. D.
Julian, Ormand C.	Shumway, Norman E.
Palm Springs	Thousand Oaks
Goldman, Alfred	Tsuji, Harold K.
Palo Alto	Torrance
Cohn, Roy B.	Moore, Thomas C.
Gonzalez-Lavin, Lorenzo	Nelson, Ronald J.
Jamplis, Robert W.	State, David
Wilson, John L.	Ventura
Pasadena	Dart, Charles H., Jr.
Ingram, Ivan N.	<b>COLORADO</b>
Penido, John R. F.	Denver
Silver, Arthur W.	Brown, Robert K.
Piedmont	Burrington, John D.
Samson, Paul C.	Condon, William B.
South Laguna	Eiseman, Ben
Oatway, William H., Jr.	Grow, John B.
South Pasadena	Hopeman, Alan R.
Brewer, Lyman A., III	Kovarik, Joseph L.
Sacramento	Newman, Melvin M.
Hurley, Edward J.	Pappas, George
Miller, George E., Jr.	Paton, Bruce C.
Smeloff, Edward A.	Pomerantz, Marvin
San Bernadino	Rainer, W. Gerald
Flynn, Pierce J.	Waddell, William R.
San Diego	Lakewood
Baronofsky, Ivan D.	Swan, Henry, II
Chambers, John S., Jr.	Wheat Ridge

Daily, Pat O.

Harper, Frederick R.

Lamberti, John Joseph, Jr.

Peters, Richard M.

Trummer, Max J.

Utley, Joe R.

*CONNECTICUT*

Miami

Hartford

Bolooki, Hooshang

Kemler, R. Leonard

Daughtry, Dewitt C.

New Haven

Gentsch, Thomas O.

Baue, Arthur E.

Jude, James R.

Carter, Max G.

Kaiser, Gerard A.

Geha, Alexander S.

Papper, Emanuel M.

Glenn, William W. L.

Reis, Robert L.

Hammond, Graeme L.

Ripstein, Charles B.

Lindskog, Gustaf E.

Thurer, Richard J.

Stansel, Horace C., Jr.

Miami Beach

Stern, Harold

Greenberg, Jake J.

Wesolow, Adam

Grondin, Pierre

Norwich

North Miami Beach

Kelley, Winfield O.

Spear, Harold C.

Sharon

Naples

Wylie, Robert H.

Linberg, Eugene J.

Wilton

Orlando

Pool, John L.

Sherman, Paul H.

*DELAWARE*

Petersburg

Wilmington

Clerf, Louis H.

Pecora, David V.

Ponte Vedra Beach

*DISTRICT OF COLUMBIA*

Gilbert, Joseph W., Jr.

Washington

Stranahan, Allan

Aaron, Benjamin J.

South Miami

Adkins, Paul C.

Chesney, John G.

Hufnagel, Charles A.

St. Petersburg

Iovine, Vincent M.

Daicoff, George R.

Keshishian, John M.

Wheat, Myron W., Jr.

Peabody, Joseph W., Jr.

Randolph, Judson G.

Simmons, Robert L.

Smyth, Nicholas P. D.

*FLORIDA*

Boca Raton

Seley, Gabriel P.

Coral Gables

Cooke, Francis N.

Delray Beach

Geary, Paul

Ft. Lauderdale

Maurer, Elmer P. R.

Gainesville

Bartley, Thomas D.

Moulder, Peter V.

Jacksonville

Stephenson, Sam E., Jr.

Lakeland

Brown, Ivan W., Jr.

Largo

Lasley, Charles H.

Dematteis, Albert

Tallahassee

Kraeft, Nelson H.

Tampa

Blank, Richard H.

Connar, Richard G.

Seller, Hawley H.

Winter Park

Bloodwell, Robert D.

*GEORGIA*

Atlanta

Hatcher, Charles R., Jr.

Hopkins, William A.

Jones, Ellis L.

King, Richard

Logan, William D., Jr.

Mansour, Kamal A.

Rivkin, Laurence M.

Symbas, Panagiotis N.

