

2002 ANNUAL MEETING PROGRAM



THE AMERICAN ASSOCIATION FOR THORACIC SURGERY 2001 – 2002

President Timothy J. Gardner, Philadelphia, PA
Vice-President Fred A. Crawford, Jr., Charleston, SC
Secretary Tirone E. David, Toronto, Ontario, Canada
Treasurer Richard A. Jonas, Boston, MA
Editor Andrew S. Wechsler, Philadelphia, PA

Councilors

James L. Cox (2002), Washington, D.C.
O. Wayne Isom (2003), New York, NY
Bruce W. Lytle (2005), Seattle, WA G.
Alexander Patterson (2002), St. Louis, MO
Carolyn E. Reed (2004), Charleston, SC
Historian John A. Waldhausen, Hershey, PA

Membership Committee Vaughn A. Starnes, Chairman, Los Angeles, CA
Joseph E. Bavaria, Philadelphia, PA
Joel D. Cooper, St. Louis, MO
Christopher M. Feindel, Toronto, Ontario, Canada
John J. Lamberti, New York, NY
Sara J. Shumway, Minneapolis, MN
Craig R. Smith, New York, NY

Association Representative, The American Board of Thoracic Surgery

Delos M. Cosgrove (2005), Cleveland, OH
Larry R. Kaiser (2007), Philadelphia, PA
Douglas J. Mathisen (2003), Boston, MA
Lawrence H. Cohn (2004), Boston, MA

Board of Governors, American College of Surgeons

Alex G. Little (2002), Las Vegas, NV

**THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY
2002 ANNUAL MEETING**

COMMITTEES

LOCAL ARRANGEMENTS

Robert B. Wallace, Chair

SPOUSE HOSPITALITY

Betty Wallace, Chair

PROGRAM COMMITTEE

Timothy J. Gardner (2002), Chair Philadelphia, Pennsylvania
W. Randolph Chitwood, Jr. (2004) Greenville, North Carolina
Fred A. Crawford, Jr. (2003) Charleston, South Carolina
Tirone E. David (2002) Toronto, Ontario, Canada
Larry R. Kaiser (2004) Philadelphia, Pennsylvania
Michael J. Mack (2002) Dallas, Texas
Constantine Mavroudis (2003) Chicago, Illinois
Roger B. B. Mee (2002) Cleveland, Ohio
D. Craig Miller (2002) Stanford, California
Freidrich W. Mohr (2002) Leipzig, Germany
Thomas W. Rice (2002) Cleveland, Ohio
Hartzell V. Schaff (2003) Rochester, Minnesota
Thomas L. Spray (2003) Philadelphia, Pennsylvania
Andrew S. Wechsler (2002) Philadelphia, Pennsylvania
Douglas E. Wood (2002) Seattle, Washington

**EVARTS A. GRAHAM MEMORIAL TRAVELING
FELLOWSHIP COMMITTEE**

Edward D. Verrier (2002), Chair Seattle, Washington
Frederick Bowman, Jr. (2005) Chapel Hill, North Carolina
Mark R de Leval (2004) London, United Kingdom
Pedro J. Del Nido (2004) Boston, Massachusetts
John A. Eleftheriades (2003) New Haven, Connecticut
David H. Harpole, Jr. (2005) Durham, North Carolina
Robert J. Keenan (2004) Pittsburgh, PA
Robert E. Michler (2004) Columbus, Ohio

**GRAHAM EDUCATION AND RESEARCH
FOUNDATION**

Tirone E. David (2002), President Toronto, Ontario, Canada
Richard A. Jonas (2002) Boston, Massachusetts
William T. Maloney (2002) Manchester, Massachusetts
Edward D. Verrier (2002) Seattle, Washington

YOUNG MEMBERS ADVISORY COMMITTEE

Robbin G. Cohen (2003), Chair Los Angeles, California
Sary F. Aranki (2002) Boston, Massachusetts
Pedro J. Del Nido (2002) Boston, Massachusetts
John P. Gott (2002) Atlanta, Georgia
Kent W. Jones (2003) Salt Lake City, Utah

James D. Luketich (2004) Pittsburgh, Pennsylvania
Michael A. Maddaus (2004) Minneapolis, Minnesota
James A. Magovern (2002) Pittsburgh, Pennsylvania
R. Scott Mitchell (2002) Stanford, California
Ralph S. Mosca (2004) New York, New York
Mehmet C. Oz (2002) New York, New York
Robert C. Robbins (2003) Stanford, California
David F. Torchiana (2002) Boston, Massachusetts
Clifford H. VanMeter (2003) New Orleans, Louisiana
Douglas E. Wood (2002) Seattle, Washington

ETHICS COMMITTEE

Martin F. McKneally (2002), Chair Toronto, Ontario, Canada
Lynda L. Mickleborough (2002) Toronto, Ontario, Canada
Marvin Pomerantz (2002) Denver, Colorado
Robert M. Sade (2002) Charleston, South Carolina
John A. Waldhausen (2002) Hershey, Pennsylvania

CARDIOTHORACIC RESIDENTS COMMITTEE

Ralph J. Damiano, Jr. (2002), Chair St. Louis, Missouri
Davis C. Drinkwater (2002) Nashville, Tennessee
Larry R. Kaiser (2002) Philadelphia, Pennsylvania
Steven J. Mentzer (2002) Boston, Massachusetts
G. Alexander Patterson (2002) St. Louis, Missouri
Frank W. Sellke (2002) Boston, Massachusetts
Thomas L. Spray (2002) Philadelphia, Pennsylvania
Edward D. Verrier (2002) Seattle, Washington

EDITORIAL ADVISORY COMMITTEE

Tirone E. David (2002), Chair Toronto, Ontario, Canada
Randall B. Griepp (2003) New York, New York
G. Alexander Patterson (2003) St. Louis, Missouri
D. Glenn Pennington (2003) Johnson City, Tennessee
Thomas L. Spray (2003) Philadelphia, Pennsylvania

NOMINATING COMMITTEE

David B. Skinner (2002), Chair New York, New York
Lawrence H. Cohn (2004) Boston, Massachusetts
Delos M. Cosgrove (2005) Cleveland, Ohio
James L. Cox (2006) Marco Island, Florida
Floyd D. Loop (2003) Cleveland, Ohio

PUBLICATIONS COMMITTEE

Tirone E. David, Chair Toronto, Ontario, Canada
Richard A. Jonas Boston, Massachusetts
William T. Maloney Manchester, Massachusetts

**THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY REPRESENTATIVES
2001-2002**

**AMERICAN COLLEGE OF SURGEONS ADVISORY COUNCIL FOR
CARDIOTHORACIC SURGERY**

William A. Baumgartner, Baltimore, Maryland (2003)
Edward L. Bove, Ann Arbor, Michigan (2003)

**AMERICAN MEDICAL ASSOCIATION
HOUSE OF DELEGATES**

L. Penfield Faber, Chicago, Illinois (2002)

**AMERICAN MEDICAL ASSOCIATION
CPT-4 ADVISORY COMMITTEE**

James M. Levett, Cedar Rapids, Iowa (2002)

**ASSOCIATION OF AMERICAN MEDICAL COLLEGES
COUNCIL OF ACADEMIC SOCIETIES**

Richard J. Shemin, Boston, Massachusetts (2002)
Gordon N. Olinger, Milwaukee, Wisconsin (2002)

**ASSOCIATION OF PHYSICIANS' ASSISTANTS
IN CARDIOVASCULAR SURGERY**

Bruce W. Lytle, Cleveland, Ohio (2002)

**COMMITTEE FOR COORDINATING CONTINUING
EDUCATION IN THORACIC SURGERY**

Douglas M. Behrendt, Iowa City, Iowa (2006)
Steven F. Boiling, Ann Arbor, Michigan (2005)
David B. Campbell, Hershey, Pennsylvania (2002)

**PERFUSION AFFAIRS
(AMSECT, ABCPT ACPE, CAHEA AND CAAHEP)**

Robert L. Kormos, Pittsburgh, Pennsylvania (2002)
Clifford H. Van Meter, New Orleans, Louisiana (2002)
Stanton P. Nolan, Charlottesville, Virginia (2002)

NATIONAL ASSOCIATION FOR BIOMEDICAL RESEARCH

Robert M. Mentzer, Jr. (2002)

AMERICAN ASSOCIATION OF BLOOD BANKS

Robert L. Thurer, Boston, Massachusetts (2002)
Gus J. Vlahakes, Boston, Massachusetts (2002)

FDA ADVISORY COMMITTEE

Erie H. Austin, III, Louisville, Kentucky (2002)

MEDICAL TECHNOLOGY LEADERSHIP FORUM

Bruce W. Lytle, Cleveland, Ohio (2002)

JOINT COUNCIL ON THORACIC SURGERY EDUCATION

Fred A. Crawford, Jr, Chair Charleston, South Carolina
Gordon F. Murray (STS) Morgantown, West Virginia
Mark B. Orringer (STS) Ann Arbor, Michigan
G. Alexander Patterson (AATS) St. Louis, Missouri
Eric A. Rose (AATS) New York, New York
William A. Baumgartner (ABTS) Baltimore, Maryland
Peter C. Pairolero (ABTS) Rochester, Minnesota
Robert L. Replogle (ACS) Chicago, Illinois
Constantine Mavroudis (ACS) Chicago, Illinois
John W. Brown (RRC) Indianapolis, Indiana
Douglas J. Mathisen (RRC) Boston, Massachusetts
Edward D. Verrier (TSDA) Seattle, Washington
Gordon N. Olinger (TSDA) Milwaukee, WI

THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY

Andrew S. Wechsler, *Editor*..... Philadelphia, Pennsylvania
Eugene H. Blackstone, *Associate Editor, Biostatistician*..... Cleveland, Ohio
Ralph J. Damiano, Jr., *Associate Editor*..... St. Louis, Missouri
D. Craig Miller, *Associate Editor*..... Stanford, California
G. Alexander Patterson, *Associate Editor*..... St. Louis, Missouri
Thomas L. Spray, *Associate Editor*..... Philadelphia, Pennsylvania
Martin F. McKneally, *Ethics Editor*..... Toronto, Ontario, Canada

ADVISORY EDITORIAL BOARD

Gary W. Akins..... Boston, Massachusetts
Friedhelm Beyersdorf..... Freiburg, Germany
R. Morton Bolman III..... Minneapolis, Minnesota
Ray Chu-Jeng Chiu..... Montreal, Quebec, Canada
Lawrence H. Cohn..... Boston, Massachusetts
Joseph S. Coselli..... Houston, Texas
Fred A. Crawford, Jr..... Charleston, South Carolina
Marc R. de Leval..... London, United Kingdom
Jean Deslauriers..... Sainte-Foy, Quebec, Canada
Davis C. Drinkwater, Jr..... Nashville, Tennessee
M. Arisan Ergin..... Englewood, New Jersey
J. William Gaynor..... Philadelphia, Pennsylvania
Donald D. Glower, Jr..... Durham, North Carolina
Frank L. Hanley..... Stanford, California
Alden H. Harken..... Denver, Colorado
Axel Haverich..... Hannover, Germany
Valluvan Jeevanandam..... Chicago, Illinois
Larry R. Kaiser..... Philadelphia, Pennsylvania
Antoon E. M. R. Lerut..... Leuven, Belgium
Robert M. Mentzer, Jr..... Lexington, Kentucky
Joseph I. Miller, Jr..... Atlanta, Georgia
John L. Ochsner..... New Orleans, Louisiana
Ivan M. Rebeyka..... Edmonton, Alberta, Canada
Thomas W. Rice..... Cleveland, Ohio
Robert C. Robbins..... Stanford, California
Jack A. Roth..... Houston, Texas
Valerie W. Rusch..... New York, New York
Hartzell V. Schaff..... Rochester, Minnesota

Frank W. Sellke.....	Boston, Massachusetts
David J. Sugarbaker.....	Boston, Massachusetts
Willem Van Oeveren.....	Groningen, Netherlands
Edward D. Verrier.....	Seattle, Washington
William G. Williams.....	Toronto, Ontario, Canada

**PAST PRESIDENTS OF
THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY**

<i>Year</i>	<i>Meeting Location</i>	<i>President</i>
1917-1918	Chicago, IL	Samuel J. Meltzer
1918-1919	Atlantic City, NJ	Willy Meyer
1919-1920	New Orleans, LA	Willy Meyer
1920-1921	Boston, MA	Rudolph Matas
1921-1922	Washington, DC	Samuel Robinson
1922-1923	Chicago, IL	Howard Lilienthal
1923-1924	Rochester, MN	Carl A. Hedblom
1924-1925	Washington, DC	Nathan W. Green
1925-1926	Montreal, QUE	Edward W. Archibald
1926-1927	New York, NY	Franz Torek
1927-1928	Washington, DC	Evarts A. Graham
1928-1929	St. Louis, MO	John L. Yates
1929-1930	Philadelphia, PA	Wyman Whittemore
1930-1931	San Francisco, CA	Ethan Flagg Butler
1931-1932	Ann Arbor, MI	Frederick T. Lord
1932-1933	Washington, DC	George P. Muller
1933-1934	Boston, MA	George J. Heuer
1934-1935	New York, NY	John Alexander
1935-1936	Rochester, MN	Carl Eggers
1936-1937	Saranac Lake, NY	Leo Eloesser
1937-1938	Atlanta, GA	Stuart W. Harrington
1938-1939	Los Angeles, CA	Harold Brunn
1939-1940	Cleveland, OH	Adrian V.S. Lambert
1940-1941	Toronto, ONT	Fraser B. Gurd
1943-1944	Chicago, IL	Frank S. Dolley
1945-1946	Detroit, MI	Claude S. Beck
1946-1947	St. Louis, MO	I. A. Bigger
1947-1948	Montreal, QUE	Alton Ochsner
1948-1949	New Orleans, LA	Edward D. Churchill
1949-1950	Denver, CO	Edward J. O'Brien
1950-1951	Atlantic City, NJ	Alfred Blalock
1951-1952	Dallas, TX	Frank B. Berry
1952-1953	San Francisco, CA	Robert M. Janes
1953-1954	Montreal, QUE	Emile Holman
1954-1955	Atlantic City, NJ	Edward S. Welles
1955-1956	Miami Beach, FL	Richard H. Meade
1956-1957	Chicago, IL	Cameron Haight
1957-1958	Boston, MA	Brian Blades
1958-1959	Los Angeles, CA	Michael E. DeBakey
1959-1960	Miami Beach, FL	William E. Adams
1960-1961	Philadelphia, PA <i>(Deceased 1/11/61)</i>	John H. Gibbon, Jr. Richard H. Sweet
1961-1962	St. Louis, MO	O. Theron Claggett
1962-1963	Houston, TX	Julian Johnson
1963-1964	Montreal, QUE	Robert E. Gross
1964-1965	New Orleans, LA	John C. Jones
1965-1966	Vancouver, BC	Herbert C. Maier
1966-1967	New York, NY	Frederick G. Kergin
1967-1968	Pittsburgh, PA	Paul C. Samson
1968-1969	San Francisco, CA	Edward M. Kent

1969-1970	Washington, DC	Hiram T. Langston
1970-1971	Atlanta, GA	Thomas H. Burford
1971-1972	Los Angeles,	John W. Strieder
1972-1973	CA Dallas, TX	Frank Gerbode
1973-1974	Las Vegas, NV	Lyman A. Brewer, III
1974-1975	New York, NY	Wilfred G. Bigelow
1975-1976	Los Angeles, CA	David J. Dugan
1976-1977	Toronto, ONT	Henry T. Bahnson
1977-1978	New Orleans, LA	J. Gordon Scannell
1978-1979	Boston, MA	John W. Kirklin
1979-1980	San Francisco, CA	Herbert Sloan
1980-1981	Washington, DC	Donald L. Paulson
1981-1982	Phoenix, AZ	Thomas B. Ferguson
1982-1983	Atlanta, GA	Frank C. Spencer
1983-1984	New York, NY	Dwight C. McGoon
1984-1985	New Orleans, LA	David C. Sabiston
1985-1986	New York, NY	James R. Malm
1986-1987	Chicago, IL	Norman E. Shumway
1987-1988	Los Angeles, CA	Paul A. Ebert
1988-1989	Boston, MA	W. Gerald Austen
1989-1990	Toronto, ONT	F. Griffith Pearson
1990-1991	Washington, DC	Keith Reemtsma
1991-1992	Los Angeles, CA	John A. Waldhausen
1992-1993	Chicago, IL	John L. Ochsner
1993-1994	New York, NY	Aldo R. Castaneda
1994-1995	Boston, MA	Robert B. Wallace
1995-1996	San Diego, CA	Mortimer J. Buckley
1996-1997	Washington, DC	David B. Skinner
1997-1998	Boston, MA	Floyd D. Loop
1998-1999	New Orleans, LA	Lawrence H. Cohn
1999-2000	Toronto, ONT	Delos M. Cosgrove
2000-2001	San Diego, CA	James L. Cox

**THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY
SECRETARIES**

1918-1923 Nathan W. Green
1923-1925 Charles Gordon Heyd
1925-1930 Ethan Flagg Butler
1930-1935 Duff S. Allen
1935-1947 Richard H. Meade
1947-1951 Brian Blades
1951-1956 Paul C. Samson
1956-1963 Hiram T. Langston
1963-1968 Henry T. Bahnson
1968-1973 Thomas B. Ferguson
1973-1978 Myron W. Wheat, Jr.
1978-1983 John L. Ochsner
1983-1988 Quentin R. Stiles
1988-1993 Martin F. McKneally
1993-1998 James L. Cox
1998- Tirone E. David

TREASURERS

1918-1923 Nathan W. Green
1923-1925 Charles Gordon Heyd
1925-1928 Ethan Flagg Butler
1928-1933 Carl Eggers

1933-1939 Edward D. Churchill
1939-1946 Isaac A. Bigger
1946-1954 William E. Adams
1954-1963 Julian Johnson
1963-1968 C. Rollins Hanlon
1968-1974 Paul C. Adkins
1974-1979 James R. Malm
1979-1984 Paul A. Ebert
1984-1989 Floyd D. Loop
1989-1994 William A. Gay, Jr.
1994-1999 Andrew S. Wechsler
1999- Richard A. Jonas

**THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY
82nd ANNUAL MEETING
Washington Convention Center
Washington, DC, May 5-8, 2002**

SUNDAY AFTERNOON, MAY 5, 2002

1:30 p.m. GENERAL THORACIC SURGERY FORUM SESSION

ROOM 15

Moderators: Shaf Keshavjee and Steven J. Mentzer

F1. Antisense Oligonucleotides Directed at the Bcl-X Gene Product Significantly Augment Chemotherapy Response in Mesothelioma

Mustafa K. Ozvaran*, Xiaobo X. Cao*, Steven D. Miller*, Brett Monia*, W. Roy Smythe*; Houston, TX and Carlsbad, CA

OBJECTIVE: Mesothelioma (MPM) is resistant to conventional chemotherapy. We have previously demonstrated that pharmacologic and antisense oligonucleotide (ASO) inhibition of bcl-xl gene expression engenders apoptotic cellular death in MPM cells. We evaluated the combination of antisense inhibition of the anti-apoptotic bcl-X gene product with conventional chemotherapeutic agents in MPM.

METHODS: Human MPM cell lines REN (epithelial) and I-45 (sarcomatoid) were utilized. The concentration of cis-platinum necessary to kill 50% of cells (IC50) was determined by XTT assay. Cells were then treated with IC50 of cis-platinum and a bcl-X ASO (ISIS 15999 - 0-200 nM) utilizing an oligofectamine delivery system. Monotherapy and sense oligonucleotide (SO) (ISIS 113524 - 0-200nM) plus cisplatinum were utilized as controls. Cell death with combination therapy was assessed with XTT. Isobologram analysis was performed to evaluate sub-additive, additive or synergistic effect.