Augusta

Ellison, Robert G.

Rubin, Joseph W.

Savannah

Yeh, Thomas J.

**HAWAII**

Honolulu

Ching, Nathaniel P. H.

Gebauer, Paul

McNamara, Joseph J.

Kailua Kona

Fell, Egbert H.

**IDAHO**

Boise

Ashbaugh, David G.

Palo Heights

Demeester, Tom R.

Peoria

Collins, Harold A.

Debord, Robert A.

Skokie

Baffes, Thomas G.

Winnetka

Mackler, S. Allen

**INDIANA**

Herr, Rodney H.	Indianapolis
<b>ILLINOIS</b>	Battersby, James S.
Chicago	King, Harold
Anagnostopoulos, Constantine	King, Robert D.
Barker, Walter L.	Mandelbaum, Isidore
Hanlon, C. Rollins	Shumacker, Harris, Jr.
Head, Louis R.	Siderys, Harry
Hudson, Theodore R.	South Bend
Hunter, James A.	Van Fleit, William E.
Idriss, Farouk S.	<b>IOWA</b>
Javid, Hushang	Cedar Rapids
Jensik, Robert J.	Lawrence, Montague S.
Kittle, C. Frederick	Des Moines
Langston, Hiram T.	Dorner, Ralph A.
Leininger, Bernard J.	Watkins, David H.
Levitsky, Sidney	Iowa City
Michaelis, Lawrence L.	Doty, Donald B.
Midell, Allen I.	Ehrenhaft, Johan L.
Moran, John M.	Rossi, Nicholas P.
Najafi, Hassan	Wright, Creighton B.
Raffensperger, John G.	<b>KANSAS</b>
Replogle, Robert L.	Cunningham
Shields, Thomas W.	Allbritten, F. F., Jr.
Skinner, David B.	Kansas City
Thomas, Paul A., Jr.	Friesen, Stanley R.
Weinberg, Milton, Jr.	Wichita
Evanston	Tocker, Alfred M.
Dorsey, John M.	Winfield
Fry, Willard A.	Snyder, Howard E.
Tatooles, Constantine J.	<b>KENTUCKY</b>
Glencoe Rubenstein, L. H.	Lexington
Glenview	Crutcher, Richard R.
Fox, Robert T.	Dillon, Marcus L., Jr.
Hines	Louisville
Keeley, John L.	Bryant, J. Ray
La Grange	Harter, John S.

Faber, L. Penfield  
Lincolnwood  
Lees, William M.  
Maywood  
Pifarre, Roque  
Oak Brook  
Nigro, Salvatore L.

Baton Rouge  
Beskin, Charles A.  
Metairie  
Ochsner, Alton, Jr.  
New Orleans  
Blalock, John B.  
Decamp, Paul T.  
Glass, Bertram A.  
Hewitt, Robert L.  
Lindsey, Edward S.  
Mills, Noel L.  
Ochsner, Alton  
Ochsner, John L.  
Pearce, Charles W.  
Rosenberg, Dennis M.  
Schramel, Robert J.  
Strug, Lawrence H.  
Webb, Watts R.

**MAINE**

Liberty  
Hurwitz, Alfred  
Portland  
Drake, Emerson H.  
Hiebert, Clement  
Rockport  
Swenson, Orvar

**MARYLAND**

Mahaffey, Daniel E.  
Ransdell, Herbert T., Jr.  
**LOUISIANA**  
Alexandria  
Knoepp, Louis F.

Berger, Robert L.  
Bernhard, William F.  
Bougas, James A.  
Boyd, David P.  
Braunwald, Nina S.  
Buckley, Mortimer J.  
Burke, John F.  
Castaneda, Aldo R.  
Cleveland, Richard J.  
Clowes, George H., Jr.  
Cohn, Lawrence H.  
Collins, John J.  
Daggett, Willard M.  
Deterling, Ralph A., Jr.  
Ellis, F. Henry, Jr.  
Frank, Howard A.  
Gaensler, Edward A.  
Grillo, Hermes C.  
Moncure, Ashby C.  
Neptune, Wilford B.  
Overholt, Richard H.  
Rheinlander, Harold F.  
Russell, Paul S.  
Scannell, J. Gordon  
Schuster, Samuel R.  
Starkey, George W. B.  
Watkins, Elton, Jr.



Baltimore	Weintraub, Ronald
Attar, Safuh M. A.	Wilkins, Earl W., Jr.
Baker, R. Robinson	Brookline
Brantigan, Otto C.	Madoff, Irving M.
Brawley, Robert K.	Cambridge
Cowley, R. Adam	Harken, Dwight E.
Donahoo, James	Concord
Gott, Vincent L.	Soutter, Lamar
Mailer, J. Alex, Jr.	Lawrence
Hankins, John R.	Cook, William A.
Mason, G. Robert	Medford
McLaughlin, Joseph S.	Boyd, Thomas F.
Michelson, Elliott	Desforges, Gerard
Rienhoff, Wm. F., Jr.	Taylor, Warren J.
Turney, Stephen Z.	Methuen
Bethesda	Wilson, Norman J.
Morrow, Andrew G.	Nantucket
Potomac	Mahoney, Earle B.
Zajtchuk, Rostik	Newton Lower Falls
Worton	Strieder, John W.
Walkup, Harry E.	Laforet, Eugene G.
<b>MASSACHUSETTS</b>	South Weymouth
Boston	Malcolm, John A.
Austen, W. Gerald	Springfield
Badger, Theodore L.	Engleman, Richard M.
Barsamian, Ernest M.	Stoughton
	Black, Harrison
<b>MICHIGAN</b>	Pluth, James R.
Ann Arbor	Wallace, Robert B.
Behrendt, Douglas M.	St. Paul
Gago, Otto	Leven, N. Logan
Kirsh, Marvin M.	Lillehei, C. Walton
Morris, Joe D.	Miller, Fletcher A.
Orringer, Mark B.	Perry, John F., Jr.

Sloan, Herbert  
Birmingham  
Magilligan, Donald J., Jr  
Detroit  
Arbulu, Agustin  
Arciniegas, Eduardo  
Day, J. Claude  
Dodrill, Forest Dewey  
Lam, Conrad R.  
Wilson, Robert F.  
Grand Rapids  
Harrison, Robert W.  
Meade, Richard H.  
Rasmussen, Richard A.  
Grosse Pointe  
Benson, Clifford D.  
Gerbasi, Francis S.  
Taber, Rodman E.  
Kalamazoo  
Neerken, A. John  
Royal Oak  
Timmis, Hilary H.  
Southfield  
Barrett, Raymond J.

#### MINNESOTA

Crookston  
Deniord, Richard N.  
Duluth  
Fuller, Josiah  
Edina  
Nicoloff, Demetre M.  
Minneapolis  
Anderson, Robert W.  
Garamella, Joseph J.  
Humphrey, Edward W.  
Johnson, Frank E.

#### MISSISSIPPI

Jackson  
Hardy, James D.  
Johnston, J. Harvey, Jr.  
Neely, William A.  
Netterville, Rush E.

#### MISSOURI

Columbia  
Silver, Donald  
Kansas City  
Adelman, Arthur  
Ashcraft, Keith W.  
Barnhorst, Donald A.  
Benoit, Hector W., Jr.  
Holder, Thomas M.  
Killen, Duncan A.  
Mayer, John H., Jr.  
Padula, Richard T.  
Reed, William A.  
Mt. Vernon  
Campbell, Daniel C., Jr.

St. Louis  
Lewis, J. Eugene, Jr.  
Earner, Hendrick B.  
Bergmann, Martin  
Clark, Richard E.  
Ferguson, Thomas B.  
Kaiser, George C.  
Lucido, Joseph L.  
Roper, Charles L.  
Weldon, Clarence S.  
Willman, V. L.

#### NEBRASKA

Omaha  
Fleming, William H.  
Malette, William G.

Kiser, Joseph C.

Sellers, Robert D.

Lillehei, Richard C.