RESULTS: The cis-platinum IC50 for REN (50 uM), and I-45 (1 uM) were determined. The combination of ASO plus cis-platinum was more effective at cell killing than SO plus cis-platinum with 70-90% of cells killed at 200nM ASO dose ($p < 0.05$ REN and I-45). Isobologram analysis

demonstrated additive or synergistic effect for the combination across all doses. Apoptosis was increased by morphologic analysis.

CONCLUSION: The combination of molecular (bcl-X ASO) and conventional chemotherapy treatment was significantly more effective than monotherapy or control combinations. As it appears likely that molecular therapies may not be effective alone, combinations such as bcl-X ASO and chemotherapy may be important for future treatment of resistant malignancies such as MPM.

**By Invitation*

F2. Clinical Implications of p53 Tumor Suppressor Gene Mutation and Protein Expression in Esophageal Adenocarcinoma: Results of A 10-Year Prospective Study

Alan G. Casson*, Susan C. Evans*, Amy Gillis*, Geoffrey A. Porter*, Paul J. Veugelers*, S. Jane Darnton*, Duane L. Guernsey*, Pierre Hainaut*; Halifax, Nova Scotia, Canada; Lyon, France

OBJECTIVE: To characterize the spectrum of p53 alterations (mutations and protein expression) in surgically resected esophageal adenocarcinoma (EADC), and to correlate molecular alterations with clinico-pathologic findings and outcome.

METHODS: Between 1991 and 2001, 91 consecutive patients with EADC underwent subtotal esophagectomy. No patient received induction therapy. Strict clinico-pathologic criteria were used to define primary EADC. Genomic DNA was extracted from esophageal tumors, each with matched histologically normal esophageal epithelium (internal control) from the resection margin. Polymerase chain reaction was used to amplify p53 exons 4-10. Mutations were studied by single-strand conformation polymorphism analysis and direct DNA sequencing. Immu-nohistochemistry (monoclonal antibody D07) was used to evaluate p53 protein distribution.

RESULTS: For all patients, 5-year overall survival (OS) was 25%, with median follow-up of 32 months. No p53 alterations were found in normal esophageal epithelium. 57% (n=52) of tumors had p53 alterations (missense mutations with protein expression, n=28; truncating mutations, n=19; protein overexpression only, n=5), and were associated with poor tumor differentiation (p=0.001), advanced (pTNM) stage (p=0.009), and number of involved lymph nodes (0, 1-3, >=4; p=0.04). Patients with p53 positive tumors (mutations and/or protein) had significantly reduced 5-year OS compared to patients with p53 negative tumors (15% vs. 46%; p=0.004).

CONCLUSION: We conclude that 1) p53 alterations (mutations and/or protein expression) are a significant predictor of reduced postoperative survival following surgical resection of EADC, and 2) p53 may be a clinically useful molecular marker for stratifying patients in future clinical trials.

**By Invitation*

F3. Adenoviral MDA-7 Induces Apoptosis in Lung Cancer Cells Through Mitochondrial Permeability Transition Independent Cytochrome C Release

Abujiang Pataer*, Sunil Chada*, Kelly K. Hunt*, Jack A. Roth, Stephen G. Swisher*; Houston, TX

OBJECTIVE: MDA-7 is a novel tumor suppressor gene that shares homology with the cytokine IL-10. Adenoviral gene transfer of MDA-7 (Ad-MDA-7) induces apoptosis in lung cancer cells resistant to p53 gene transfer. The mechanism of action is unknown, but may involve release of cytochrome c from the mitochondria with subsequent caspase activation.

METHODS: The lung cancer cell lines A549 (p53 resistant) and H1299 (p53 sensitive) were treated with Ad-MDA-7 and a control adenoviral vector expressing the luciferase reporter gene (Ad-Luc). Staurosporine was used to induce cytochrome c release through mitochondrial permeability transition (MPT) dependent pores and cyclosporine (CsA) was used to specifically inhibit these MPT dependent pores. Apoptosis was evaluated by FACS analysis of subdiploid populations.

RESULTS: 48 hours after Ad-MDA-7 treatment of H1299 and A549 lung cancer cells, sharp increases in cytosolic cytochrome c levels were noted which were followed by caspase activation, apoptosis and cellular death. The release of cytochrome c from the mitochondria occurred without changes in the mitochondrial membrane potential. Unlike with Staurosporine (ST), an MPT pore dependent agent, Ad-MDA-7 release of cytochrome c and apoptosis (see figure) was not blocked by cyclosporine A (CsA) in either H1299 (see figure) or A549 cells (data not shown) suggesting an MPT pore independent pathway.

CONCLUSION: These data suggest that MDA-7 induces apoptosis in lung cancer cells through mitochondrial cytochrome c release. This process is not dependent on mitochondrial membrane changes and occurs through MPT independent pores. This unique mechanism of action may allow treatment of lung cancer patients resistant to p53 gene transfer and other MPT dependent cell death processes.

**By Invitation*

F4. Prognostic Significance of VEGF Expression, CD31, CD34, CD105 and Tumor Vessel Invasion in Radically Resected IB-IIA Non-small Cell Lung Cancer

Tommaso C. Mineo*, Vincenzo Ambrogi*, Giuseppe Tonini*, Carla Rabitti*, Italo Nofroni*; Rome, Italy

OBJECTIVE: We evaluate the prognostic value of tumor angiogenesis assessed by vascular endothelial growth factor (VEGF) overexpression, microvessel density (MVD), and tumor vessel invasion (TVI) in patients who underwent radical resection for stage (IB-IIA) non-small cell lung cancer (NSCLC).

METHODS: Sixty-five patients (53 males, 12 female, mean age 63.3±9-42) undergoing complete surgical resection (minimal n=14, lobectomy n=35, pneumonectomy n=16) of pathologic stage IB (n=50) and IIA (n=15) NSCLC were retrospectively evaluated. Nobody underwent chemo or radiotherapy before or after surgery. Minimum followup period for alive patients was 36 months (mean 42.3±33.4). Paraffin embedded tumor specimen were stained for VEGF and specific MVD markers: CD31, CD34 and CD105. VEGF overexpression was considered when more than 25% of carcinoma cells were stained. CD31, CD34, CD105 were evaluated per high power field at

400xmagnification; median values were chosen as cutoff points. TVI was assessed after vessel delineation by CD34.

RESULTS: Five-year Kaplan-Meier survival rate significantly correlated with VEGF overexpression (35% vs 92%, $p=0.001$), high MVD by CD34 (29% vs 69%, $p=0.001$), and TVI (18% vs 60%, $p=0.001$), whereas MVD by CD31, or by CD 105, age, sex, histology, grading did not. Multivariate analysis selected MVD by CD34 ($p=0.001$, OR=3.3, CI 95%=1.5-7.0) and TVI ($p=0.0002$, OR=3.6, CI95%=1.8-7.2): the presence of both risk-factors was highly predictive of poor outcome ($p=0.00001$, OR=12.5, CI 95%=3.7-35).

CONCLUSION: VEGF overexpression, high MVD by CD34 and TVI in stage IB-IIA NSCLC are associated with worsening prognosis. Simultaneous presence of the last two positive factors strongly orientates to target those patients at further adjuvant treatment.

**By Invitation*

F5. Comparison of Mutational Alterations in Involved (N1) Lymph Nodes to Primary Tumor in Stage 2 Lung Cancer May Predict Outcome

Hiran C. Fernando*, Neil A. Christie*, Percivai O. Buenaventura*, Peter F. Person*, Eizaburo Sasatomi*, Sydney D. Finkelstein*, Samuel A. Yousem*, Ryan Soose*, James D. Luketich; Pittsburgh, PA

OBJECTIVE: Surgical resection is the standard treatment for Stage II non-small cell lung cancer (NSCLC) but recurrence rates approach 60%. The aim of this study was to compare mutational changes in involved lymph nodes (LNs) and primary tumors from patients with Stage II NSCLC to determine if risk factors for recurrence could be identified.

METHODS: Patients with Stage II NSCLC (excluding T3N0) treated by resection were included. Microdissection was performed on primary tumor and involved LNs. Analysis was performed across 9 genomic loci using PCR amplification. The severity of mutational changes was calculated by dividing the fractional allelic loss (FAL) in histologically positive lymph nodes by the FAL in primary tumor. Patients were stratified into high risk (HR) (FAL ratio 1 or more) and low risk (LR) (FAL ratio less than 1) groups.

RESULTS: Forty patients (27 male/13 female) were studied. Median follow-up was 34 months. Mean age was 66 (42-85) years. Median survival was not reached in LR and was 38 months in HR patients ($p=ns$). Disease-free survival (DPS) was not reached in LR and was 24 months in HR patients ($p=ns$). In the adenocarcinoma subset ($n=19$), there were no deaths in LR patients compared to a median survival of 24 months in HR patients($p=0.01$); also DPS was not reached in LR patients compared to 14 months in HR patients ($p=0.05$).

CONCLUSION: Patients with adenocarcinoma of the lung with greater mutational damage in metastatic LNs compared to their primary tumor had significantly worse survival. Further evaluation using this approach should be investigated.

**By Invitation*

F6. Flavopiridol Enhances Depsipeptide-mediated Apoptosis in Lung and Esophageal Cancer Cells via Activation of Mitochondria! Death Signal Pathways

Dao M. Nguyen*, William D. Schrump*, G. Aaron Chen*, John H. Stewart

IV*, Federico A. Steiner*, David S. Schrump; Bethesda, MD

OBJECTIVE: Alterations in chromatin structure resulting from aberrant histone acetylation and mutational events which inactivate Rb and p53 tumor suppressor pathways cooperate to disrupt cell cycle regulation in thoracic malignancies. The present study was undertaken to determine if the cdk inhibitor, Flavopiridol (Fl.), could enhance cytotoxicity mediated by the histone deacetylase (HDAC) inhibitor Depsipeptide FR901228 (DP) in NSCLC and esophageal cancer (EsC) cells.

METHODS: H460 and H322 NSCLC and TE12 and TE13 EsC cells as well as primary normal cells were treated with sequential DP (1 to 50 ng/ml x 6h) / FL (50 to 200 nM x 3d). Cell proliferation in drug treated and control cells was assessed by MTT assays. ApoBrdU techniques were used to evaluate apoptosis following drug exposure in the presence or absence of caspase inhibitors. Mitochondria membrane potential depolarization was evaluated by JC-1 staining techniques.

RESULTS: FL dramatically enhanced DP-mediated growth inhibition in cultured cancer cells (Table 1). FL potentiated DP-mediated apoptosis in all cancer lines irrespective of p53 status. Sequential DP/FL mediated a 3- to 7-fold increase in cells with depolarized mitochondria membrane potentials relative to DP treatment alone (4 to 7% in DP-treated cells versus 22 to 51% in DP/FL-treated cells). Apoptosis following sequential DP/FL exposure was inhibited approximately 80% by selective inhibitors of caspase 3 or caspase 9. Minimal cytotoxicity was observed in primary normal cells following DP/FL exposure.

Cell Lines	H460	H322	TE12	TE13
DP IC50 (ng/ml)*				
DP alone	12.6 ± 2.3	25.8 ± 3.7	14.1 ± 2.7	20.5 ± 3.1
DP+FL(100nM)	4.8 ± 0.6	6.4 ± 2.1	3.2 ± 1.1	2.4 ± 0.6
Apoptosis **				
Control	2.2 ± 0.8	2.0 ± 0.5	3.0 ± 1.0	3.5 ± 1.2
FL(100nM)	1.0 ± 0.5	4.0 ± 1.2	1.1 ± 0.4	12.8 ± 3.6
DP	5.1 ± 2.3	5.3 ± 1.0	2.07 ± 5.8	21.9 ± 4.6
DP+FL	3.26 ± 2.4	4.62 ± 8.1	9.42 ± 6.5	87.5 ± 8.8

*Concentrations of DP that inhibited 50% of cell growth, n = 5, Mean ± SD

**% TUNEL-positive cells, n = 3, Mean ± SD

CONCLUSION: Under clinically achievable conditions, FP markedly enhances DP mediated apoptosis in lung and esophageal cancer cells via activation of mitochondrial death pathways.

**By Invitation*

F7. Mediastinal Metastatic Progression of Human Female Lung Adenocarcinoma is Associated with Cyclooxygenase-2 Over-expression in a Murine Model

Ross M. Bremner*, Costanzo A. DiPerna*, Robert D. Bart*, Yanling Ma*,

Michael E. Bowdish*, Vaughn A. Starnes; Los Angeles, CA

OBJECTIVE: To describe a murine model of orthotopic tumor growth with mediastinal nodal metastasis utilizing human lung adenocarcinoma, and compare cyclooxygenase-2 (COX-2) over-expression in primary tumor with nodal disease.

METHODS: Human lung adenocarcinoma cells(CRL5908, female nonsmoker) were implanted under direct visualization through the parietal pleura in the upper lobe, left lung(2 million cells/animal) of SCK mice. Mice were killed at 1, 2, 3, and 4 weeks (n=5, 6, 15, 6 respectively). Primary tumor and metastatic disease were assessed grossly and histologically. COX-2 enzyme expression was identified in primary and mediastinal metastatic lesions using immunohistochemistry.

RESULTS: At 1 week, small upper lobe tumor nodules were present in all animals. At 2 weeks, all animals exhibited primary tumor growth with histologic lymphatic invasion. At 3 weeks, 14/15(93.3%) animals had mediastinal lymph node metastases and a significant decline in projected weight gain($p=0.035$). At 4 weeks, extensive primary tumor growth was noted with invasion of the chest wall 3/6(50%), contralateral lung 1/6(16.7%), and large mediastinal lymph nodes 6/6(100%). In vitro, $75/500 \pm 20$ (15% \pm 4%, n=4) of CRL5908 confluent cells were immunoreactive for COX-2. In primary tumors, COX-2 immunoreactivity was focal and limited to $125/500 \pm 20$ cells(25% \pm 4%); whereas, in metastatic lesions COX-2 immunoreactivity increased to $400/500 \pm 35$ cells(80% \pm 7%).

CONCLUSION: Orthotopic xenotransplantation of human lung adenocarcinoma cells in SCID mice is a reproducible model that mirrors the human nodal metastatic process. COX-2 over-expression may be implicated in the development of mediastinal metastases for lung adenocarcinoma. Modulation of COX-2 may have the potential to interfere with the metastatic process.

**By Invitation*

F8. The Immunological Role of Thymectomy in the Treatment of Myasthenia Gravis with Special Reference to Reduction of Anti-Acetylcholine Receptor Antibody

Meinoshin Okumura*, Mitsunori Ohta*, Yoshitaka Fujii*, Hikaru Matsuda;
Osaka, Japan; Aichi, Japan

OBJECTIVE: Myasthenia gravis (MG) is an autoimmune disease caused by anti-acetylcholine receptor antibody (AChRAb) and well known for its relation with abnormality of the thymus such as thymoma and lymphoplasia characterized by germinal center formation in the medulla. Extended thymectomy (KT) has been shown to contribute to remission of MG through decrease in the serum AChRAb. The aim of this study is to elucidate the role of thymectomy in the treatment of MG focusing on the immunological characteristics of the MG thymus.

METHODS: Immunological examination was done in 34 AChRAb-positive and non-thymomatous MG patients who experienced KT. The lymphocytes were freshly recovered from the resected thymus, and the number of lymphocytes present in 1 gram of the thymic tissue was counted. The number of B lymphocytes present in 1 gram of the thymic tissue (B cell number /g

thymus) was calculated by examining the CD 19 expression by flow cytometric method. Serum AChRAB was also measured, and its reduction after 1"1" was evaluated in terms of the proportion of AChRAB at 1 year after ET to that prior to ET (% AChRAB at 1 year).

RESULTS: There was a significant inverted correlation between the logarithmic number of B cell number/lg thymus and % AChRAB at 1 year. ($r=-1.476$, $p=0.004$)

CONCLUSION: Resection of the thymus which has a large population of B lymphocytes was shown to be effective in the reduction of serum AChRAB. The significance of B lymphocytes in the thymus in the pathogenesis of MG was suggested.

3:00 p.m. BREAK

**By Invitation*

3:15 p.m. GENERAL THORACIC AND CARDIOPULMONARY SURGERY FORUM SESSION

ROOM 15

Moderators: Irving L. Kron and Vaughn A. Starnes

F9. Evaluation of a New Preservation Strategy for Lung Retrieval from Non-Heart-Beating Donors

Thorsten Wittwer*, Ulrich F.W. Franke*, Antonia Fehrenbach*, Felix Pfeifer*, Tim Sandhaus*, Thomas Mueller*, Harald Schubert*, Peter Petrow*, Hartwig Kosmehl*, Joachim Richter*, Thorsten Wahlers*; Jena and Goettingen, Germany

OBJECTIVE: Successful lung transplantation is limited by the severe scarcity of donor organs. Currently, aside from living-related lobar donors, transplanted organs are retrieved from brain-dead heart beating donors. A potential method to extend the donor pool would be lung retrieval from non-heart-beating donors (NHBD). However, in most studies standard antegrade organ preservation via the pulmonary artery was investigated. So far, no studies in NHBD exist using retrograde preservation with Perfadex via the left atrium.

METHODS: Using heparinized pigs ($n=5$, 30-35 kg), asystole was induced, and ventilation was continued for 90 minutes. Lungs were then retrogradely preserved with Perfadex and stored for 3 hours. Left lung transplantation was performed followed by ligation of the right pulmonary artery and bronchus. All results were compared to sham-operated animals. Relative oxygenation capacity index (ROCI), pO_2/FiO_2 , hemodynamics, PIP and dynamic lung compliance were monitored for 6 hours. Quantification of alveolar edema was performed stereo-logically. Statistical analysis comprised ANOVA analysis for repeated measurements.

RESULTS: No mortality was observed. During flush, continous elimination of blood clots via the pulmonary artery venting site could be observed. ROCI, pO_2/FiO_2 and compliance were comparable to shams, but the latter revealed lower PAP and PVR ($p=0.044$). Stereology revealed more edema in NHBD-grafts ($p=0.03$), while W/D-ratio and neutrophil-infiltration were similar between groups.

CONCLUSION: Use of lungs from NHBD results in excellent outcome. Especially in the NHBD with high risk for intravascular thrombi, retrograde preservation might optimize graft quality by eliminating debris via the afferent artery incision. This novel approach might also be considered

when a brain-dead organ donor becomes hemodynamically unstable prior to onset of organ harvest. Further trials with longer warm and cold ischemic periods are initiated to further elucidate this promising approach of donor pool expansion.

**By Invitation*

F10. Hyperinflation During Lung Preservation Results in Increased Reperfusion Injury

Mayank R. Patel*, Steven M. Fiser*, Aditya K. Kaza*, Stewart M. Long*,

Victor E. Laubach*, John A. Kern*, Curtis G. Tribble, Irving L. Kron;

Charlottesville, VA

OBJECTIVE: Reperfusion injury after lung transplantation remains a perplexing and unpredictable problem. Most surgeons preserve the lung inflated. Our hypothesis is that donor lung hyperinflation during storage contributes to early allograft dysfunction during reperfusion.

METHODS: To test our hypothesis we used an isolated, blood-perfused, ventilated rabbit lung model. Group I lungs underwent immediate reperfusion (control). Group II lungs (low inflation, maintained at 6 mm Hg) and group III lungs (high inflation, maintained at 20 mm Hg) were stored for 4 hours in saline (4°C). Measurements of arterial oxygenation (PO₂, mm Hg), pulmonary artery pressure (PAP, mmHg), peak inspiratory pressure (PIP, cm H₂O), and wet to dry weight ratio (WTD) were obtained.

RESULTS: Results after 20 minutes of reperfusion are illustrated in the table below (mean + SEM).