**NEW HAMPSHIRE**

Wangensteen, Owen H.

Hanover

Rochester

Tyson, M. Dawson

Bernatz, Philip E.

Jaffrey Center

Clagett, O. Theron

Woods, Francis M.

Danielson, Gordon K., Jr.

Kaye, Michael P.

McGoon, Dwight C.

Moersch, Herman

Olsen, Arthur M.

Payne, W. Spencer

**NEW JERSEY**

Brooklyn Sawyer, Philip N.

Bellville

Levowitz, Bernard S.

Gerard, Franklyn P.

Buffalo

Browns Mill

Adler, Richard H.

Fernandez, Javier

Andersen, Murray N.

East Orange

Lajos, Thomas Z.

Auerbach, Oscar

MacManus, Joseph E.

Haworth

Subramanian, Sambumurthy

Edie, Richard N.

Camden

Hillsdale

Camishion, Rudolph C.

Amberson, J. B.

Cooperstown

North Caldwell

Blumenstock, David A.

Wychulis, Adam R.

Fayetteville

New Brunswick

Bugden, Walter F.

Kunderman, Philip J.

Floral Park

Newark

Crastrapol, Philip

Aberdeen, Eoin

Mineola

Neville, William E.

Mangiardi, Joseph L.

Parsonnet, Victor

Moorestown

Pennsauken

Morse, Dryden P.

Pierucci, Louis, Jr.

North Hyde Park

Piscataway

Wisoff, B. George

Mackenzie, James W.	New Paltz
Short Hills	Johnson, Elgie K.
Demos, Nicholas J	New York
Timmes, Joseph L.	Bailey, Charles P.
South Orange	Beattie, Edward J., Jr.
Abel, Ronald M.	Bowman, Frederick O., Jr.
Tenafly	Boyd, Arthur D.
Gerst, Paul H.	Bregman, David
<b>NEW MEXICO</b>	Cahan, William G.
Albuquerque	Clauss, Roy H.
Edwards, W. Sterling	Conklin, Edward F.
Las Vegas	Cournand, Andre
Thai, Alan P.	Cunningham, Joseph, Jr.
Rociada	Findlay, C. W., Jr.
Wilson, Hugh E., III	Fitzpatrick, Hugh F.
Santa Fe	Ford, Joseph M.
Wilson, Julius L.	Friedlander, Ralph
<b>NEW YORK</b>	Gay, William A., Jr.
Albany	Giannelli, Stanley, Jr.
Alley, Ralph D.	Glenn, Frank
Kausel, Harvey W.	Green, George E.
McKneally, Martin F.	Holman, Cranston W.
Bay Shore	Holswade, George R.
Ryan, Bernard J.	Humphreys, G. H., II
Bronx	Hutchinson, John E., III
Altai, Lari A.	Isom, O. Wayne
Bloomberg, Allan E.	Jaretzki, Alfred, III
Hirose, Teruo	King, Thomas C.
Robinson, George	Kirschner, Paul A.
Bronxville	Lambert, Adrian
Prater, Robert W. M.	Litwak, Robert S.
	Maier, Herbert C.
	Malm, James R.
Martini, Nael	Charlotte

Nealon, Thomas F., Jr.	Robicsek, Francis
Okinaka, Arthur J.	Taylor, Frederick H.
Redo, S. Frank	Durham
Reemtsma, Keith	Hart, Deryl
Rubin, Morris	Oldham, H. Newland, Jr.
Spencer, Frank C.	Sabiston, David C.
Steichen, Felicien M.	Sealy, Will C.
Tice, David	Smith, David T.
Veith, Frank J.	Wechsler, Andrew S.
Wichern, Walter A., Jr.	Wolfe, Walter G.
Wolff, William I.	Young, W. Glenn, Jr.
Davidson, Louis R.	Greensboro
Patchogue	Deaton, W. Ralph, Jr.
Finnerty, James	Oteen
Plattsburg	Belts, Reeve H.
Potter, Robert T.	Pinehurst
Poughkeepsie	Fischer, Walter W.
Douglas, Richmond	Winston-Salem
Rochester	Cordell, A. Robert
Deweese, James A.	Hudspeth, Allen S.
Schwartz, Seymour I.	Johnston, Frank R.
Stewart, Scott	Meredith, Jesse H.
Zaroff, Lawrence I.	<b>OHIO</b>
Roslyn	Akron
Thomson, Norman B., Jr.	Falor, William H.
Saramac Lake	Chardon
Merkel, Carl G.	Mautz, F. R.
Decker, Alfred M., Jr.	Cincinnati
Scottsville	Carter, Noland B.
Emerson, George L.	Gonzalez, Luis L.
Setauket	Helmsworth, James A.
Dennis, Clarence	Rosenkrantz, Jens G.
Southampton	Cleveland
Heroy, William W.	Ankeney, Jay L.
Staten Island	Clatworthy, William H., Jr.
Garzon, Antonio A.	Cross, Frederick S.