Group	P02	PAP	PIP	WTD
I-Immediate (n=6) (control)	383±66.9	31.5±5.8	20.7±1.2	5.6±0.91
II-low inflation (n=5)	197±57.3	17.4±4.3	19.2±1.2	7.2±0.88
III-High inflation (n=6)	66±10.7*	47.5±11.3	24.5±1.4*	9.0±1.2
ANOVA	*p=0.02 I vs. III, p=0.08 II vs. III	p=0.06 II vs. III	*p=0.01 II vs. III	p=1.00 II vs. III

CONCLUSION: High inflation during cold storage results in considerable pulmonary dysfunction as illustrated by a significant decrease in pO₂ and increase in PIP as well as a trend towards higher PAP. Careful monitoring of inflation pressure during storage may improve graft function following lung transplantation.

**By Invitation*

F11. Importance of the NO-Pathway in Experimental Lung Preservation with Perfadex Solution

Johannes M. Albes*, Thorsten Wittwer*, Antonia Fehrenbach*, Daniel

Meyer*, Ulrich F.W. Franke*, Harald Brandes*, Thorsten Wahlers*; Jena and

Goettingen, Germany

OBJECTIVE: Optimal preservation of postischemic graft function is essential in lung transplantation. Single antegrade flush perfusion with modified Euro-Collins (EC) solution represents the standard technique worldwide. However, in recent years growing evidence suggests the superiority of extracellular-type Perfadex solution over EC. During ischemia, endogenous pulmonary NO synthesis is decreased, therefore therapeutic stimulation of the NO pathway might be beneficial in ameliorating ischemia/reperfusion (I/R) damage. However, research mainly focuses on NO-supplementation of intracellular solutions, while no studies exist when Perfadex with the NO-donor nitroglycerin is compared to EC-solution using an isolated perfused rat lung model.

METHODS: Eight lungs, each, were preserved using Perfadex with (PER-NO) or without (PER) nitroglycerine (NTG, 0.1 mg/ml) and compared to low-potassium-Euro-Collins (LPEC). Postischemic lungs were re-ventilated and reperfused with bovine erythrocytes. Oxygenation capacity (paO₂-pvO₂), pulmonary vascular resistance (PVR) and inspiratory pressures (PIP) were monitored continuously. Edema was assessed both by wet-to-dry weight ratio (W/D) and by stereological analysis. Statistics were performed using ANOVA for repeated measurements.

RESULTS: Oxygenation capacity of Perfadex-preserved groups was higher as compared to LPEC (p=0.03). Using nitroglycerine, flush-perfusion time was reduced, and PER-NO-protected lungs showed superior Oxygenation capacity compared to PER-organs (p=0.01). Furthermore, PVR and PIP values were improved in the nitroglycerin-group (p=0.01). No differences in pulmonary edema were found among the groups.

CONCLUSION: Perfadex provides superior lung preservation in terms of postischemic oxygenation capacity than Euro-Collins solution. Supplementation of the NO-pathway by nitroglycerin further enhances pulmonary hemodynamics, respiratory parameters and especially oxygenation of Perfadex-preserved organs and might be an easily applicable tool in clinical lung transplantation.

**By Invitation*

F12. Depopulated Venacaval Homograft: A New Venous Conduit

Mahmoud Malas*, Craig J. Baker*, Suzanne M. Quardt*, Mark L. Barr*,

Winfield J. Wells; Los Angeles, CA

OBJECTIVE: Completion of Fontan is frequently performed by using an extracardiac conduit (ECC) between the IVC and PA. Most centers use a Cortex graft for the ECC and because re-endothelialization is unlikely, anticoagulation is used for a variable period. This study explores the use of an alternate large caliber venous conduit.

METHODS: The SVC was replaced in seven mini-pigs using either a Cortex interposition graft (2) or a depopulated (acellular) cryopreserved superior venacaval homograft (5). After 6 months

animals were sacrificed and the grafts were examined for patency and histology including immunostaining. No anticoagulation was used.

RESULTS: Cortex grafts had a cross-sectional luminal narrowing ranging from 16-40%. Histology showed only partial intimal ingrowth with excessive subendothelial fibrosis and early calcification. In contrast the depopulated venous homografts showed minimal luminal narrowing ranging from 2-9% ($p < .05$ vs Cortex). These grafts were completely repopulated by the recipient with an endothelial lining which stained positively for Factor VIII and a subendothelial region appropriately re-cellularized by myofibroblasts which stained positively for smooth muscle actin and procollagen. There was no evidence of an immune response to the venous homografts as judged by negative staining for CD4 and CDS. Thrombus was not seen in any of the grafts.

CONCLUSION: Depopulated, cryopreserved venacaval homografts may be superior conduits for cavopulmonary connection during completion Fontan by the ECC technique.

**By Invitation*

F13. Does Antegrade Cerebral Perfusion Protect the Brain During Deep Hypothermic Circulatory Arrest?

Vicki Lynn Mahan*, Saroja Ilangovan*, Reuben Cuison*, Jyothi Patil*, Sarah Docktor*, Vincent Rizzo*, Michel N. Ilhawi; Oak Lawn and Chicago, IL

OBJECTIVE: Antegrade cerebral perfusion during deep hypothermic circulatory arrest (DHCA) is selectively used to protect the brain during cardiac surgery in neonates. This study compares cerebral protection using no cerebroprotection and using variable antegrade flow rates of cerebroprotection during DMCA.

METHODS: Twenty neonatal piglets underwent 60 minutes of DHCA. No cerebroprotection was used in 5 piglets (Group 1.) Cold (15 C) antegrade cerebral perfusate (Plasmalyte-A, Landrace pig whole blood to maintain a hemoglobin between 5 and 6 grams/dl, sodium bicarbonate - 25 meq/liter, heparin 300 units/liter, and 25% albumen - 10 cc/liter) was administered through the innominate artery at 10 cc/kg/min in 5 (Group 2), at 25 cc/kg/min in 5 (Group 3), and at 50 cc/kg/min in 5 (Group 4.) Samples for serum lactate, pyruvate, S-100B protein, and CPK-BB were drawn from a catheter placed in the internal jugular vein prior to cardiopulmonary bypass and after discontinuation of cardiopulmonary bypass at 5 minutes, 5 minutes, 30 minutes, and 6 hours, respectively. Piglets were sacrificed 6 hours post bypass and the brain harvested for histologic and immunologic studies. Extent of damage and apoptosis was assessed using a semiquantitative score of 0 to 4.

RESULTS: Apoptosis and necrosis was apparent in all groups. The mean score for Group 1 was 2.67, for Group 2 was 2.40, for Group 3 was 2.47, and for Group 4 was 2.23. Although pathologic changes appeared to be greater in the piglets that did not have antegrade cerebral perfusion during DHCA, this was not statistically significant. (Table)

	GROUP 1	GROUP 2	GROUP 3	GROUP 4	P
LAC PRE	1.84±1.43	1.54±.51	1.78±.88	1.88±.33	NS
LAC POST	6.4±1.58	6.6±1.57	8.64±3.94	7.12±1.32	NS
PYR PRE	.27±.15	.3±.12	.2±.12	.24±.13	NS

PYR POST	.3±.1	.42±.19	.32±.22	.36±.22	NS
S-100 PRE	1568±676	1241±643	1105±500	1170±410	NS
S-100 POST	1543±263	1754±621	1737±593	1741±336	NS
CPK PRE	136±117	216±216	156±68	148±92	NS
CPK POST	264±56	270±211	142±93	297±238	NS

CONCLUSION: These results suggest that unmodified antegrade cerebral perfusion during DHCA does not provide adequate protection of the brain during conditions simulating clinical practice and that serum lactate, pyruvate, S-100B protein, and CPK-BB do not reflect the extent of brain damage incurred during DHCA with or without antegrade cerebropoplegia.

**By Invitation*

F14. High-Flow Selective Cerebral Perfusion Maintains Cerebral Microvascular Oxygen Tension During and After Deep Hypothermic Circulatory Arrest

William M. DeCampli*, Gregory Schear*, Steven Schultz*, Richard J. Myung*, Jennifer Creed*, David F. Wilson*, Anna Pastuszko*, Philadelphia, PA

OBJECTIVE: Deep hypothermia circulatory arrest (DHCA) is associated with neurologic injury. Selective cerebral perfusion (SCP) during DHCA has been proposed to maintain cerebral oxygen delivery. We obtained quantitative measurements of microvascular oxygen tension (PO₂) and distribution in the cerebral cortex of neonatal piglets during DHCA and SCP at high and low flows.

METHODS: Fourteen neonatal piglets underwent cardiopulmonary bypass (CPB). After cooling to 18°C, the animals were subjected to 90 minutes of either: DHCA (n=6), low-flow SCP (SCP-20, 20 cc/kg/min, n=4), or high-flow SCP (SCP-40, 40cc/kg/min, n=4), followed by four hours of post-CPB reperfusion. SCP was accomplished by advancing the aortic cannula into the innominate artery. Microvascular PO₂ was measured by oxygen-dependent phosphorescence quenching of the phosphor, Pd-tetra (4-carboxyphenyl) tetrabenzoporphyrin. One-way ANOVA was used to calculate significant differences (p<0.05).

RESULTS: In the figure, results are expressed as means. Points a and b represent significant decreases in cortical oxygen tension compared to each group's pre-CPB value. Histograms of the oxygen distribution in the cortex revealed the presence of regions of hypoxia that persisted after DHCA alone that were not observed in either SCP group.

CONCLUSION: Both SCP schemes, as opposed to DHCA, allow a return of cortical P_O₂ during reperfusion to pre-CPB values and prevent the persistence of regions of cortical hypoxia that may contribute to neurologic injury following DHCA. However, only SCP-40 completely eliminates a significant drop in cortical PO₂ in all perfusion phases.

**By Invitation*

F15. Activation of Mitogen-Activated Protein Kinases in the Brains of Neonatal Piglets During Circulatory Arrest and Low Flow Cardiopulmonary Bypass

Alon S. Aharon*, Mahlon D. Johnson*, Davis C. Drinkwater, Oliver B. Lao*,

Seth Brindis*, Megan Thunder*, Paul A. Chang*; Nashville, TN; St. Louis,

MO

OBJECTIVE: Mitogen-activated protein kinases (MAPKs) are important intermediates in signal transduction pathways implicated in neuronal and endothelial dysfunction secondary to ischemia reperfusion injury. Members of the mitogen-activated protein kinase/extracellular regulated kinase and extracellular signal-regulated kinase 1 and 2 (ERK1/ERK2) cascade may participate in the pathogenesis of post ischemic cellular injury inducing phosphorylation/activation of ERK1/ERK2 during low flow Cardiopulmonary bypass (LFCPB) or circulatory arrest (CA). This study evaluates ERK1/ERK2 phosphorylation and its cellular distribution in cerebral cortical areas susceptible to ischemia in a neonatal piglet model subjected to LFCPB or CA.

METHODS: Neonatal piglets were subjected to CA (n=5), LFCPB (n=5), or normal flow CPB (control) at 20°C for a period of 45 minutes. Following 24 hours of post CPB recovery the piglet brains were harvested for analysis. After fixation, right and left anterior and posterior watershed, temporal lobes, hippocampi, basal ganglia, thalamus, cerebellum and vermis, mesencephalon, pons and medulla were evaluated using hematoxylin and eosin stained sections. A section of cortex exhibiting ischemic injury was evaluated with a rabbit polyclonal antibody to phosphorylated ERK1/ERK2 and avidin biotin complex immunohistochemistry.

RESULTS: Compared to the control, brains from LFCPB and CA piglets exhibited early ischemic changes primarily in the watershed zones, hippocampus, basal ganglia, thalamus and cerebellum with overlapping severity and distribution between groups. No significant phospho-ERK1/2 immunoreactivity was detected in the control, however immunoreactivity was demonstrated in endothelia of leptomeningeal and cortical blood vessels of LFCPB and CA groups.

CONCLUSION: These findings indicate that this neonatal piglet model is able to demonstrate early cortical ischemic changes following CA and LFCPB. Moreover, preliminary findings indicate that ERK1/ERK2 may be involved in early ischemia/reperfusion injury and cerebral endothelial dysfunction. Studies in progress utilizing inhibitors of ERK1/ERK2 phosphorylation may represent a novel pharmacologic intervention which may mitigate ischemic injury following focal cerebral ischemia.

5:30 p.m. ADJOURN - WELCOMING RECEPTION

HALL B

**By Invitation*

SUNDAY AFTERNOON, MAY 5, 2002

1:30 p.m. CARDIAC SURGERY FORUM SESSION

ROOM 33

Moderators: Frank W. Sellke and Henry M. Spotnitz

F16. Bone Marrow Stromal Cells can Differentiate into Cardiac Lineage and Contract Synchronously with Cardiomyocytes by Direct Cell-to-Cell Interaction in Vitro

Shinji Tomita*, Takeshi Nakatani*, Shinya Fukuhara*, Takayuki Morisaki*,
Chikao Yutani*, Soichiro Kitamura; Osaka, Japan

OBJECTIVE: Cardiac environmental factors are thought to be powerful differentiation inducers. In this study, we simulated the cardiac environment using a co-culture system and evaluated cardiomyogenic differentiation of bone marrow stromal cell and synchronous contraction with other cardiomyocytes.

METHODS: In group 1, bone marrow stromal cells derived from transgenic mouse expressing GFP (GFP-BM) were cultured (n=5). In group 2, GFP-BM and cardiomyocytes (CM) from neonatal rat were co-cultured separately with membrane (n=5). In group 3, GFP-BM were co-cultured with CM (n=5). We cultured these cells for 7 days and evaluated cardiomyogenic differentiation of GFP-BM morphologically and immunohistologically.

RESULTS: In group 1 and 2 GFP-BM did not show any contraction and myogenic phenotype for 7 days. In contrast, in group 3, GFP-BM incorporated into CM mole and revealed myotube-like formation on the first day. On the second day some GFP-BM started to contract synchronously with CM. Isoproterenol (25nM) increased heart rate of GFP-BM and CM from 80 to 100/min. One fifth of GFP-BM contracted synchronously with CM on the seventh day. GFP-BM showed myosin heavy chain-positive at 2.5_0.9% (mean_S.E) and cardiac specific troponin I-positive at 1.9_0.5%, respectively. Desmin and atrial natriuretic peptide was also seen in GFP-BM and connexin 43 was detected between CM and GFP-BM.

CONCLUSION: Direct interaction with cardiomyocytes is essential for bone marrow stromal cells to differentiate into cardiomyocytes and contract synchronously. This co-culture system is a simple tool to simulate the cardiac environment in vitro.

**By Invitation*

F17. Angiogenic Growth Factors vs Cellular Therapy for Myocardial Infarction

Juan Carlos Chachques*, Fabricio Duarte*, Barbara Cattadori*, Abdel Shafy*,

Patrick Meimoun*, Marie Christine Iliou*, Patrick Bruneval*, Pantelis

Argyriadis*, Jean Noel Fabiani*, Alain Carpentier; Paris, France

OBJECTIVE: Locally delivered angiogenic growth factors and cell implantation have been proposed for patients presenting myocardial infarcts without possibility of percutaneous or surgical revascularization. The goal of this study was to compare the effects of these techniques, in an experimental model of myocardial infarct.

METHODS: Myocardial infarction was created in 18 sheep by ligation of 2 coronary arteries. Animals were then randomized in 3 groups: Group 1: infarction + injection of culture medium. Group 2: infarction + injection of Vascular Endothelial Growth Factor (VEGF 100 microgr). Group 3: infarction + implantation of autologous cultured myoblast (70 million). Myocardial injections and cell implantation were performed 3 weeks after creation of infarction. Evaluation included:

serum troponin I postoperative levels; 3 months after infarction: echocardiography (2D and Color Kinesis) and immunohistological studies for quantitative analysis of capillaries.

RESULTS: Three animals died intraoperatively. Serum troponin rose to 45.6 ± 4.7 ng/ml at post-infarction day 2. Echocardiography showed a significant limitation of LV dilatation in the cell group: 57 ± 5.5 ml (control group: 74.4 ± 5 ml, VEGF group: 68 ± 1.6 ml), Color Kinesis showed improvement of regional fraction area change (RFAC) only in the cell group, from $3-6 \pm 0.8$ to $21 \pm 1.5\%$, $p < 0.05$. The number of capillaries increased significantly in the peri-infarct area of VEGF group: 1036 ± 175 (control group: 785 ± 131 , cell group: 830 ± 75).

CONCLUSION: In the cell therapy group regional contractility improved and heart dilatation was reduced as compared to either VEGF or control, thus reducing postischemic remodeling. Significant angiogenesis was demonstrated only in the VEGF group, without improvement of ventricular function and remodeling.

**By Invitation*

F18. Isolation, Expansion, and Genetic Modification of Bone Marrow Derived Mesenchymal Stem Cells for In Vivo Repair of Damaged Myocardium

Abeel A. Mangi*, Victor J. Dzau*; Boston, MA

OBJECTIVE: The characterization and conditions for differentiation of bone marrow (BM) derived cells for cardiac repair need further definition. We isolated, characterized, and propagated a CD34-/c-kit+ population of BM-derived mesenchymal stem cells (MSCs). These cells were transduced to stably express reporter genes, and differentiated into cardiomyocytes, thereby normalizing cardiac function when transplanted into ischemic myocardium.

METHODS: The buffy coat from adult rat bone marrow was harvested. MSCs attached preferentially to uncoated plastic surfaces, and were further purified using negative immuno-magnetic bead sorting. MSCs were efficiently retrovirally transduced with green fluorescent protein (GFP), and Lac Z. Reverse-transcriptase PCR and immunohistochemistry were performed. Sixty minutes after coronary artery ligation in rats, 1×10^7 MSCs were injected into the border zone of the ischemic left ventricle. Echocardiography was performed before injury, and two weeks later, immediately prior to sacrifice.

RESULTS: Undifferentiated MSCs expressed connexin-43 and c-kit but did not express hematopoietic markers CD34, CD45, CD11b; or mature cardiac markers troponin, myosin heavy chain or desmin. After sacrifice, newly regenerated cardiomyocytes in the free wall and apex of the left ventricle exhibited extensive blue coloration by beta-galactosidase staining. Separately, cells expressing GFP also expressed alpha-sarcomeric-actin, troponin, myosin heavy chain, N-cadherin and connexin-43, suggesting that the MSCs derived cardiomyocytes were mature, and were mechanically and electrically coupled with native myocardium. Echocardiography revealed normalization of fractional shortening and ejection fraction in the cell-treated group.

CONCLUSION: This novel strategy suggests that bone marrow derived MSCs can be expanded to sufficient scale ex vivo, and genetically engineered to treat damaged myocardium.

**By Invitation*

F19. Myocardial Viability 24 Hours after Orthotopic Heart Transplantation from Non-heart-beating Donors

Juergen Martin*, Georg Lutter*, Christian Ihling*, Matthias Siepe*, Susanne Wagner*, Koppany Sarai*, Friedhelm Beyersdorf; Freiburg, Germany

OBJECTIVE: Experimental studies have shown that orthotopic heart transplantation from non-heart-beating donors is feasible, but irreversible damage could not be excluded due to the short observation time. The aim of this study was to assess viability and myocardial function of these hearts 24 hours after transplantation.

METHODS: Cardiac arrest in pigs was induced by exsanguination. After 30 min of unprotected normothermic ischemia controlled reperfusion with leucocyte-depleted cold blood cardioplegia containing the Na-H-exchange inhibitor HOE 642 and adenosine was performed. The hearts were stored in ice-cold solution and transplanted orthotopically. Controlled reperfusion in the recipient was started during implantation and was continued for 30 min. In the control group, animals were transplanted in a conventional fashion using Bretschneider's HTK solution. Cold ischemia time was 4 hours in both groups.