Stony Brook

Soroff, Harry S.

Syracuse

Effler, Donald B.

Parker, Frederick B., Jr.

Valhalla

Reed, George E.

West Hampton Beach

Sarot, Irving A.

### **NORTH CAROLINA**

Asheville

Scott, Stewart M.

Sethi, Gulshan K.

Takaro, Timothy

Chapel Hill

Murray, Gordon F.

Starek, Peter J. K.

Wilcox, Benson R.

Groves, Laurence K.

Kay, Earle B.

Kilman, James W.

Loop, Floyd

Meckstroth, Charles V.

Williams, Thomas E., Jr.

Wright, George W.

Vasko, John S.

Dayton

Dewall, Richard A.

Pepper Pike

Mendelsohn, Harvey J.

Toledo

Blakemore, William S.

Selman, Morris W.

### **OKLAHOMA**

Oklahoma City

Elkins, Ronald C.

Felton, Warren L., II

Fisher, Robert Darryl

Greer, Allen E.

Munnell, Edward R.

Wilder, Robert J.

Williams, G. Rainey

Zudhi, M. Nazih

Tulsa

Guernsey, James M.

### **OREGON**

Days Creek

Miller, Arthur C.

Portland

Rosemond, George P.

Stayman, Joseph W.

Templeton, John Y., III

Wallace, Herbert W.

Pittsburgh

Bahnson, Henry T.

Ford, William B.

Magovern, George J.

Pontius, Robert G.

Rams, James J.

Ravitch, Mark M.

Rydal

Frobese, Alfred S.

Sayre

Sewell, William H.

Wynnwood

Lawrence, G. Hugh

Poppe, J. Karl

Starr, Albert

**PENNSYLVANIA**

Bethlehem

Snyder, John M.

Buck Hill Falls

Thompson, Samuel A.

Fairfield

McClenathan, James E.

Gladwyne

Johnson, Julian

Hamburg

Judd, Archibald R.

Haverford

Flick, John B.

Havertown

Chodoff, Richard J.

Hershey

Demuth, William E., Jr.

Pierce, William S.

Waldhausen, John A.

Lancaster

Witmer, Robert H.

Lumberville

O'Neill, Thomas J. E.

Philadelphia

Brockman, Stanley K.

Center, Sol

Edmunds, L. Henry, Jr.

Fineberg, Charles

Haupt, George J.

Lemmon, William M.

Lemole, Gerald M.

MacVaugh, Horace, III

Mendelssohn, Edwin

McKeown, John J., Jr.

Yardley

Sommer, George N., Jr.

**RHODE ISLAND**

Providence

Karlson, Karl E.

McEnany, M. Terry

Simeone, Fiorindo A.

**SOUTH CAROLINA**

Charleston

Bradham, Randolph R.

Hairston, Peter

Parker, Edward F.

Sade, Robert M.

Columbia

Almond, Carl H.

State Park

Ryan, Thomas C.

**TENNESSEE**

Chattanooga

Adams, Jesse E., Jr.

Hall, David P.

Jackson

Chandler, John H.

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.

Mundth, Eldred D.

Nemir, Paul, Jr.

Pate, James W.

Robbins, S. Gwin

Rosensweig, Jacob

Skinner, Edward F.

Nashville

Alford, William C., Jr.

Bender, Harvey W., Jr.

Dale, W. Andrew

Diveley, Walter L.

Foster, John H.

Gobbel, Walter G., Jr.

Johnson, Hollis E.

Sawyers, John L.

Scott, Henry W., Jr.

Stoney, William S.

Sewanee

Thrower, Wendell

### TEXAS

Austin

Hood, R. Maurice

Ross, Raleigh R.

Beaumont

Harrison, Albert

Dallas

Adam, Maurice

Davis, Milton V.

Holland, Robert H.

Kee, John L., Jr.

Lambert, Gary J.

Mitchel, Ben F., Jr.

Paulson, Donald L.

Platt, Melvin R.

McBurney, Robert P.

Lackland AFB

Stanford, William

Laporte

Barkley, Howard T.

Lubbock

Bricker, Donald L.

Dalton, Martin L, Jr.

San Antonio

Arom, Kit V.

Dooley, Byron N.

French, Sanford W., III

Grover, Frederick L.

Heaney, John P.

Hood, Richard H., Jr.

Nixon, James W.

Proctor, Oscar S.

Trinkle, J. Kent

Temple

Brindley, G. Valter, 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, Lawrence H.



Razzuk, Maruf A.  
Shaw, Robert R.  
Sugg, Winfred L.  
Urschel, Harold C., Jr.  
Galveston

Derrick, John R.  
Tyson, Kenneth R. T.  
Houston

Beall, Arthur C., Jr.  
Burdette, Walter J.  
Cooley, Demon A.  
Crawford, E. Stanley  
De Bakey, Michael E.  
Hallman, Grady L., Jr.  
Henly, Walter S.  
Mattox, Kenneth L.  
Morris, George C., Jr.  
Mountain, Clifton F.  
Norman, John C.  
Overstreet, John W.  
Reul, George J., Jr.  
Seybold, William D.  
Wukasch, Don C.

Richmond  
Bosher, Lewis H.  
Brooks, James W.  
Cole, Dean B.  
Greenfield, Lazar J.  
Gwathmey, Owen  
Johns, Thomas N. P.  
Lower, Richard R.  
South Arlington  
Klepser, Roy G.

**WASHINGTON**

Miller, Donald B.  
Chester Depot  
Adams, Herbert D.  
White River Junction  
Crandell, Walter B.

**VIRGINIA**

Arlington  
Conrad, Peter W.  
Charlottesville  
Crosby, Ivan K.  
Dammann, John F.  
Minor, George R.  
Muller, William H., Jr.  
Nolan, Stanton P.  
Wellons, Harry A., Jr.

Greatfalls  
Mills, Mitchell  
Lynchburg  
Moore, Richmond L.

Lepley, Derward, Jr.  
Litwin, S. Bertrand  
Mullen, Donald C.  
Narodick, Benjamin G.  
Tector, Alfred J.  
Weisel, Wilson  
Wausau  
Davila, Julio C.  
West Bend  
Gardner, Robert J.

**WYOMING**

Bellingham

Varco, Richard L.

Seattle

Anderson, Richard P.

Cantrell, James R.

Hill, Lucius D., III

Jarvis, Fred J.

Jones, Thomas W.

Mansfield, Peter B.

Merendino, K. Alvin

Miller, Donald W.

Mills, Waldo O.

Pinkham, Roland D.

Sauvage, Lester

Thomas, George I.

Spokane

Berg, Ralph, Jr.

Dillard, David H.

**WEST VIRGINIA**

East Charleston

Walker, James H.

Huntington

Littlefield, James B.

Morgantown

Tarney, Thomas J.

Warden, Herbert E.

**WISCONSIN**

La Crosse

Gundersen, Erik A.

Madison

Chopra, Paramjeet S.

Kahn, Donald R.

Young, William P.

Marshfield

Myers, William O.

Ray, Jefferson F., III

Teton Village

Kaunitz, Victor H.