RESULTS: All animals could be weaned from cardiopulmonary bypass. Preload recruitable stroke work of the left ventricle 24 hours after transplantation in the control vs. experimental group was 108 ± 24 % versus 103 ± 18 % of baseline. Myocardial blood flow of the left and right ventricle was increased to 146 ± 32 % and 176 ± 51 % in the control group versus 176 ± 29 % and 194 ± 27 % in the experimental group. Myocardial oxygen consumption was 11.2 ± 2.1 versus 12.8 ± 2.2 ml/100g/min at baseline and 11.6 ± 2.6 versus 13.2 ± 3.1 ml/100g/ min 24 hours after transplantation (n.s.). Histological examination using Luxol fast blue staining revealed that 2.6 ± 4.8 % of myocytes in the control group versus 1.8 ± 1.9 % in the experimental group were damaged irreversibly (n.s.).

CONCLUSION: In this model, myocardial viability was well preserved after orthotopic heart transplantation from non-heart-beating donors. Recovery of donor hearts harvested from NHBD's after 30 minutes of normothermic ischemia is comparable to organs harvested from beating heart donors if the above mentioned preservation technique is used. These results could encourage the use of so-called marginal donor hearts and help to expand the limited donor pool.

**By Invitation*

F20. Toward More Structure-Oriented Left Ventricular Volume Reduction Surgery for Better Outcomes

Tadaaki Koyama*, Kazunobu Nishimura*, Yoshiharu Soga*, Oriyanhan Unimonh*, Koji Ueyama*, Hisayoshi Suma, Masashi Komeda*; Kyoto, Hayama and Kamakura, Japan

OBJECTIVE: Volume reduction surgery (VRS) has not yielded predictable outcomes. To avoid disruption of the muscle-band helical loop structure of the heart, we modified VRS to spare the LV apex and to decrease the basal diameter, and confirmed better LV function after the modified VRS in acute heart failure model. The purpose of this study was to test the efficacy of modified VRS in chronic DCM model.

METHODS: Eleven beagle dogs were weekly injected 0.7mg/kg of doxorubicin (in total 5 times) at left coronary ostium. VRS was performed one month thereafter. Dogs were randomized to the following two groups: Group A, apex-sacrificing VRS (i.e., simulating conventional VRS) @by plicating LV wall between papillary muscle bases from middle to apex (n=5); Group B, apex-sparing (i.e., modified) VRS - plication from base to middle (n=6).

RESULTS: One week after VRS, all 6 dogs in Group B survived while only 3 out of 5 in Group A. LVEF of group B was better soon and one week after VRS than that of group A. LVEDP of group A remarkably increased one week after VRS, but not in group B. LVDD of group A remarkably increased one week after VRS, but not in group B. LV histological examination of group A showed increase of interstitial fibrosis and nuclear swelling two weeks after VRS, in contrast to group B.

	Base line	Pre-op.	Post-op.	1 week post-op.
Group A-LVDD (mm)	28±0.2	35±0.5	31±0.5	39±1.6
Group B-LVDD(mm)	27±0.5	35±0.2	29±0.5	32±0.3
Group A-LVEF(%)	74±1.1	49±1.9	56±2.5	41±7.7
Group B-LVEF(%)	78±1.9	47±1.8	72±1.9	65±1.4
Group A-CO(L/min)	3.9±0.3	3.7±0.3	2.1±0.2	2.8±0.5
Group B-CO(L/min)	3.3±0.2	3.5±0.2	2.3±0.1	2.7±0.3
Group A-LVEDP(mmHg)	1±0.2	6±1.3		12±2.2
Group B-LVEDP(mmHg)	1±0.3	4±0.9		5±1.3

CONCLUSION: LV function, fibrosis, and late redilatation after apex-sparing VRS was better than that after apex-sacrificing VRS. Apex-sparing VRS may improve the results of LVVRS.

**By Invitation*

F21. Passive Ventricular Constraint Improves Myocardial Energetics in a Model of Heart Failure Secondary to Acute Infarction

James J. Pilla*, Daniel J. Brockman*, Aaron S. Blom*, Qing Yuan*, Michael

A. Acker; Philadelphia, PA

OBJECTIVE: Left ventricular (LV) remodeling after infarction is characterized by dilatation and myocardial function impairment. This study hypothesized that limiting dilatation would improve myocardial energetics and efficiency in a model of heart failure secondary to myocardial infarction (MI).

METHODS: Heart failure was created in ten sheep by ligating the diagonal vessels emanating from the LAD. Five sheep were randomized to the cardiac support device (CSD) (Acorn Cardiovascular) group while, the remaining five served as controls. A terminal PVA/MRI study was performed on both groups at two-months post-infarct.

RESULTS: Myocardial energetics was calculated by combining the PVA end-systolic elastance with the pressure-volume data obtained from MRI. A comparison of the two groups at two-months post-infarct demonstrates that the CSD group had superior myocardial energetics (Table, Mean (SEM), $p < 0.05$, t-test). Total pressure-volume area (PVarea) was significantly greater in the control

versus the CSD group. External work (EW) was similar in both groups, whereas potential energy (PE) or, the energy lost as heat, was significantly greater in the controls. This resulted in a higher left ventricular efficiency (LVEff) for the CSD animals. In addition, the left ventricular oxygen consumption (LWO2) calculated from the PVarea demonstrates that the CSD group consumed less oxygen than the controls while generating comparable external work.

	PV area (mmHg/ml)	EW (mmHg/ml)	PE (mmHg/ml)	LVEff (%)	LWVO2 (mlO2/beat)
Control	3402 (386)	1387 (370)	2015(251)	39.4 (6.7)	0.0726 (0.0066)
CSD	2210 (206)*	1325(156)	885(110)*	59.8 (4.2)*	0.0519 (0.0035)*

CONCLUSION: This study demonstrates that limiting dilatation after an MI will improve myocardial energetics and efficiency. These findings may be attributed to the reduction in myocardial wall stress resulting from a decrease in LV volume and an increase in wall thickness. It suggests that placement of a CSD clinically after an MI will improve the working conditions of the heart, preventing the ultimate development of heart failure.

**By Invitation*

F22. Downregulation of Myocardial SERCA Expression Predicts Ventricular Decompensation in Aortic Regurgitation Patients

Pedro A. Catarino*, Graham V. Harrod*, Xu Y. Jin*, Narain Moorjani*, Kay

E. Davies*, Stephen Westaby; Oxford, England

OBJECTIVE: Molecular phenotyping provides a potential avenue for objective load-independent assessment of cardiac function. Impaired myocardial function has been associated with defective excitation-contraction coupling and altered expression of calcium handling genes. A marker to predict the onset of left ventricular (LV) failure could improve decision making in aortic regurgitation (AR). We performed LV biopsies in patients with AR undergoing valve replacement in order to compare the expression of calcium handling genes with cardiac function.

METHODS: 50 meg biopsies were obtained from the left ventricle of 22 AR patients and snap frozen in liquid nitrogen. Biopsies were also taken from 6 patients with normal LV function (NLV) and from 4 patients with end-stage dilated cardiomyopathy (DCM) for comparison. Ventricular function was assessed preoperatively by echocardiography. Competitive reverse transcriptase-polymerase chain reactions (RT-PCR) of samples spiked with serial dilutions of a gene-specific deletion construct were used to quantitate the mRNA of interest. We examined mRNA for the sarcoplasmic reticulum ATPase-2a (SERCA), for the calcium release channel (CRC) and for the sodium-calcium exchanger (NCE). Expression levels were normalised for GAPDH mRNA.

RESULTS: SERCA mRNA expression was reduced in patients with heart failure compared to NLV. In particular, it was downregulated by two-fold on average ($p < 0.05$) in the transition from compensated AR to AR with heart failure. No relationship was identified for CRC or NCE expression.

	NLV	AR (preserved LV)	AR (impaired LV)	DCM
SERCA	0.96±0.40	1.38±0.56	0.85±0.44	0.41±0.28
CRC	0.91±0.65	1.13±0.65	0.84±0.47	0.33±0.24

NCE 0.85±0.56 1.01±0.58 0.95±0.48 1.25±1.85

Arbitrary Units ± standard deviation

CONCLUSION: Competitive RT-PCR enables mRNA quantitation of multiple genes in limited myocardial biopsies. SERCA downregulation at mRNA level predicts decompensation of LV function in AR.

**By Invitation*

F23. Left Ventricular Reverse Remodeling After Surgical Correction of Aortic Stenosis Correlates to Myocardial Gene Expression

Thomas Walther*, Andreas Schubert*, Volkmar Falk*, Christian Binner*, Niko Doll*, Jan Gummert*, Friedrich W. Mohr; Leipzig, Germany

OBJECTIVE: Surgical correction of aortic stenosis leads to reverse remodeling of the left ventricle. Aim of this study was to understand myocardial gene expression (Extracellular Matrix (ECM) and Renin Angiotensin System (RAS)) under these circumstances.

METHODS: A standard supracoronary banding model was applied to 44 growing sheep (age 6-8 month) for controlled induction of left ventricular hypertrophy (LVH) (A). Surgical therapy to completely release the pressure gradient was performed 8.3±1 months later (B). Final measurements were performed after another 10.1±2 months (C). Hemodynamic evaluations as well as subtractive hybridisation and competitive PCR to quantify mRNA expression for Matrix Metalloproteinases-1,-2,-3,-9, their tissue inhibitors -1,-2,-3 (TIMPs), ACE, angiotensin receptors 1 and 2 (AT1-R and AT2-R) were performed.

RESULTS: Left ventricular function and cardiac index were stable throughout the study. Left ventricular mass index (LVMI) was 82±21g (A), 150±33g (B) and 78±18g (C), p<0.01. Myocardial fiber diameter was 11.3±0.8 (A), 15.9±1.2 (B) and 11.4±1 (C) um, p<0.01. AtB gene expression was significantly increased for all parameters besides TIMP-3 and AT2-R that were decreased. At C there was a significant reverse in gene expression with all parameters returning to baseline levels. There was a significant correlation between changes in LVMI and myocardial gene expression.

CONCLUSION: LVH as present in patients with aortic stenosis is associated with significant changes in myocardial gene expression. Surgical therapy leads to complete reverse remodeling with regression of both LVH and myocardial gene expression to baseline levels.

3:00 p.m. BREAK

**By Invitation*

3:15 p.m. CARDIAC SURGERY FORUM SESSION

ROOM 33

Moderators: Frank W. Sellke and Henry M. Spotnitz

F24. Early Preconditioning Without Hypotension Prevents Spinal Cord Injury Due to Descending Aortic Occlusion

Ioannis K. Toumpoulis*, George E. Drossos*, Vassiliki Malamou-Mitsi*,

Demosthenes G. Katritsis*, Constantine E. Anagnostopoulos; Ioannina and

Athens, Greece; New York, NY

OBJECTIVE: Postoperative neurologic deficits following thoracic aortic reconstruction vary widely. Our previous study showed that delayed ischemic preconditioning (PC) could prevent spinal cord injury due to occlusion of the descending thoracic aorta in pigs. We investigated early preconditioning in the same model.

METHODS: Twenty eight pigs were divided in five groups: Group I (n=6) underwent a sham operation. Group II (n=9) underwent aortic occlusion for 20 minutes. Group III (n=6) underwent aortic occlusion for 35 minutes (no PC). Group IV (n=6) underwent aortic occlusion for 20 min without hypotension and, 1 h and 20 min later, aortic occlusion for 35 min (early PC). Group V (n=6) underwent aortic occlusion for 20 min and, 48 h later, aortic occlusion for 35 min (late PC). Aortic occlusion was performed by using two balloon occlusion catheters placed fluoroscopically just above the diaphragm and at the aortic bifurcation. At 24, 48 and 120 h, after the end of the experiment, neurologic evaluation according to Tarlov score was performed by an independent observer.

RESULTS: There was no significant difference in blood pressure proximal and distal to the occlusion between groups. Group IV (early PC) had a better neurologic outcome at 24 (mean Tarlov score 4 vs 2.33), 48 (4 vs 2) and 120 h (4 vs 2) in comparison with group III ($p<0.002$, $p<0.001$, $p<0.001$). This compared favorably with group V late PC ($p<0.002$, $p<0.001$, $p<0.002$ in comparison with group III, respectively). Histologic evaluation at 120 h disclosed severe neurologic damage in animals with poor Tarlov score.

CONCLUSION: Early PC without hypotension, as well as late PC, protects against spinal cord injury after aortic occlusion

**By Invitation*

F25. A New Equine Pericardial Stentless Valve

Xavier Michel Mueller*, Eric Eeckhout*, Frank Stumpe*, Ludwig Karl von

Segesser; Lausanne, Switzerland

OBJECTIVE: Small aortic valve replacement remains a hemodynamic challenging problem. A new bioprosthesis (3F Therapeutics, Lake Forest, CA) was designed to further improve the hemodynamic performance currently achieved with Stentless bioprosthesis. This valve consists of a tubular structure assembled from 3 equal sections of equine pericardial material with virtually no foreign material except for a thin polyester ring. Its hemodynamic performance was compared to that of a commercially available Stentless prosthesis in a bovine model.

METHODS: Ten calves (56.5±3kg) received a 19mm 3F valve (3F group, n=5) or a 19mm Stentless control valve (control group, n=5). The animals were fully equipped for hemodynamic monitoring and transvalvular gradient measurements. After implantation, dopamine was infused in increasing doses and the hemodynamic values were recorded at each step of 100µ/min increase.

RESULTS: Mean transvalvular gradient at 4.5L/min was 3.3±0.5mmHg (+ISO) in the 3F group and 6±0.7mmHg in the control group ($p<0.0001$), and at 6.5L/min, 7.9±0.2mmHg and

11.3±2.1mmHg respectively (p<0.0001). The effective orifice area at 4.5L/min was 2.5±0.2cm² in the 3F group and 1.7±0.1cm² in the control group (p<0.0001), and at 6.5L/min, 2.5±0.2cm² and 2±0.2cm² respectively (p<0.0001).

CONCLUSION: This new bioprosthesis without stent and without supporting wall, which has its commissures fixed directly to the aorta, outperforms in vivo all existing designs.

**By Invitation*

F26. Ischemia in Three Left Ventricular Regions- Insights into the Pathogenesis of Acute Ischemic Mitral Regurgitation

Tomasz A. Timek*, David T.M. Lai*, David Liang*, Frederick A. Tibayan*,

George T. Daughters*, Paul Dagum*, Sidney Lo*, Trevor Hastie*, Neil B.

Ingels*, D. Craig Miller; Stanford and Palo Alto, CA

OBJECTIVE: Acute posterolateral ischemia in sheep results in ischemic mitral regurgitation (IMR), the pathogenesis of which is incompletely understood. We induced acute ischemia in three different LV regions in an attempt to clarify these mechanisms

METHODS: Six adult sheep had radiopaque markers placed on the left ventricle, mitral annulus (MA), central edge of each mitral leaflet, and anterior (APM) and posterior (PPM) papillary muscle tip and base. After 6-8 days, animals were studied with biplane videofluoroscopy and transesophagealechocardiography before (Pre-isch) and during (Isch) ischemia induced by sequential balloon occlusion of the LAD, distal LCx (DLCX), and proximal LCx (PLCX) coronary arteries. MA area (MAA) and distance from each PM tip to mid-anterior MA (PM-MA) were calculated from 3-D marker coordinates. Systolic leaflet edge separation distance was calculated at end LV ejection. PM contraction was determined as % change in distance between tip and base markers from end-diastole (ED) to end-systole (ES).

RESULTS: A similar degree of LV dysfunction was induced by the three ischemic insults. *p<0.05 vs Pre-isch by paired t-test

	LAD Pre-isch	LAD Isch	DLCX Pre-isch	DLCX Isch	PLOi Pre-isch	PLCX Isch
MR (0-4)	0.5±0.5	0.7±0.8	0.5±0.5	0.8±1.1	0.5±0.5	2.0±1.1*
MAA ED (mm ²)	739±202	759±144	725±171	776±178*	711±151	846±201*
Leaflet Separation (mm)	4.7±1.8	4.5±1.5	4.1±1.2	4.9±1.4	4.3±1.8	7.4±2.3*
APM-MA ES (mm)	48.4±4.9	49.6±5.7	48.1±1.6	48.4±5.0	48.2±4.9	49.6±5.1*
PPM-MA ES (mm)	50.1±4.1	51.2±3.7*	50.1±4.2	51.1±4.2*	50.0±4.2	52.6±3.9*
APM Contraction (%)	15±5	3±4*	14±8	14±9	12±7	12±8
PPM Contraction (%)	16±7	16±8	16±8	1±6*	21±8	1±5*

CONCLUSION: Acute IMR occurred only after proximal LCx occlusion, and was associated with increased leaflet edge separation and substantial MA dilatation and displacement of both PMs away from the mid-anterior MA. Papillary muscle contractile dysfunction did not contribute significantly. Altered valvular and subvalvular 3-D geometry must both be considered in surgical approaches to repair acute IMR.

**By Invitation*

F27. Esmolol is Superior to Potassium For Preserving Myocardial Metabolism and Endothelial Function During Clinical Warm Cardioplegic Arrest

Marcio Scorsin*, Nawwar Al Attar*. Alexandre Mebezaa*, Jacques Callebert*,
Alain Ruffenach*, Richard Raffoul*, Susanna Salvi*, Ramzi Ramadan*, Paul
Le Besnerais*, Jean-Michel Maillat*, Patrick Nataf*, Arrigo Lessana*; St
Denis and Paris, France

OBJECTIVE: To compare the effects of potassium (K) and esmolol (E) on myocardial oxygen consumption (MVO₂) and coronary released nitric oxide (NO) during retrograde warm blood cardioplegia.

METHODS: Forty-one patients operated on for isolated aortic valve stenosis were randomly assigned to continuous coronary infusion with either K (40 mmol/30 min n=18) or E (500 mg/30 min, n=23). MVO₂ and coronary lactate release were analyzed by simultaneous blood samplings from the cardioplegia perfusion line (CPL) and left coronary ostium (LCO) 10 & 30 minutes after aortic cross clamping (ACC). Coronary NO release was quantified by measurements of NO₂/NO₃ (Griess method) in CPL and LCO, 30 min after ACC. Hemodynamic parameters were recorded by Swan-Ganz catheter in addition to pre- & post-operative echocardiographic measurements and determination of plasma troponin I (TnI) levels. Data are presented as mean + SD and compared by Anova.

RESULTS: Although cardioplegia flow rate and pressure were similar, E markedly reduced MVO₂ as compared to K, 10±1 vs 18±2 ml O₂/min (p<0.0001) and 11±2 vs 20±6 ml O₂/min, (p<0.0001) at 10 min and 30 min, respectively. Coronary lactate production was similar in both groups at 10 and 30 min (all inferior to 0.22 mmol/min) indicating adequate myocardial perfusion in all patients. Furthermore, E dramatically reduced coronary NO release (E, 1.5±0.2 µM/min vs K group, 10.9±2.4 µM/min, p=0.04). Hemodynamic parameters and TnI remained unchanged post-operatively with either form of cardioplegia.

CONCLUSION: Esmolol provides potentially superior myocardial protection compared with potassium by reducing myocardial oxygen consumption and preventing coronary endothelial activation.

**By Invitation*

F28. Poly-ADP-Ribose Polymerase-Inhibition Protects Against Myocardial and Endothelial Reperfusion Injury After Hypothermic Cardiac Arrest.

Gabor Szabo*, Terezia Andrasi*, Volker Buhmann*, Violetta Kekesi*,
Susanne Bahrle*, Csaba Szabo*, Siegfried Hagl*; Heidelberg. Germany;
Budapest, Hungary; Beverly, MA

OBJECTIVE: Free radical production and related cytotoxicity during ischemia/reperfusion may lead to DNA strand-breakage which activates the nuclear enzyme poly-ADP-ribose

polymerase(PARP) and initiates an energy consuming, inefficient repair cycle, with transfer of the ADP-ribosyl moiety of NAD + to protein acceptors. We investigated the effects of PARP inhibition on myocardial and endothelial function during reperfusion after hypothermic cardiac arrest.

METHODS: Twelve anesthetized dogs, underwent hypothermia cardiopulmonary bypass. After 60 minutes of hypothermic cardiac arrest, reperfusion was started after application of either saline vehicle (control, n = 6), or PJ34 (10 mg/kg) a selective PARP-inhibitor (n=6). Left ventricular hemodynamic variables were measured by a combined pressure-volume-conductance catheter and the slope of the end-systolic pressure volume relationship (Ees) was calculated at baseline and after 60 minutes of reperfusion. Left anterior descending coronary blood flow (CBF), endothelium-dependent vasodilatation to acetylcholine (ACH) and endothelium-independent vasodilatation to sodium nitroprusside (SNP) were also determined.

RESULTS: The administration of PJ34 led to a significantly better recovery (given as percent of baseline) of left ventricular systolic function (dp/dt: 91±7% vs. 68±10%, p<0.05 and Ees 87±6 % vs. 66±6%, p<0.05). CBF was significantly higher in PJ34 group (32±4 vs. 21±3, ml/ min, p<0.05). While the vasodilatory response to SNP was similar in both groups, ACH resulted in a significantly higher increase in CBF in the PJ34 group (59±10% vs. 28±15%, p<0.05).

CONCLUSION: PARP inhibition improves the recovery of myocardial and endothelial function after hypothermic cardiac arrest.

**By Invitation*

F29. Pretreatment with Statins Enhances Myocardial Protection During Coronary Revascularization

Harold L. Lazar, Yusheng Bao*, Yu Zhang*, Shelia Bernard*; Boston, MA

OBJECTIVE: Previous studies have shown that statins reduce myocardial ischemic events in patients with elevated serum cholesterol; however their effects during coronary surgery is unknown. This experimental study was undertaken to determine whether pretreatment with statins would enhance myocardial protection and minimize ischemic injury during revascularization of acutely ischemic myocardium.

METHODS: In 20 pigs (35-40kg), the second and third diagonal arteries were occluded for 90 minutes, followed by 45 minutes of blood cardioplegic arrest and 180 minutes of reperfusion. Ten pigs received Atorvastatin (40mg PO QD) for 21 days prior to surgery; 10 others received no statins. Ischemic damage was assessed by the need for cardioversions for ventricular arrhythmias, regional wall motion scores (WMS) were determined by two-dimensional echocardiography, endothelial function was assessed by bradykinin induced coronary artery relaxation using organ chamber methodology, and infarct size was calculated from the Area Necrosis/ Area Risk (AN/AR) using histochemical staining.

RESULTS: Results are Mean ± Standard Error

	Statins	No Statins	P Value
Serum Cholesterol Pre Op(mg/dl)	59±5.0	60±5.1	P>0.9
# Cardioversions	0.11±0.11	2.77±0.22	P<0.001
WMS(4= normal to -1 =dyskinesia)	2.8±0.04	1.5±0.07	P<0.001

Maximum Coronary Relaxation(%)	38±5.4	7±4.0	P<0.001
AN/AR (%)	22.4±1.14	40.0±1.70	P<0.01

CONCLUSION: Pretreatment with statins enhances myocardial protection during surgical revascularization by decreasing ventricular irritability, improving regional wall motion, lowering infarct size and preserving endothelial function. These beneficial effects occurred in the absence of significant changes in serum cholesterol from non-treated animals suggesting that an alternative mechanism may be responsible for the enhanced myocardial protection.

**By Invitation*

F30. Cardioplegic Arrest Induces Apoptosis Signal-Pathway in Myocardial Endothelial Cells and Cardiac Myocytes.

UvveM. Fischer*, Oliver Klass*, Ulrike Stock*, Jerry Easo*, Hans J. Geissler*,

Juergen H. Fischer*, Wilhelm Bloch*, Uwe Mehlhorn*; Cologne, Germany

OBJECTIVE: Myocardial ischemia/reperfusion is associated with free radical-mediated injury and may be involved in cardiac apoptosis. The purpose of our study was to investigate (1) if cardioplegia-induced ischemia/reperfusion initiates apoptosis in myocardial endothelial cells and myocytes, and (2) if this is mediated by free radicals.

METHODS: We subjected 7 pigs (56±10 kg) to 1 hour of cold crystalloid cardioplegic arrest (CA) on cardiopulmonary bypass (CPB), and collected 5 transmural LV biopsies: prior to CPB (Baseline), at 60 min CA, at 15 and 30 min reperfusion on CPB, and at 120 min post CPB. Two additional pigs were not subjected to CPB and CA and served as time controls. LV specimen were cut at 15 µm and immuno-cytochemically stained against active caspase-3, the apoptosis signal-pathway key-enzyme, nitrotyrosine as indicator for peroxynitrite (NO₃⁻) -mediated tissue injury, and 8-isoprostane as indicator for oxygen free radical-mediated lipid peroxydation. Specimen were judged using a semi quantitative score from 0 (negativ) to 3 (highly positive).

RESULTS: At 60 min CA, 6 of the 7 hearts showed active caspase-3 but only 2 hearts demonstrated nitrotyrosine formation. The time courses for active caspase-3 and nitrotyrosine are depicted in the figure. At 120 min post CPB, all 7 hearts were positive for caspase-3, nitrotyrosine, and 8-isoprostane, but there were no cell deaths. In contrast, the 2 control hearts remained negative for all variables.

CONCLUSION: Our data show that CA initiates apoptosis signal-pathway in myocardial endothelium and myocytes. As caspase-3 activation preceded both nitrotyrosine and 8-isoprostane formation, CA-induced apoptosis is not mediated by free radicals.

5:30 p.m. ADJOURN - WELCOMING RECEPTION

HALLB

**By Invitation*

MONDAY MORNING, MAY 6, 2002

7:45 a.m. BUSINESS SESSION (Limited to Members)

HALL C

8:00 a.m. SCIENTIFIC SESSION

HALL C

Moderators: Timothy J. Gardner and Tirone E. David

1. Long-Term Outcome of Bilateral Lung Volume Reduction in 250 Consecutive Emphysema Patients

¹A. Ciccone*, B. F. Meyers*, T. J. Guthrie*, G. Davis*, R. Yusen*, S Lefrak*, G. A. Patterson, J. D. Cooper; St. Louis, MO

Discussant: Larry R. Kaiser

OBJECTIVE: Numerous reports have confirmed the early benefit of lung volume reduction surgery(LVRS) for selected emphysema patients. This report documents the long-term functional outcome and survival following LVRS.

METHODS: Between January 1993 and June 2000, 250 consecutive patients underwent bilateral LVRS through median sternotomy at our institution. All patients had disabling dyspnea, thoracic hyperinflation and a heterogeneous pattern of emphysema with suitable target areas for resection. Preoperative pulmonary rehabilitation was required and post rehabilitation data were used as the baseline for data analysis. One patient was lost to follow up and mean follow up is 4.4±1.9 years.

RESULTS: One patient required overnight ventilation following the procedure. Complications included prolonged air leak (>7 days) 44%(n= 110); reintubation and mechanical ventilation 7.2%(n=18); reoperation for air leak or bleeding 7.2%(n=18). Median length of stay was 9(range: 4-168) days. Actuarial survival was 93.6%, 83.7%, and 67.0% at one, three, and five years respectively. Nineteen patients(7.6%) underwent subsequent lung transplantation after a mean interval of 4.23±1.31 years. Health related quality of life showed significant postoperative improvement and over time correlated well with the improvement in FEV1. The table below reports functional results.

	Preoperative	6 Months	1 Year	3 Years	5 Years
N	250	238	235	177	106
FKV1(% predicted)	25.6±7.4	40.0±13.8	37.5±14.1	35.8±17.6	31.7±11.3
RV(liters)	5.8±1.3	4.0±1.2	4.2±1.2	4.2±1.3	4.8±1.3
Six Minute Walk(feet)	1120±214	1335±328	1411±350	1283±429	1278±508
MRC Dyspnea Score	2.8±0.8	1.2±0.9	0.7±0.8	1.7±1.1	2.3±1.0

CONCLUSION: LVRS produces significant functional improvement in selected emphysema patients. For the majority of patients, benefit appears to last at least five years.

¹2001-02 Graham Fellow

*By Invitation

2. Evolving Trends Causes of Death After Heart Transplantation: A 10-Year Multi-Institutional Study

Janies K. Kirklin, David C. Naftel*, Robert C. Bourge*, David C. McGiffn, James A. Hill*, Richard J. Rodeheffer*, Brian E. Jaski*, Paul J. Hauptman*, Mark Weston*, Connie White-Williams*; Birmingham, AL

Discussant: Axel Haverich

OBJECTIVE: As therapeutic options evolve for advanced heart failure, the appropriate role of cardiac transplantation (CTx) will require survival analyses that reflect changing trends in causes of death and patient and institutional risk profiles. Results from multi-institutional studies could be used to monitor progress in individual centers.

METHODS: Between 1990 and 1999, 7,290 patients undergoing CTx in 42 institutions entered a formal outcomes study. Changing survival, causes of death, and patient risk profiles were analyzed. Multivariable risk factor equations were applied to a single institution (300 CTx's) to examine differences in risk-adjusted expected versus actual outcomes over time.

RESULTS: Overall survival improved during the decade ($p=.01$). One and four year survival was: era 1 (1990-1992) 84% and 73%; era 2 (1993-1995) 85% and 76%; and era 3 (1996-1999) 85% and 77%. Causes of death changed over time (figure). Pre-CTx risk profiles increased over time ($p=.0001$); with increases in re-operations, devices, diabetes, severely ill recipients, pulmonary vascular resistance, sensitization, ischemic times, donor age, and donor inotropic support. Four year actuarial survival in a single institution was 8% less than risk adjusted predicted survival in era 1, 2% lower in era 2, and 5% higher than predicted in era 3.

CONCLUSION: 1) CTx survival is gradually improving despite increasing risk profiles. 2) Further improvement requires periodic re-evaluation of risk profiles and causes of death to target areas of surveillance, therapy and research. 3) Using these methods, progress at individual institutions can be assessed in a risk-adjusted manner.

**By Invitation*

3. The Influence of Hemodilution on Outcome after Cardiopulmonary Bypass: Results of a Randomized Trial in Infants

Richard A. Jonas, David Wypij*, Stephen J. Roth*, David C. Bellinger*, Peter C. Laussen*, Jane W. Newburger*; Boston, MA

Discussant: William A. Baumgartner

BACKGROUND: We hypothesized that cognitive decline after cardiopulmonary bypass (CPB) might be exacerbated by hemodilution. In a randomized trial, outcomes were compared after use of two hemodilution protocols during hypothermic CPB in infants free from carotid and cerebrovascular disease.

METHODS: Enrollment criteria included reparative cardiac surgery, age <9 months, birth weight >2.3 kg, and absence of associated disorders. Randomization was stratified by surgeon, type of congenital heart disease, and age.

RESULTS: Among 147 infants enrolled, 74 were assigned to the lower-hematocrit (hot) strategy (21.1%±2.4%, mean±SD during hypothermic bypass) and 73 to the higher-hot strategy (27.7%±3.2%). One infant (<1%) died. Blood product use was similar between groups. The lower hct group had worse perioperative outcomes, including higher serum lactates 60 minutes after CPB (P=.03), lower nadir of cardiac index (P=.02), and greater total body water by bioimpedance on the first postoperative day (P=.006). In 113 children returning at age 1 yr, the lower hot group had worse scores on the Psychomotor Development Index (PDI) (81.9±15.7 vs. 89.7±14.7, P=.008) and more PDI scores %±2SD below population mean (16/56, 29% vs. 5/53, 9%, P=.01). The groups had similar Mental Development Index scores and neurologic examination. Analyses using hot as a continuous variable showed results similar to intent-to-treat analyses.

CONCLUSIONS: Hemodilution to a hot level currently in wide use and thought to be "safe" for CPB is associated with adverse perioperative and developmental outcomes in infants. Future studies should investigate whether hemodilution contributes to the cognitive decline observed in adults after CPB.

**By Invitation*

4. Predictors of Delayed Neurologic Deficit Following Repair of Thoracic and Thoracoabdominal Aortic Aneurysm

Anthony L. Estrera*, Charles C. Miller*, Turn T.T. Huynh*, Ali Azizzadeh*, Eyal E. Porat*, Hazim J. Safi; Houston, TX

Discussant: Lars G. Svensson

OBJECTIVE: Delayed paraplegia or paraparesis (neurologic deficit - DND) has been recognized in recent years as a source of morbidity following thoracic and thoracoabdominal aortic (TAA) repair. We studied risk factors for DND in our experience.

METHODS: We repaired 854 TAA between Feb 1991 and May 2001. We excluded 26 patients who died before post-op neurologic status could be evaluated and 38 who had ND on first post-op evaluation, leaving 790 consecutive patients in this study. We evaluated a wide range of demographic, preoperative and intraoperative data, using univariate and multivariate analyses.

RESULTS: 21/790 (2.6%) patients had DND. Significant univariate predictors

included preoperative renal dysfunction (creatinine = 2 or pre-op dialysis) (odds ratio - O.R. 5.6; p=0.001), acute dissection (O.R. 3.8; p=0.03), extent II aneurysm (O.R. 3.0; p=0.01), and use of adjunct (cerebrospinal fluid drainage and distal aortic perfusion) (O.R. 11.3; p=0.003). Extent II aneurysm dropped out of the multivariate model, but all other terms remained significant. No other risk factors were identified. 12/21 (57%) patients recovered neurologic function with oxygen delivery optimization and CSF decompression.

CONCLUSION: Preoperative renal dysfunction, acute dissection and extent II aneurysm are significant predictors of delayed neurologic deficit; two thirds of which occurred in patients with at least one of these risk factors. Previous studies have demonstrated that adjunct protects against immediate neurologic deficit. The findings of this study are consistent with the hypothesis that adjunct reduces ischemic insult enough to prevent immediate neurologic deficit, but that a period of increased spinal cord vulnerability remains for several days postoperatively.

**By Invitation*

5. Long-Term Results of Left Ventricular Reconditioning and Anatomical Correction for Systemic Right Ventricular Dysfunction Following Atrial Switch Procedures

Nancy C. Poirier*, Jae-Hyeon Yu*, Christian P. Brizard*, Roger B.B. Mee;
Montreal, Quebec, Canada; Melbourne, Australia; Cleveland, OH.

Discussant: Constantine Mavroudis

OBJECTIVE: Systemic right ventricular (sRV) failure following atrial switch procedures for transposition of the great arteries has been addressed with the reconditioning of the morphologically left ventricle (mLV) by banding the pulmonary artery (PAB) followed by an arterial switch operation (ASO) and an atrial resection in 2 institutions (Institution 1: 1981-1993; Institution 2: 1993 -).

METHODS: Between 1981 and 1999, 39 patients (Institution 1: 19, Institution 2: 20) with a mean age of 10.8 years (13 months - 24 years) entered this protocol, a mean of 9 years (5 months - 22 years) following an atrial switch procedure.

RESULTS: The mean duration of PAB was 19 months (1 month - 5.4 years). Nine patients (26%) responded unfavorably to mLV reconditioning (5 mortalities, 4 transplanted or listed for transplantation). Twenty-one of the 26 (81%) patients who underwent ASO and atrial resection survived. During a mean follow-up period of 9.6 years (2 months - 17 years), 4 patients had cardiac-related deaths. All 16 long-term survivors are asymptomatic. At last echocardiographic evaluation, the mLV function was normal in 10 patients (62%), all had normal morphologically right ventricular function with only mild tricuspid regurgitation. Age greater than 12 years was associated with a higher operative mortality ($p = 0.04$) and a greater probability of developing mLV failure ($p = 0.03$).

CONCLUSION: mLV reconditioning and anatomical correction is an alternative to cardiac transplantation for sRV failure following Mustard and Senning procedures, however the response mLV reconditioning in adolescent and young adult population is inconsistent and unpredictable.

9:40 a.m. EDWARD D. CHURCHILL SCHOLAR PRESENTATION

Joseph B. Shrager, Philadelphia, PA

9:45 a.m. EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOW PRESENTATION

Anna Maria Ciccone, Rome, Italy

9:50 a.m. INTERMISSION - VISIT EXHIBITS

HALL B

10:30 a.m. SCIENTIFIC SESSION

HALL C

Moderators: Fred A. Crawford, Jr. and Tirone E. David

6. Off-Pump Coronary Artery Bypass Grafting Provides Complete

Revascularization While Reducing Myocardial Injury, Transfusion

Requirements and Length of Stay: Prospective Randomized Comparison

of 200 Unselected Patients Having OPCAB Versus Conventional CABG

John D. Puskas*, Willis H. Williams, Peggy G. Duke*, James Staples*, John Jeffrey Marshall, Susan A. McCall*, Bonnie H. Summons*, Rebecca J. Petersen*, Dianne K. Bailey*, Elizabeth M. Mahoney*, William S. Weintraub*, Robert A. Guyton; Atlanta, GA

Discussant: Bruce W. Lytle

Objectives: Retrospective comparisons of selected patients having OPCAB versus conventional CABG (CABG/CPB) have yielded inconsistent results and raised concern about completeness of revascularization (COR) in OPCAB.

Methods: 200 unselected patients referred for elective primary CABG were randomized to OPCAB with Octopus™ stabilization or CABG/CPB by a single surgeon. Revascularization intent determined prior to randomization was compared to revascularization performed. All management followed strict, unbiased, criteria-driven protocols. Patients and non-operative care providers were blinded.

Results: Baseline characteristics were similar. Number (#) of grafts performed per patient (OPCAB 3.39±1.04, mean±STD; CABG/CPB 3.40±1.08) and the index of COR (ICOR), #grafts performed/tfgrafts intended (OPCAB 1.00±0.18, CABG/CPB 1.01±0.09), were similar. ICOR was likewise similar between groups for the lateral wall. Combined hospital and 30-day mortality and stroke were similar. Postoperative myocardial serum enzyme measures were significantly lower in OPCAB, suggesting less myocardial injury (Figure).

Adjusted postoperative thromboelastogram indices, fibrinogen, INK and platelet levels all showed significantly less coagulopathy after OPCAB. OPCAB patients received fewer units of blood, were more likely to avoid transfusion altogether, and had higher hematocrit at discharge. CPB was an independent predictor of transfusion (odds ratio 2.42, p=0.0073) by multivariate analysis. More OPCAB patients were extubated in the operating room and within 4 hours. Postoperative LOS was shorter in OPCAB (5.1±6.5 days OPCAB, 6.1±8.2 CABG/CPB, p=0.005 Wilcoxon). One patient (CABG/CPB) required angioplasty for graft closure within 30 days.

Conclusions: Compared to CABG/CPB, OPCAB achieved similar completeness of revascularization, similar in-hospital and 30-day outcomes, shorter LOS, reduced transfusion requirement and less myocardial injury.

**By Invitation*

7. Initial Clinical Experience with the AbioCor Implantable Replacement Heart

Robert D. Dowling, Laman A. Gray, Jr., O.K. Frazier, Steven W. Etoch*, Hillel Laks, Daniel Marelli, Louis Samuels, John Entwistle, Greg Couper, Gus Vlahakes; Louisville, KY; Houston, TX; Los Angeles, CA; Philadelphia, PA; Boston, MA

Discussant: Roland Hetzer, M.D.

Objective: To evaluate the safety and efficacy of the first available totally implantable artificial heart (AbioCor™ IRH) in the treatment of severe, irreversible biventricular heart failure in humans.