**CANADA**

**ALBERTA**

Calgary

Miller, George E.

Edmonton

Callaghan, John C.

Meltzer, Herbert

Sterns, Laurence P.

**BRITISH COLUMBIA**

Vancouver

Allen, Peter

Ashmore, Philip G.

Harrison, Elliott

Tyers, G. Frank O.

Victoria

Stenstrom, John D.

West Vancouver

Robertson, Ross

**MANITOBA**

Winnipeg

Barwinsky, Jaroslaw

Cohen, Morley

**NEWFOUNDLAND**

St. John

Brownrigg, Garrett M.

Couves, Cecil M.

St. Anthony

Thomas, Gordon W.

**NOVA SCOTIA**

Halifax

Murphy, David A.

Kentville

Quinlan, John J.

Sautter, Richard D.

Milwaukee

Bonchek, Lawrence I.

Flemma, Robert J.

Hausmann, Paul F.

Johnson, W. Dudley

**ONTARIO**

Dorset

Mustard, William T.

Hamilton

Sullivan, Herbert J.

London

Heimbecker, Raymond O.

Ottawa

Keon, Wilbert J.

Sudbury

Field, Paul

Walker, George R.

Toronto

Delarue, Norman C.

Baird, Ronald J.

Bigelow, Wilfred G.

Cooper, Joel D.

Delarue, Norman C.

Ginsberg, Robert J.

Goldman, Bernard S.

Henderson, Robert D.

Key, James A.

Lockwood, A. L.

MacGregor, David c.

Joynt, George H. C.

Pearson, F. Griffith

Trimble, Alan S.

Trusler, George A.

Westbrook

Lynn, R. Beverly

Woodbridge

Laird, Robert

### **QUEBEC**

Montreal

Blundell, Peter E.

Bruneau, Jacques

Chiu, Chu-Jeng (Ray)

Dobell, Anthony R. C.

Grondin, Claude M.

Kunstler, Walter E.

MacLean, Lloyd D.

McIntosh, Clarence A.

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.

### **BRAZIL**

Sao Paulo

Zerbini, E. J.

### **ENGLAND**

Bristol

Belsey, Ronald

Buckinghamshire

Sellers, Sir Thomas Holmes

Hereford

Thompson, Vernon

### **JAPAN**

Sendai, Moniwa

Mohri, Hitoshi

### **NEW ZEALAND**

Auckland

Barratt-Boyes, Sir Brian

### **SCOTLAND**

Edinburgh

Logan, Andrew

### **SPAIN**

Lisbon

Macedo, Manuel E. Machado

Santander

London

Brock, Lord

Kennedy, J. H.

Ross, Donald

**FRANCE**

Paris

Dubost, Charles

**GERMANY**

Aachen

Messmer, Burno J.

**GUATEMALA**

Guatemala City

Herrera, Rudolfo

**HOLLAND**

Amsterdam

Boerema, I.

Leiden

Brom, Gerard A.

**INDIA**

Bikaner, Raiputana

Van Allen, Chester M.

Noakhali, Bangladesh

McCord, Colin W.

**IRELAND**

Dublin

O'Malley, Eoin

**ISRAEL**

Jerusalem

Blair, Emil

Duran, Carlos M. G.

**SWEDEN**

Karolinska

Crafoord, Clarence

Stockholm

Bjork, Viking O.

**SWITZERLAND**

Geneve

Hahn, Charles J.

Zurich

Senning, Ake

**VENEZUELA**

Caracas

Tricerri, Fernando E.

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

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

Section 11. Notwithstanding Section 10, any member of the Association over 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 five members: the President, Secretary, and Editor of the Association, and two members-at-large. 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.

**The American Association for Thoracic Surgery  
Charter Members  
June 7, 1917**

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

## 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

## 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	Theodor M. 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. Chirurgische 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	Severi 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. Darlinghurst2010 AUSTRALIA
29th	1978-79	Eduardo Otero Goto, M.D. Servicio de Cirugia Cardiovascular Ciudad Sanitaria "Le Fe" Valencia, SPAIN