Methods: Six male adult patients with severe, irreversible biventricular failure (>80% 30 day predicted mortality) who were not candidates for transplantation met all IRB study criteria and had placement of the AbioCor IRH. All were in cardiogenic shock despite maximal medical therapy including inotropes and IABP. Mean age was 66.7 ± 10.4 (Range 51 to 79). Three of six had prior surgery. Five of six had ischemic and one idiopathic cardiomyopathy. All had 3D computerized simulated implant of the AbioCor that predicted adequate fit. At operation, the internal transcatheter energy transfer coil, battery and controller were placed. Bi-ventriculectomy was then performed and the AbioCor thoracic unit was placed in an orthotopic position and attached to the atrial cuffs and outflow conduits via quick-connects. The AbioCor flow was adjusted to 4-8 L/min. CVP and LAP were maintained at 5-15 mmHg.

The device is powered via transcatheter energy transfer. An atrial flow-balancing chamber is used to adjust L/R balance. The balance chamber and transcatheter energy transfer eliminate the need for percutaneous lines.

Results: There was one intraoperative death due to coagulopathic bleeding. There have been multiple morbidities: 5/5 patients had prolonged intubation, two had hepatic and renal failure (resolved in one), and there was one patient each with recurrent GI bleeding, acute cholecystitis requiring laparotomy, respiratory failure that resolved after three days on ECMO, and malignant hyperthermia (resolved). There were two late deaths: one due to MSOF (POD 56) and one due to a retroperitoneal bleed and resultant MSOF(POD151). This latter patient was not able to tolerate anticoagulation (no anticoagulation or antiplatelet therapy alone 80% of the first 60 days) and developed a TIA on POD 61 and a CVA on POD 130. Before his CVA, he had taken over 20 trips out of the hospital. There have been no other T-E events. There has been no significant hemolysis. The balance chamber has allowed for L/R balance in all patients (LAP within 5 mm Hg RAP). There have been no malfunctions of the AbioCor and no device related infections. The remaining patients are currently extubated and ambulatory. Two patients have taken out of hospital trips. Total days on support with the AbioCor are 369.

Conclusion: The initial clinical experience in gravely ill patients suggests that the AbioCor may be effective therapy in patients with advanced biventricular failure.

11:15a.m. PRESIDENTIAL ADDRESS -

"Our Heritage and Our Future"

Timothy J. Gardner, Philadelphia, PA

Introduced By: Fred A. Crawford, Jr.

Charleston, South Carolina

12:00 p.m. ADJOURN FOR LUNCH - VISIT EXHIBITS

HALL B

**By Invitation*

MONDAY AFTERNOON, MAY 6, 2002

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION - ADULT CARDIAC SURGERY HALL C

*Moderators: D. Craig Miller and
Hartzell V. Schaff*

8. Surgery Versus Stenting in Multivessel Disease: 3 Years Results in A

Randomized Study

Felix Unger, Patrick Serruys*, MC. M. Vrolix*, G. Fransen*, P. Materne*, G. Dekoster*, R. Seabra-Gomes*, J. Q. Melo; Salzburg, Austria; Rotterdam, The Netherlands; Dienst Hartcatheterisatie and Liege, Belgium; Carnaxide,

Portugal

*Discussant: Kenneth M. Kent**

OBJECTIVE: The recent appreciation that Stenting has improved the short and long-term outcome of patients treated with coronary angioplasty has made it imperative to reconsider the comparison between surgery and percutaneous interventions in patients with multivessel disease.

METHODS: Twelve hundred and five patients were randomly assigned to bypass surgery or stent implantation when there was consensus between the cardiac surgeon and interventional cardiologist on equivalent 'treatability'. The primary clinical endpoint was freedom from major adverse cardiac and cerebrovascular events at one year. Major adverse cardiac and cerebrovascular events at 3 years constitute a secondary endpoint.

RESULTS: At three years 88.2% of the surgical and 87.2% of the stented patients were free of death, stroke and myocardial infarction ($P=0.813$). Among patients who survived without stroke or myocardial infarction, 21.3% underwent a second revascularisation in the stent group, as compared with 5.1% in the surgical group. At three years 83.6% of the surgical and 65.7% of the stented patients were event-free survivors ($P<0.001$). At three years 87% in the surgical cohort and 81.6% in the stent group were angina free. In patients with diabetes 81.3% in the surgical group and 52.7% in the stented patients were free of any events after two years ($P<0.001$).

CONCLUSION: The difference in outcome between surgery and Stenting observed at one year in patients with multivessel disease remains essentially unchanged at two years. Stenting is associated with a greater need for repeat revascularisation. In view of the relatively greater difference in outcome in diabetics, surgery seems to be the preferred treatment in these patients.

**By Invitation*

9. Partial Cardiac Denervation Reduces the Incidence of Atrial Fibrillation After Coronary Revascularization

Joao Melo, Manuel M. Ferreira*, Miguel Abecassis*, Maria Rebocho*, Ana Timoteo*, Bingur Sonmez*, Selim Tansal*, Harun Arbatli*, Peter Voigt*, Robert Dion; Carnaxide, Portugal; Istanbul, Turkey, The Netherlands

Discussant: Ralph J. Damiano, Jr

OBJECTIVE: Because the autonomic nervous system is an important determinant for the appearance of atrial fibrillation after coronary artery bypass surgery, we have assessed the role of partial cardiac denervation , for its prevention.

METHODS: In a 3 month period, 416 low risk coronary artery bypass surgery patients, were enrolled. No routine antiarrhythmic drugs were administered before or after surgery. Ventral cardiac denervation was performed in 207 patients (group A) and 219 were used as controls (group B). In one institution 100 patients were prospectively randomized. Denervation was performed before cardiopulmonary bypass started. The groups were comparable regarding age (60 vs 62 years), gender (female, 24% versus 25%), presence of diabetes (26% vs 27%).

RESULTS: The additional length of time required for the denervation was on average 5 ± 2 minutes, and there were no complications associated with the procedure. Cardiopulmonary bypass duration (95 vs and 99 minutes), myocardial ischemic time (50 vs 53 minutes), number of grafts performed (3.2 vs 2.9), post operative bleeding (946 vs 941 ml), were similar in both groups. Postoperative atrial fibrillation was present in 15 patients from group A (7% - CL 95 - 4-12%) and in 56 patients from group B (27 % . CL95 -18-35 %).

CONCLUSION: Ventral cardiac denervation is a fast and low risk procedure. Its use reduces significantly the incidence of atrial fibrillation after routine coronary artery bypass surgery

**By Invitation*

10. Early Results of Stent-Graft Treatment of DeBakey Type III Aortic Dissection

Kiyotaka Imoto*, Shin-ichi Suzuki*, Keiji Uchida*, Susumu Isoda*, Yoshihiro Iwai*, Naoki Hshiyama*, Takayuki Kosuge*. Yoshihisa Karube*. Hiromasa Yanagi*, Hiroshi Tamagawa*, Iitoshi Inari*, Jiro Kondo*; Yokohama, Japan
Discussant: D. Craig Miller

OBJECTIVE: To assess the safety and effectiveness of transcatheter stem-graft treatment in patients with DeBakey type III aortic dissection.

METHODS: Transcatheter stem-graft implantation was performed in 32 patients who had aortic dissection with entry sites in the descending thoracic aorta. Entry sites were closed with a stent-graft consisting of a Z stent covered with an UBE woven Dacron graft. Early postoperative results were analyzed.

RESULTS: There were no in-hospital deaths_@or serious complications after operation. In 5 patients, a new intimal tear developed at the distal end of the stent-graft 8 to 13 months after operation. It was closed by additional stent-graft placement in 2 patients. The rate of thrombosis of the descending thoracic aorta false lumen 1 week, 3 to 6 months, and 12 months after operation was 69% (22/32), 81% (26/32), and 81% (26/32), respectively. As compared with before operation, the short axis of the true lumen increased and that of the false lumen decreased, indicating enlargement of the true lumen and shrinkage of the false lumen. There was no significant change in aortic diameter 3 to 6 months after operation. In all 15 patients in whom entry sites were closed within 2 years after onset, the false lumen shrank 3 to 6 months after operation.

Changes in diameter of true lumen, false lumen, and aorta

	PreOp	5-10 d	3-6 m
True lumen	1.5±0.6	2.3±0.5*	2.8±0.5*

False lumen	2.5±0.8	1.9±0.7*	1.2±0.9*
Aorta	3.9±0.6	4.1±0.5	4.0±0.6

* P < 0.001 compared with PreOp, (cm)

CONCLUSION: Transcatheter stent-graft implantation is a safe and effective procedure for the management of DeBakey Type III @aortic dissection. Devices with a minimal risk of causing intimal tears should be developed.

**By Invitation*

11. Results of Surgery for Aortic Root Aneurysm in Patients with Marfan Syndrome

Nilto Carias de Oliveira*, Tirone E. David, Joan Ivanov*, Susan Armstrong*, Gary Webb*; Toronto, Ontario, Canada

Discussant: Magdi Yacoub

OBJECTIVE: To evaluate the results of surgery for aortic root aneurysm (ARA) in patients with Marfan syndrome.

METHODS: 102 consecutive patients with Marfan syndrome (Gent criteria) had surgery for ARA. There were 78 men and 24 women with a mean age of 36±12 years. Fifteen patients had acute and 7 had chronic type A aortic dissection. Previous cardiac operations had been performed in 14 patients, including aortic valve replacement in 8. Four patients had active infective endocarditis. Aortic root replacement (Bentall) was performed in 43 patients and aortic valve sparing operations (Sparing) in 59- In addition, mitral valve repair was performed in 14 patients and replacement in 4. Follow-up was complete at 4.3±4.0 years.

RESULTS: Patients who had Bentall had higher functional class, larger ARA (59±11mm vs. 51±0.5 mm, p=0.03) and more severe aortic insufficiency, (AI grade 2.8±0.9 vs. 1.6±0.5, p=0.01). The severity of AI correlated well with the diameter of the ARA (r=0.95). There was one operative death in a patient with end-stage AI. There were 6 late deaths, all cardiovascular-related. The 10-year survival was 90±5% for all patients; it was 86±7% for Bentall and 97±3% for Sparing (p=0.08). Patients who had Bentall had a higher rate of valve related mortality and morbidity than those who had Sparing (p=0.001).

CONCLUSION: Although the clinical profile of patients who had Bentall was different from those who had Sparing, the long-term outcomes appear to be better after Sparing. To increase the probability of valve sparing, surgery should be performed before the aortic root exceeds 50 mm in diameter.

**By Invitation*

12. Safety and Usefulness of Composite Grafts for Total Arterial Myocardial Revascularization: A Perspective Randomized Evaluation

Claudio Muneretto*, Alberto Negri*, Jacopo Manfredi*, Alberto Terrini*, Giulia Rodella", Gianluigi Bisleri*, Suad Elqarra*, Adriano Decarli*; Brescia, Italy

Discussant: ¹Thoralf M. Sundt

OBJECTIVE: In order to evaluate the results of total arterial revascularization with composite grafts compared to conventional CABG surgery, we enrolled 200 consecutive pts. undergoing myocardial revascularization.

METHODS: Pts. were random assigned to one of the two groups (100 units each): Group I (GI) for full arterial revascularization; Group II (GII), LITA on IAD plus saphenous grafts (SG). The groups were comparable in terms of age(mean:GI=67±9 vs GII=68±8 yrs.), gender and pre-operative risk factors (mean Euroscore : GI=4 vs GII=4,2). The arterial grafts in GI were :100 LITA, 40 RITA and 48 Radial Artery (RA) in composite grafts.

RESULTS: There were no differences between the groups in terms of number of grafted vessels (mean: GI=2,8 vs GII=2,9), cross clamping (mean: GI=38±7 vs GII=40±6 min), ventilation time (mean: GI=9±6 vs GII=11±7 hrs.), ICU stay (mean: GI=25±8 vs GII=24±7 hrs.) and post-operative complications (such as bleeding, respiratory insufficiency and sternal dehiscence). Significant differences were found in mean CPB time (GI=56±9 vs GII=81±7 min). Hospital mortality was 1% in both groups. Recurrence of angina was noted in 2 pts. in GI and in 13 pts. in Gil. Angiograms in symptomatic pts. or at random were performed at 12 months in 72% of pts. in GI and in 68% in Gil: graft patency was 99% in GI and 89% SG in GII.

CONCLUSION: Total arterial myocardial revascularization with composite grafts provided superior clinical results and significantly higher graft patency rate when compared to the standard technique.

3:20 p.m. INTERMISSION - VISIT EXHIBITS

HALL B

¹1994-96 AATS Research Scholar

**By*

Invitation

MONDAY AFTERNOON, MAY 6, 2002

4:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION - ADULT CARDIAC SURGERY HALL C

Moderators: D. Craig Miller and Hartzell V. Schaff

13. CABG in Cardiogenic Shock or Cardiopulmonary Resuscitation, Is This

Appropriate Use of Resources?

Paul Sergeant, Bart Meyns*, Patrick Wouters*, Roland Demeyere*, Peter Lauwers*; Leuven, Belgium

Discussant: Ludwig K. von Segesser

OBJECTIVE: CABG in patients in cardiogenic shock and cardiopulmonary resuscitation (CPR) is a rarely performed procedure, definitely at risk in health care environments where cost reduction and closed annual budgeting are the obsession. The resource consumption might not be balanced by an acceptable late benefit.

METHODS: A consecutive series of patients undergoing CABG in cardiogenic shock (N=167) and in CPR (N=92) from 08/1979 till 08/2001 in a single institution were studied using time-related

and multivariate methodologies. A "common closing date" follow up methodology was used. The age of the patients was 62±10 years (range 30-90). The events leading to the preop-erative condition were a recent catheterisation (N=52), a recent CABG (N=24), a recent PTCA (N=85), an infarct at home (N=45), an infarct in hospital (N=42) and an infarct after a recent infarct(N=11).

RESULTS: The 1-, 10- and 15- year survival was 60±3, 49±3 and 42±4 % respectively. The peri-procedural hazard of death extends for 50 days in patients in cardiogenic shock and 100 days for patients in CPR. Beyond that timeframe and up to 15 years, a normal hazard of late death is observed. Multivariate analysis, after stratification for shock and CPR, identified an increased risk in the presence of additional co-morbidity (EuroSCORE) and treated diabetes. The absence of 3-vessel disease decreased the risk. The 10-, 20- and 30-day freedom from hospital discharge alive was 84±3, 42±4 and 27±4% respectively. The 8-day freedom from decerebration or stroke was 94±1%. An IABP was present or inserted in 40% of the patients. The 8-day freedom from mechanical uni- or biventricular support was 87±2%. The 10-year freedom from cardio-surgical re-intervention was 90±3%.

CONCLUSION: CABG in cardiogenic shock and CPR does indeed consume considerable resources, has an extremely high and protracted peri-procedural risk but is balanced by a normal late survival. It should be attempted in patients where comorbidity will permit late survival.

**By Invitation*

14. The Cox-Maze Operation for Atrial Fibrillation: Long-Term Efficacy in Patients Undergoing Lone versus Concomitant Procedures

Ralph J. Damiano, Jr., Sunil M. Prasad*, Hersh S. Maniar*, Cindy J. Camillo*, Richard B. Schuessler*, John P. Boineau*, §Thoralf M. Sundt, James L. Cox; St. Louis, MO

Discussant: Hartzell V. Schaff

OBJECTIVE: For the last decade, the Cox-MAZE procedure has been available for the treatment of atrial fibrillation (AF). It is unknown whether the operation has similar efficacy in patients with lone AF as compared to AF associated with coronary, valve, or congenital heart disease. This study examined the long-term outcome of patients who underwent this procedure either as a lone operation (LM) or as a concomitant procedure (CM).

METHODS: From 1987 to 2001, 237 consecutive patients underwent a Cox-MAZE procedure; 145 were LM and 92 were CM. The CM group included 39 coronary artery bypass, 32 valve, and 21 congenital repair procedures. Major complications included renal failure, reoperation for bleeding, mediastinitis, neurologic events, and balloon pump insertion. Follow-up was performed by mail and telephone questionnaires.

RESULTS: The LM group was significantly younger, and had a higher male/female ratio. There was no difference in the length of stay (LOS) or operative mortality between groups. At a mean follow-up of 7±2 years, 95% (91/96) of patients were not in AF, free from antiarrhythmic medication, and not taking coumadin. There was no difference in long-term outcome between the two groups. There have been no late strokes and only 1 patient has had a transient ischemic attack.

	LM	CM	p-value
N	145	92	

Age	49.9±11.5	57.8±10.6	< 0.001
M:F	115:30	59:33	0.015
Chronic AF	40% (58/145)	50% (46/92)	NS
Operative Mortality'	1.3% (2/145)	3.2% (3/92)	NS
ICU (median)	2 days	3 days	0.002
LOS (median)	10	10	NS
Major Complications	15.1% (22/145)	14.1% (13/92)	NS
Pacemakers	26.9% (39/145)	37.0% (34/92)	NS

CONCLUSION: The Cox-MAZE procedure has equivalent operative risk and excellent long-term efficacy in patients undergoing both LM and CM. The operation eliminates the risk of stroke in these high-risk patients. The Cox-MAZE procedure remains the gold standard against which all less extensive procedures must be judged.

§1994-96 AATS Research Scholar

*By Invitation

15. The Impact of Residual Mitral Regurgitation on Survival after CABG and Annuloplasty for Ischemic Mitral Regurgitation

†Lishan Aklog, Edward G. Soltész*, Farzan Filsoouf*, Jerome Sepic*,
Lawrence H. Cohn, Luigino Nascimben, #David H. Adams: Boston, MA

Discussant: Irving L. Kron

OBJECTIVE: To determine the impact of residual mitral regurgitation (RMR) on survival after combined CABG/mitral valve annuloplasty (MVA) and the potential importance of annuloplasty technique (partial restrictive versus complete remodeling).

METHODS: 288 pts (168/288 [58%] male, median age 73 years, 167/288 [58%] NYHA Class III-IV, median ejection fraction 35%) with Carpentier type I or IIIb ischemic MR (259/288[90%] 3+ or 4+/4+ MR) underwent primary CABG/MVA. A complete remodeling, semi-rigid ring was used in 92 patients and a partial, restrictive, flexible band in 196. RMR was assessed by echocardiography within 6 weeks of surgery. Mean follow-up was 28 months. Predictors of RMR and late mortality were determined in patients with RMR \geq 2+ (n=55) compared with RMR 0-1+ (n=233).

RESULTS: Pre-operative characteristics and 30-day mortality were similar within the two groups. Univariable predictors of RMR \geq 2+ included preoperative 4+ MR and pulmonary hypertension (OR's, 2.0 and 2.7, respectively, all p<0.05). Univariable predictors of late mortality included RMR \geq 2+, concomitant tricuspid repair and renal failure (OR's, 2.5,4.4, 2.2, respectively). In the multivariable model, RMR \geq 2+ remained an independent predictor of late mortality (adjusted OR 2.3, 95%CI[1.3-4.0]). Kaplan-Meier survival was lower in those with RMR \geq 2+(p=0.0007). Patients receiving a partial, flexible band tended to have RMR \geq 2+ rather than 0-1+(41/55[75%] vs. 151/233[65%], p=0.11). Complete resolution (0 RMR) was more likely in the complete, semi-rigid ring group (55/92[60%] vs. 74/196[38%], p=0.001).

CONCLUSION: RMR predicts late mortality in patients undergoing surgery for ischemic MR. A complete remodeling semi-rigid ring annuloplasty may reduce the degree of RMR.

†1998-99 *International Traveling Fellow*

§1992-94 *Research Scholar*

**By Invitation*

16. Early and Late Stroke after Mitral Valve Replacement with Mechanical Prostheses: Risk Factor Analysis of a 24-year Experience

²Ko Bando*, Junjiro Kobayashi*, Mitsuhiro Hirata*, Yoshikado Sasako*, Toshihiko Sato*, Kazuo Niwaya*, Osamu Tagusari*, Takeshi Nakatani*, Toshikatsu Yagihara*, Soichiro Kitamura; Osaka and Kanagawa, Japan

Discussant: Gary W. Akins

OBJECTIVE: We evaluated risk factors for mortality and stroke following mechanical mitral valve replacement (MVR) between 7/77 and 6/01.

METHODS: Early and late mortality and stroke were assessed. Potential predictors of mortality and stroke were entered into a Cox proportional hazards model. Actuarial survival and freedom from stroke were determined by a log rank test.

RESULTS: MVR was performed in 822 patients (pts). Concomitant procedures included left atrial (LA) appendage closure in 459, tricuspid annuloplasty/replacement in 396, Maze procedure in 171, plication of LA in 123, and others in 43. Five year actuarial survival was 91%. Only recurrent atrial fibrillation (AF) predicted early stroke (OR: 1.97, 95% CL: 1.08-3.60, p=0.02). Freedom from stroke at 5 years was significantly better in pts with sinus rhythm versus AF (98% vs 86%, p<0.0001). Sixty-four pts had late stroke; 62 pts (97%) were in AF and 50 pts (78%) had the LA appendage closed. At the time of their stroke, 55 pts (86%) had an INR>2.3. Nine pts (14%) had stopped warfarin for minor surgery or dental care. Multivariable analysis showed the addition of a Maze procedure was the only predictor of freedom from late stroke (OR: 1.98, 95% CL: 1.06-3.70, p=0.03).

CONCLUSION: Persistent AF was the most significant risk factor for early and late stroke after mechanical MVR. Restoration of sinus rhythm with a Maze procedure nearly eliminated the risk of late stroke, while neither closure of LA appendage nor therapeutic anticoagulation prevented this complication.

²1991-92 *Graham Fellow*

**By Invitation*

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

ROOM 20

Moderators: Carolyn E. Reed and Thomas W. Rice

17. Accelerated Induction Chemoradiotherapy Provides Enhanced Survival in Both Stage IMA and 1MB Non-small Cell Lung Cancer

Malcolm M. DeCamp*, Thomas W. Rice, David J. Adelstein*, Mark A. Chidel*, Lisa A. Rybicki*, Sudish C. Murthy*, Eugene H. Blackstone; Cleveland, OH

Discussant: Robert J. Ginsberg

OBJECTIVE: Neoadjuvant therapy followed by resection is acceptable care for stage IIIA, N2 non-small cell lung cancer (NSCLC), yet remains controversial for stage IIIB, N3 patients. Failure to sterilize mediastinal nodal disease with induction regimen has been associated with dismal outcome. We sought to explore the safety, feasibility, and efficacy of accelerated Chemoradiotherapy and surgery in patients with both N2 and N3 disease.

METHODS: Between 10/94 and 9/00, 105 patients (58% male, mean age 57±10) with stage IIIA (n=78) or IIIB (n=27) NSCLC were enrolled in a study of hyperfractionated radiotherapy with concurrent chemotherapy (paclitaxel/cisplatin) followed by resection and consolidative chemoradiation. Correlates of downstaging and survival were assessed.

RESULTS: 98 patients (93%) were operable and 83 (79%) resectable (lobectomy 47, pneumonectomy 36). Treatment-related and operative mortality was 8.6% and 7%, respectively. Median, two-, and five-year survival was 27 months, 54%, and 32%, respectively. Final nodal status predicted superior survival for downstaged (N0, N1) patients (n=35) versus those with residual N2 (n=44) versus N3 (n=20) disease (P<0.001; Figure). Sterilization of mediastinal nodes was equally likely in patients with initial N2 disease (27 of 78, 35%) as in N3 patients (8 of 27, 29%).

CONCLUSION: Hyperfractionated radiotherapy with concurrent chemotherapy followed by resection is feasible and safe for selected patients with both stage IIIA and IIIB NSCLC. Patients with N2 and N3 disease benefit similarly. Although nodal downstaging predicts the best outcome, 30% of patients with residual N2 disease at time of surgery experience long-term survival.

**By Invitation*

18. Single Station Metastasis Assures Good Survival for Patients with Stage IMA Lung Cancer Even Accompanied by Persistent N2 Disease After Induction Therapy

Noriyoshi Sawabata*, Akihide Matsumura*, Yoshihiko Osaka*, Shima Fukai*, Takashi Mori*, Osamu Kawashima*; Toyonaka and Osaka, Japan

*Discussant: Steven M. Keller**

OBJECTIVE: This study was undertaken to determine the predictive value of mediastinal nodal status at resection among patients with stage IIIA non-small cell lung cancer (NSCLC) accompanied by persistent N2 disease.

METHODS: We reviewed the medical records of patients with p-N2- NSCLC who underwent induction therapy followed by surgery between 1988 and 1999 at 11 Japanese national referral hospitals. As dichotomous parameters, single station N2, complete resection, chemo alone, radiographic responder, adenocarcinoma and T status more than 2 were indicated.

RESULTS: There were 42 patients (32 men) who had persistent N2 (17 single station and 25 multiple) with 36 complete resections, 20 adenocarcinomas, 17 chemo alone, 21 radiographic responder and 31 p-T1 or T2. The single station N2 group had a 46 months mean survival time (MST) and a 44.8% 5-year survival rate (5-YSR) as compared to a 12 months and a 9.9% for the multiple group (p=0.02). Moreover, complete resection group (a 16 months MST and a 28% 5-YSR vs. a 9 months MST and a 0% 5-YSR, p=0.01) and radiographic responder (a 35 months MST and a 46% 5-YSR vs. a 11 months MST and a 5% 5-YSR, p=0.01) achieved superior actuarial survival, respectively. However, in multivariable analysis, these three parameters were not statistically significant but trend factors. In a subset analysis, patients who had every single station N2, radiographic responder, and complete resection (n=12) achieved better survival than those with each multiple N2, non-responder, or incomplete resection (n=30) (a 78% 5-YSR vs. an 11% 5-YSR, p=0.002)

CONCLUSION: Patients with single station N2-positive NSCLC following induction therapy could enjoy good survival after surgery even if there was persistent N2 disease in combination with radiographic response and complete resection.

**By Invitation*

19. Laryngotracheal Resection as an Effective Definitive Surgical Treatment for Idiopathic Laryngotracheal Stenosis

Simon K. Ashiku*, Akin Kuzucu*, Hermes C. Grille, Cameron D. Wright, John C. Wain, Douglas J. Mathisen; Boston, MA

Discussant: F. Griffith Pearson

OBJECTIVE: Little was known about ILTS when first described. We have now operated upon 75 patients and have confirmed its mode of presentation, response to surgical therapy and have at last established long-term follow-up.

METHODS: Between 1971 and 2001, 75 patients were treated at our institution for ILTS. Their charts were retrospectively reviewed to abstract demographic and clinicopathologic data.

RESULTS: All patients were treated by a single-staged laryngotracheal resection, (35/75) with and (45/75) without a posterior membranous wall flap. The vast majority were women (III 75) with a mean age of 33 (range 13 - 74). 26/75 (%) had undergone a previous procedure with laser (18), dilation (8), laryngeal or tracheostomy procedures (14). Following laryngotracheal resection, the majority of patients (66/75) were able to be extubated in the OR and 9 required temporary tracheostomies, only 1 in the last 30 cases. All but one was successfully decannulated. There was no perioperative mortality. The major morbidity was alteration in voice quality, which improved with time. 67/75 (89%) patients required no further intervention for their ILTS. Eight patients required at least one bronchoscopy for granulation tissue excision and/or dilatation. Only one patient required a permanent tracheostomy.

CONCLUSION: ILTS is an entity that occurs almost exclusively in women without a known cause. Timing of operation is crucial. A single-staged laryngotracheal resection is successful in over 90% of patients. Protecting tracheostomy is now rarely required (1/30). Long-term follow-up now suggest a stable outcome and improvement in voice quality.

**By Invitation*

20. Surgical Management of Sternoclavicular Joint Infections

Harold M. Burkhart*, Claude Deschamps, Mark S. Allen, Daniel L. Miller,
Francis C. Nichols*, Peter C. Pairolero; Rochester, MN

Discussant: Malcolm M. DeCamp, Jr

OBJECTIVE: Sternoclavicular joint infection is rare and its management is controversial. We reviewed our experience with the surgical management of this condition.

METHODS: From August 1988 to August 2001, 26 patients (16 men and 10 women) were treated surgically for infected Sternoclavicular joints. Median age was 56 years (range, 20-77). Patients who had a previous median sternotomy were excluded.

RESULTS: All patients were symptomatic. Chest wall pain was present in 21 patients, swelling in 14, fever in 11, and erythema in 9. Twelve patients had recent or ongoing infections in other areas (pneumonia in 4, multiple joint infections in 2, other in 6). One patient had an ipsilateral indwelling central venous catheter. Five patients had a history of trauma in the region of the joint. Four patients had prior joint incision and debridement. Unilateral Sternoclavicular joint resection was performed in 18 patients, incision and debridement in 6, and bilateral Sternoclavicular resections in 2. Wound cultures were positive in 24 patients and the most common organism isolated was *Staphylococcus aureus* (n=17). Eleven patients required chest wall reconstruction with an ipsilateral pectoralis major muscle flap. Two patients (7.7%) developed complications and one died (operative mortality 3.8%). Cause of death was sepsis. Follow-up was complete in all 25 operative survivors and ranged from 2 months to 10 years (median, 25 months). Twenty-one patients are alive without infection or limitations in range of motion. Four patients have died secondary to causes unrelated to their joint infections.

CONCLUSION: Sternoclavicular joint infections require surgical intervention. Aggressive surgical debridement or resection combined with a muscle transposition are key to successful management.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

HALL B

**By Invitation*

3:50 p.m. SIMULTANEOUS SCIENTIFIC SESSION -GENERAL THORACIC SURGERY

ROOM 20

Moderators: Carolyn E. Reed and Thomas W. Rice

21. Extracapsular Lymphnode Involvement is a Negative Prognostic Factor in T3

Adenocarcinoma of the Distal Esophagus and Gastroesophageal junction

T. Lerut, W. Coosemans*, P. De Leyn*, N. Ectors*, J. Moons, P. Nafteux*, D. Van Raemdonck*; Leuven, Belgium

Discussant: Mark J. Krasna

OBJECTIVE: To assess prognosis according to whether LN involvement is intracapsular (I.C.) or with extracapsular breakthrough (E.G.) in adenocarcinoma.

MATERIALS AND METHODS: 195 consecutive patients with T₃ adenocarcinoma of the distal esophagus and G.E.J. between 1990 -1999 were studied. All patients underwent primary R₀ esophagectomy. The mean number of resected nodes per patient was 36,9. Survival was analysed according to I.C. and E.C. involvement and number of positive nodes.

RESULTS: 5 year survival results are shown in Table I.

	n	5 year survival (%)
pN0	33	57
I.C. 1-6 pos. nodes	56	45,1
I.C. > 6 pos. nodes	4	0
E.C. 1-6 pos. nodes	55	17,1
E.C. > 6 pos. nodes	47	20

There was no significant difference in survival between N₀ versus 1-6 I.C. (p=0,64), I.C. > 6 and E.C. 1 - 6 (p=0,71), I.C. > 6 and E.C. > 6 (p=0,65) and E.C. 1 - 6 and E.C. > 6 (p=0,08).

However there was a significant difference in survival between N₀ and E.C. 1- 6 (p=0,002), N₀ and E.C. > 6 (p=0,0001) and I.C. 1 - 6 and E.C. 1 - 6 (p=0,02). In the latter subset the mean number of positive nodes was 3,5 for I.C. and 3,6 for E.C. (p=0,07 NS).

CONCLUSIONS: This study shows a significant difference in survival curves according to whether lymphnode involvement was intracapsular or extracapsular. Patients with 6 or less intracapsular lymphnodes involved have similar survival rates as N₀ patients. Extracapsular lymphnode involvement irrespective the number of involved lymphnodes is a bad prognostic factor. These data may have an impact on therapeutic strategies.

**By Invitation*

22. The Natural History of Bronchiolitis Obliterans Syndrome is Predicted by Early (3-Months) Lung Allograft Function

Renzo Pessotto*, Heyman Luckraz*, Keith McNeil*, Susan Charman*, John Wallwork*; Cambridge, United Kingdom *Discussant: Duane R. Davis, Jr.*

OBJECTIVE: The development of bronchiolitis obliterans syndrome (BOS) hampers the long-term success of lung transplantation. Identification of early clinical predictors of BOS could influence the treatment strategy and improve long-term survival.

METHODS: Retrospective analysis of all 279 double and heart lung transplant recipients performed between April 1984 and July 2001 and surviving over 3-months post transplantation. Patients were categorized according to their percent predicted FEV1 at 3 months: Group 1 (n=78) = FEV1 <60% predicted, Group 2 (n=89) = FEV1 = 60-80% predicted and Group 3 (n=112) = FEV1 >80% predicted.

RESULTS: Actuarial freedom (95% CI) from BOS at 3 years was 43 (28,55)% in group 1, 49 (37,60) % in group 2 and 62 (53,72) % in group 3. Overall freedom from BOS was significantly different across the three groups ($p=0.01$). In those who developed BOS, the median (IQR) time for BOS development was 1.3 (0.7, 1.9), 2.2 (1.2,3.4) and 2.7 (1.2,4.7) years from transplant, respectively ($p <0.001$ for overall). The actuarial 3-year survival from transplantation was 60 (44,77)%, 81 (70,90)% and 84 (76,92)% for recipients who developed BOS in groups 1,2 and 3 respectively ($p <0.001$ for overall). The 2-year post-BOS actuarial survival was 46 (29,63)%, 67 (54,80)% and 61 (49,74)% ($p=0.06$).

CONCLUSION: The 3-month, post transplant, percent predicted FEV1 is an early clinical marker that highlights an increased risk of developing BOS. It allows early identification of high-risk patients (FEV1 $<60\%$) who could benefit from changes to an anti-proliferative immuno-suppression strategy.

**By Invitation*

23. Improved Lung Allograft Function Following Fundoplication in Lung Transplant Recipients with GERD

Duane R. Davis, Jr.* , Christine L. Lau* , W. Steve Eubanks* , Theodore N. Pappas* , Dennis Hadjiliadis* , Scott M. Palmer* ; Durham, NC

Discussant: G. Alexander Patterson

OBJECTIVE: Bronchiolitis obliterans is the greatest limitation to the long-term applicability of lung transplantation. While allo-immune events are important, non-immune events such as gastroesophageal reflux may contribute to lung injury and the development of bronchiolitis obliterans syndrome (BOS).

METHODS: We prospectively studied 108 lung transplant recipients with esophageal pH probe and manometry. Patients who subsequently went on to have fundoplication were evaluated for changes in spirometry and survival.

RESULTS: Abnormal pH studies (total acid contact time $>5\%$) were present in 70 patients (65%). Thirty-four patients subsequently had fundoplication. Twenty-one of the patients met criteria for BOS. There were no in-hospital or 30-day mortality in the patients undergoing fundoplication. There have been 8 late deaths. Twelve of the 21 patients with BOS had an improvement in their BOS grade. In the 11 patients in BOS-1, there have been 3 late deaths and 7 patients became BOS-0. In the 6 patients who were BOS-2, there was 1 late death, 2 improved to BOS-1 and 2 to BOS-0. In the 4 patients who were BOS-3, there have been 2 late deaths and one patient improved to BOS-2. Overall, in 25 patients who were at least 6 months post lung transplant and post fundoplication, the FEV1 improved by an average of 27% (404ml/sec, pre FEV1 mean 1.85L/sec, postFEV1 2.25 L/sec, $p<0.0001$).

CONCLUSION: GERD is common after lung transplantation. Fundoplication in lung transplant recipients with GERD is associated with significant improvements in lung particularly if performed prior to the late stages of BOS.

**By Invitation*

24. Results of Superior Vena Cava Resection for Non-Small Cell Lung Cancer: Factors Influencing Long-Term Survival

Lorenzo Spaggiari*, Pierre Magdeleinat*, Haruhiko Kondo*, Pascal Thomas*, Maria Elena Leon*, Cathy Ratcliffe*, Gilles Rollet*, Jean Francois Regnard*, Ryosuke Tsuchiya*, Ugo Pastorino*; Milan, Italy; Paris, France; Tokyo, Japan; Marseilles, France

*Discussant: Erino Rendina**

OBJECTIVE: The benefits of Superior Vena Cava (SVC) resection for lung cancer remain controversial. Data from four international centers were analyzed in order to identify prognostic factors and guide future patient's selection.

METHODS: Retrospective study. Prognostic factors examined by logistic regression for postoperative morbidity/mortality, and by Kaplan Meier method (log rank test) and Cox proportional-hazards modeling for survival.

RESULTS: From 1963 to 2000, 109 patients underwent SVC resection. Induction treatment was given to 23 (21%) patients. SVC was resected for T and N involvement in 78 (72%) and 31 (28%) cases respectively. Fifty-five patients (50%) underwent pneumonectomy (20 with carinal resection), whereas the remaining underwent lobar resections. Prosthetic SVC replacement was performed in 28 patients (26%); partial resection with running suture (53%), vascular stapler (13%), or patch (7%) was performed in 80 patients; one did not undergo reconstruction. Pathological examination identified direct involvement (T4) and N2 disease in 66 (60%) and 55 (50%) patients respectively. Major postoperative morbidity and mortality were 30% and 12%. Median intensive care unit and hospital stay were 3 and 16 days. Actuarial 5-year survival was 21% and median survival 11 months. At multivariate analysis, induction treatment showed a significant impact on major postoperative complications ($p=0.016$), whereas pneumonectomy (0.017) and year of surgery ($p=0.049$) on survival. Prosthetic replacement appeared of borderline significance ($p=0.068$).

CONCLUSION: Radical resection of lung cancer involving SVC may achieve permanent cure in carefully selected patients. Best candidates are those requiring lobectomy and partial SVC resection without prosthetic replacement.

**By Invitation*

25. Positron Emission Tomography Defines Metastatic Disease But Not Locoregional Disease In Patients Undergoing Resection For Malignant Pleural Mesothelioma

Raja M. Flores*, Timothy Akhurst*, Valerie W. Rusch; New York, NY

Discussant: David J. Sugarbaker

OBJECTIVE: Computed tomography (CT) and magnetic resonance imaging (MRI) often fail to predict resectability in patients with MPM. Small studies suggest that PET may improve preoperative staging. Therefore, we studied this prospectively.

METHODS: All patients fasted and received a minimum of 10 mCi of F18-fluorodeoxyglucose. Whole-body emission studies were acquired, followed by whole-body transmission studies with iterative reconstructions. Patients were clinically staged based on blinded PET readings and CT using the IMIG mesothelioma staging system. Spearman's correlation coefficients were calculated

based upon PET scan and surgical pathological evaluation, a p-value of 0.05 was considered statistically significant.

RESULTS: From 1998 to 2001, 44 patients with MPM underwent PET scans. All 44 patients had FDG-18 uptake in the primary tumor, standardized uptake value (SUV) range 2-23. Surgical-pathological comparison was available in 35 patients for at least one of the T, N, and M variables. T status, N status, and SUV did not correlate with resectability or final pathological evaluation. Blinded PET readings of nodal status demonstrated a sensitivity of 25% and a specificity of 72%. However, PET was useful in identifying patients with N3 disease (supraclavicular adenopathy) and distant metastasis that were not identified by computed tomography. Stage IV disease was identified in 6 patients.

CONCLUSION: PET does not define the local extent of tumor or regional nodal involvement reliably, but is useful in detecting distant metastasis, and therefore may obviate inappropriate exploratory thoracotomy.

**By Invitation*

MONDAY AFTERNOON, MAY 6, 2002

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION -CONGENITAL HEART DISEASE
ROOM 31

Moderators: Constantine Mavroudis and Thomas L. Spray

26. Outcomes After the Norwood Operation in Neonates with Critical Aortic

Stenosis or Aortic Valve Atresia

David A. Ashburn*, Brian W. McCrindle*, Christo I. Tchervenkov, Marshall L. Jacobs, Gary K. Lofland*, Edward L. Bove, Thomas L. Spray, Eugene H. Blackstone, William G. Williams; Toronto, Ontario. Canada

*Discussant: James S. Tweddell**

OBJECTIVE: To determine the institutional, anatomic, and procedural risk factors associated with various outcomes after the Norwood operation (NOR).

METHODS: From January 1994 to December 2000, 985 neonates with critical aortic stenosis (AVS) or atresia (AVA) were entered into a prospective 29-institution study, among whom 710 underwent NOR. Admission echocardiograms were independently reviewed for 64% of neonates. Time-related events were analyzed by parametric hazard function modeling, and incremental risk factors for mortality were sought.

RESULTS: 449 [63% (95% confidence limits: 59% to 66%)] were intermediate survivors of NOR (AVS: 187/261, 72%; AVA: 262/449, 58%). 390 (87%) underwent further palliation: cavopulmonary shunt 371, mortality=8%; Fontan 200, mortality=3%; transplant 18, mortality=39%; biventricular repair (BVR) 4, mortality=50%. Survival after NOR was 72%, 60% and 54% at 1 month, 1 year and 5 years. By competing risk analysis, 92% reached an "end-state" at 5 years post-NOR, consisting of death (43%), Fontan (45%), or transplant/BVR, (4%). Incremental risk factors for death after NOR but before transition to any other state include institutions entering <10 cases, lower birth weight, age >28 days at NOR, small ascending aorta, or small mitral valve. When institution was excluded, non-cardioplegic arrest, lower birth weight, age

>28 days, and diagnosis of aortic atresia increased risk. Type of aortic reconstruction, modified perfusion, and shunt variations were not risk factors, and operative mortality did not decrease during the study period.

CONCLUSION: Outcome is affected by institutional experience. In certain high-risk anatomic subgroups, further refinements of NOR or alternative therapy might improve outcome.

**By Invitation*

27. Outcome of Aortic Valve Repairs in Children: A Word of Caution

Nahidh Hasaniya*, Steven R. Gundry, Anees J. Razzouk*, Neda Mulla*,
Leonard L. Bailey; Loma Linda, CA

Discussant: Michel N. Ilbawi

OBJECTIVE: Although surgical aortic valvotomy has a long history of providing excellent palliation for aortic stenosis in infancy and childhood, it is not clear what is the long or short term fate of aortic valve repairs for aortic regurgitation (AI) in this same age group.

METHODS: From 1990 to 2000, a total of 22 patients underwent aortic valve repair for severe AI at our institution; 18 patients were under age 17 at the time of repair (age 3-17 yrs mean 7.3 yrs) 6/22 (27%) had bicuspid valves while 73% (16/22) had tricuspid valves. Type of repair varied with valve type but generally consisted of commissure resuspension, partial commissure closure and/or triangular resection of leaflets.

RESULTS: There were no deaths; 4 patients have been lost to followup. Followup ranges from 1-11 yrs (mean 5 yrs). 3/18 (17%) have mild AI, 6/18 (33%) have moderate AI by echo. In 9/ 18 pts (50%), the repair failed requiring reoperation from 1-60 months post-op (mean 26.3 months). Reoperation consisted of 6 Ross procedures and 3 mechanical AV replacements. There were no deaths at the second operation.

CONCLUSION: Although technically feasible, aortic valve repair for AI in children should be considered as short-term palliation only. Unless contraindicated, longer term solutions, such as the Ross procedure or mechanical valve replacement should be utilized at the primary operation.

**By Invitation*

28. Midterm Results Following Restoration of the Morphologic Left Ventricle to the Systemic Circulation in Patients with Congenitally Corrected Transposition of the Great Arteries

Stephen M. Langley*, Oliver Stumper*, Kami Dhillon*, Joseph V. de
Giovanni*, John G. Wright*, Paul Miller*, Bahulal Sethia*, David J. Barren*,
William J. Brawn*; Birmingham, United Kingdom

Discussant: Pedro J. Del Nido

OBJECTIVE: To determine outcome of patients with Congenitally Corrected Transposition of the Great Arteries (CCTGA) following restoration of the morphologic left ventricle (LV) to the systemic circulation.

METHODS: Between November 1991 and June 2001, 54 patients (median age 3.2 years, range 7 weeks - 40 years) underwent anatomic repair of CCTGA. This comprised a Senning procedure in all plus arterial switch (double switch) in 29 (53.7%), Rastelli in 22 (40.7%) and intraventricular rerouting in 3 (5.6%). LV training by pulmonary artery banding was performed in 9/29 (31%) prior to double switch. Follow up is complete (median 4.4 years).

RESULTS: Early mortality was 5.6% (3 patients) with two late deaths. Kaplan-Meier survival (\pm 1 SEM) was $94.4\% \pm 3.1\%$ at 1 year and $89.7\% \pm 4.4\%$ at 9 years. Survival following double switch was $84.9\% \pm 7.1\%$ at 7 years versus $95.5\% \pm 4.4\%$ in the Rastelli group ($p=0.32$). Of the 49 survivors, 46 (94%) were in NYHA class I. Seven had evidence of LV dilatation or impaired systolic function (6/25 for double switch versus 1/24 for other patients, $p=0.1$) including one requiring heart transplant for LV failure. Four patients in the double switch group have developed moderate aortic valve regurgitation (two requiring valve replacement). Overall freedom from reoperation at 1 and 9 years was $96.0\% \pm 2.8\%$ and $79.5\% \pm 8.7\%$ with no significant difference between the groups ($p=0.85$).

CONCLUSION: Anatomic repair of CCTGA can be carried out with low early mortality. Excellent functional status can be achieved with good midterm survival. Continued surveillance is necessary to determine the longer-term function of the aortic valve and the left ventricle in the systemic circulation.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

HALL B

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION -CONGENITAL HEART DISEASE

ROOM 31

Moderators: Constantine Mavroudis and Thomas L. Spray

29. Determinants of Acute Homograft Failure in Right Ventricular Outflow Tract:

A Multi-Institutional Study

Zahid Amin*, David A. Danford*, David Gremmels*, Marcus Schamberger*, William G. Williams, Thomas J. Forbes*, Jennifer A. Strawn*, Vadiyala M. Reddy, Kim F. Duncan*, Philip Moore*, Frank L. Hanley; Omaha, NE; San Francisco, CA; Indianapolis, IN; Toronto, Ontario Canada; Detroit, MI
MI Discussant: John W. Brown

OBJECTIVE: Right ventricular outflow tract (RVOT) homograft (HG) failure commonly results from a non-growing HG in a rapidly growing patient. However, it is unknown why some HG fail a few months after placement. The objective of this study was to identify risk factors for acute HG failure (AHF).

METHODS: Five institutions (INST) participated in the study of AHF (HG Stenosis with a gradient of 45 mm HG or, moderate to severe insufficiency or, removal within one year of placement). Two INST provided data for AHF (group F1, n=46) and concurrent control without AHF (group C, n=289). A binary logistic regression model was derived to identify risk factors for AHF among the following variables: diagnosis (Dx), HG Z-value, type and use of HG extension,

postoperative fever, body surface area (BSA), use of anti-inflammatory agents and type of HG (aortic or pulmonary). Three INST provided data on AHF only (group F2, n=25). Chi-square test and t-test were used to determine whether risk factors were over-represented in group F2 vs C.

RESULTS: There were 72 AHF among 879 cases (8.2%). Risk factors for AHF are summarized in the table. Dx, type of extension or HG, and use of anti-inflammatory agents were not significant risk factors. In group F2, small BSA and HG extension were over-represented relative to group C ($P<0.05$), but post-operative fever and Z-value were not.

Variable	Odds Ratio	95% CI.	P
Use of Extension	16.28	5.98-44.35	<0.001
Smaller BSA	0.10	0.02-0.43	0.002
Fever	9.12	1.38-60.16	0.022
Higher HG Z-value	1.52	1.13-2.05	0.005

CONCLUSION: Higher HG Z-value, use of HG extension, smaller BSA and post-operative fever are associated with AHF. Dx, HG type, and anti-inflammatory use were not independent risk factors. HG extension should be avoided during surgery, if possible.

**By Invitation*

30. Home Surveillance Program Prevents Interstage Mortality Following the Norwood Procedure

Nancy S. Ghanayem*, George M. Hoffman*, Kathy A. Mussatto*, Joseph R. Cava*, Peter C. Frommelt*, Nancy Rudd*, Michele Steltzer*, Sarah Bevandic*, Robert D. Jaquiss*, S. Bert Litwin, James S. Tweddell*;
Milwaukee, WI

*Discussant: J. William Gaynor**

OBJECTIVE: Mortality between stage 1 palliation (SIP) for hypoplastic left heart syndrome and bidirectional cavopulmonary connection (BDCPC) has been associated with decreased SaO₂ and hypovolemia. To identify these physiologic risk factors, we developed a home surveillance program enlisting parents to monitor daily SaO₂ and weight.

METHODS: Cohorts were created from all patients who underwent SIP since 7/96. Patients from 7/96 - 9/00 (GpA, 65 patients) were discharged without home monitoring and compared to patients from 9/00 - 7/01 (GpB, 17 patients) who were discharged with a pulse oximeter and an infant scale. Thresholds for parents to contact their physicians included SaO₂ < 70% or weight loss of 30 grams over 2 days. Survival analysis was performed using Kaplan Meier methods.

RESULTS: Hospital survival was 91% (59/65) for GpA and 100% (17/17) for GpB. The groups did not differ by age, diagnosis, shunt size, or length of stay. Interval mortality was 15% (9/59) in GpA and 0% in GpB ($p=0.04$). Average age at BDCPC was 5.8 ± 3 mo for Gp A and 4.2 ± 1.8 mo for GpB ($p=0.06$). In GpA, interval mortality occurred at 85 ± 25 days; in GpB, 6/17 had interval hypoxemia detected with subsequent BDCPC at less than 100 days.

CONCLUSION: Home surveillance allows for early detection of physiologic variances that may not be well tolerated following S1 P. Identification of decreasing SaO₂ led to earlier BDCPC and

improved survival in home monitored patients. Hence, progression to BDCPC may be better determined through physiologic monitoring rather than arbitrary timing.

**By Invitation*

31. Immunogenicity of Decellularized Cryopreserved Allografts in Pediatric Cardiac Surgery: Comparison with Standard Cryopreserved Allografts

John A. Hawkins, Jamison Jones*, Neal D. Hillman*, Linda M. Lambert*, Gregory B. Di Russo*, Tracie Profaizer*, Thomas C. Fuller*, Robert E. Shaddy*; Salt Lake City, LIT

Discussant: John E. Mayer

OBJECTIVE:: Recognition of the immunogenicity of standard Cryopreserved allografts has led to the development of new, decellularized, allografts (Cryovalve SG®). This preliminary study examines the HLA antibody response to these decellularized allografts and compares it to standard allograft material

METHODS: We prospectively measured the frequency of panel-reactive human leukocyte antigen (HLA) Class I (HLA -A, B and C) and Class II (HLA-DR/DQ) alloantibodies (PRA) in 14 children (age=8.5±7.9 years) receiving decellularized, Cryopreserved allografts, including 6 undergoing allograft patch insertion and 8 with a valved pulmonary allograft. We compared them to 20 patients (age= 1.7±2.4 yr.) undergoing implantation of standard Cryopreserved allografts; 8 with valves and 12 with allograft patch. All patients had PRA measured prior to, 1 month and 3 months post-operatively. Class I and Class II PRA levels were determined with flow cytometry.

RESULTS: We found the following PRA levels in decellularized and standard allografts (*p<0.05 for decellularized compared to standard allografts, p<0.05 compared to pre-op):

	Pre-op (%)	1 month (%)	3 months (%)
Decellularized Class I	0.3±1.1	8±16*	16±30*_
Decellularized Class II	0±0	14±28*	22±28*_
Standard Class I	5±11	60±38_	83±27_
Standard Class II	0.3±1.1	35±36_	57±34_

CONCLUSION: Decellularized grafts elicit significantly less Class I and Class II HLA antibody formation than standard Cryopreserved allograft material. However, compared to pre-op levels, decellularized allografts elicit a significant formation of Class I and II alloantibodies by 3 months postoperatively. Further experience is necessary to determine if the reduced immunogenicity of decellularized allografts will truly allow tissue ingrowth and improved durability in patients.

**By Invitation*

32. Novel Cerebral Physiologic Monitoring to Guide Low-Flow Cerebral Perfusion During Neonatal Aortic Arch Reconstruction

Charles D. Fraser, Jr.*, Dean B. Andropoulos", Stephen A. Stayer*, Emmitt D. McKenzie* ; Houston, TX

*Discussant: Frank A. Pigitla**

OBJECTIVE: This study describes the use of near infrared spectroscopy (NIRS) combined with transcranial Doppler measurement of cerebral blood flow (TCD) to guide bypass flow during regional low flow cerebral perfusion (RLFP) (1) for neonatal aortic arch reconstruction.

METHODS: Cardiopulmonary bypass was instituted (minimum flow 150 ml/kg/min) utilizing pH stat blood gas management. During aortic arch reconstruction, RLFP was used, either through a Goretex graft in the innominate artery, or through ascending aortic cannulation. DHCA was minimized. MRS of the right frontal cortex and TCD of the right middle cerebral artery were utilized. During RLFP, the pump flow was adjusted to maintain the cerebral saturation and blood flow at baseline values obtained on full flow at 17-22°C.

RESULTS: 9 patients had hypoplastic left heart syndrome and 10 had interrupted or hypoplastic aortic arch. Average age was 15 days and weight 3.3 kg. RLFP time was 47 ± 23 min, and DHCA 19 ± 18 min. 18 of 19 patients survived to hospital discharge without apparent neurologic morbidity. See Table. Bypass flow necessary to maintain baseline cerebral saturation and blood flow varied from 29-84 ml/kg/min, and mean arterial pressure (MAP) varied from 19-49 mm Hg. Bypass flow during RLFP had a poor correlation with pressure in the radial artery (r = 0.11).

Table

	Baseline full CPB flow (17-22° C)	During low-flow cerebral perfusion (17-22° C)	After repair full flow (17-22° C)
MAP radial/brachial artery (mm Hg)	35 ± 5	30 ± 8*	35 ± 6
MAP umbilical/femoral artery (mm Hg)	34 ± 4	13 ± 4*	36 ± 6
CPB flow (ml/kg/min)	151 ± 9	62 ± 19*	152 ± 5
TCD (mean, cm/sec)	21 ± 10	22 ± 10	22 ± 10
NIRS (% cerebral saturation)	86 ± 10	87 ± 8	86 ± 8
Calculated base excess/deficit	+1.9 ± 3.3	+0.4 ± 2.6*	-0.5 ± 2.7¶
Hematocrit (%)	23.9 ± 2.2	23.2 ± 2.2	23.4 ± 2.0

* p < 0.05, vs. baseline and post-repair values; ¶ p < 0.05, vs. baseline, two way ANOVA

CONCLUSION: TCD directly measures cerebral blood flow, and combined with NIRS may assure appropriate bypass flows during RLFP, thus affording maximal cerebral protection. Reference: 1. Pigitla FA, Nemoto EM, Griffith BP et al. J Thorac Cardiovasc Surg 2000;119:331

**By Invitation*

33. Right Ventricle-to-Pulmonary Artery Shunt in First-Stage Palliation of Hypoplastic Left Heart Syndrome

Sliunji Sano*, Kozo Ishino*, Masaaki Kawada*, Sadahiko Arai*, Shingo Kasahara*, Tomohiro Asai*, Zen-ichi Masuda*; Okayama, Japan

Discussant: S. Bert Litwin

OBJECTIVE: To prevent sudden circulatory collapse due to coronary malperfusion, we have constructed a RV-PA shunt in first-stage palliation of HLHS. Postoperative cardiac and pulmonary function of these patients are reviewed.

METHODS: Between February 1998 and September 2001, 18 consecutive infants, aged 1 to 57 days (median, 9 days) and weighing 1.6 to 3.7 kg (median, 2.6kg) underwent a modified Norwood operation with the RV-PA shunt. The procedure included a Damus-type aortic arch reconstruction and a non-valved polytetrafluoroethylene shunt between a small right ventriculo-tomy and a distal stump of the main pulmonary artery. The size of the shunt used was 4mm in 6 patients, 5mm in 11 and 6mm in 1. All survivors were evaluated by cardiac catheterization and echocardiography.

RESULTS: All patients could be managed without any particular ventilator manipulation. There were 16 survivors (89%), including 3 patients weighing less than 2kg. There were 2 late deaths due to shunt obstruction, however no patient died due to ventricular dysfunction. Twelve patients underwent bidirectional Glenn anastomosis after a mean interval of 4.5 months, 2 underwent Fontan operation. Remaining 2 patients are awaiting Glenn anastomosis.

	RVEDP (mmHg)	PAP (mmHg)	PA index (Nakata)	PVR	Sat
post RV-PA Norwood (12)	5.3 (0-12)	12.3(10-15)	200(119-280)	2.0(1.5-3.0)	71 (63-78)
post BDG (3)	6.3 (2-9)	11.0(7-14)	150(140-158)	2.0(1.7-2.5)	83 (80-87)

CONCLUSION: The modified Norwood operation with the RV-PA shunt offers excellent clinical result without delicate postoperative management. Postoperative hemodynamics are comparable to those of classical Norwood operation.

**By Invitation*

34. Re-operations and Survival Following Primary Repair of Congenital Heart Defects in Children

James L. Monro*, Christos Alexiou*, Qiang Chen*, Hyam Mahmoud*, Ahmed Al-Khaddour*, Anthony P. Salmon", Barry R. Keeton*; Southampton, United Kingdom

Discussant: Marshall L. Jacobs

OBJECTIVE: To evaluate the incidence of re-operation, the associated risk and the survival following repair of congenital heart defects (CUD) under cardiopulmonary bypass (CPB) in children.

METHODS: 1220 consecutive children, undergoing primary repair of CUD under CPB between 1976 and 2001 by one surgeon were studied. 585 (48%) were infants (1 day-1year) (group A) and 635 (52%) were older children (1-16years) (group B). Mean follow up was 14.4years, range 0-25.6years, total 15532 patient years.

RESULTS: Hospital mortality was 4.4% (6.5% for group A Vs 2.5% for group B, p=0.001). 150 patients (12%) underwent a total of 180 re-operations. Of these re-operations, 25 were planned (e.g. staged Fontan), 128 were inevitable (e.g. valve replacement late after valvotomy) and 28 were unexpected (e.g. repair of pulmonary artery stenosis after switch procedure). Mortality at re-operation was 6.1% (7% Vs 5.1%, p=0.7). Twenty-year freedom from any re-operation was 84±1% (80±2 Vs 89±1%, p=0.002) and from unexpected re-operation this was 98±0.4% (97±0.7 Vs 99±0.4%, p=0.1). Overall 20-year survival, including hospital mortality, was 90±0.9% (86±2 Vs 94±1%, p=0.0001). Kaplan-Meier, freedom from re-operation and survival at 20 years for selected conditions are shown below:

Condition	Follow-up (Patient years)	Freedom from re-op. (%±SEM)	Freedom from unexp. re-op.(% ±SEM)	Survival (%±SEM)
Aortic stenosis	952	50 ± 12	100	90 ± 5
ASD	2652	100	100	100
VSD	3103	96 ± 2	99 ± 0.5	96 ± 1
AVSD	670	74 ± 6	96 ± 3	87 ± 4
TOP	2780	91 ± 2	96 ± 2	95 ± 2
Fontan	443	69 ± 8	98 ± 2	70 ± 8
TAPVD	374	89 ± 6	93 ± 5	90 ± 5
TGA (Mustard)	810	88 ± 5	94 ± 4	83 ± 5
TGA (Switch)	271	83 ± 6	83 ± 6	88 ± 6

CONCLUSION: In our experience, 12% of children having primary repair of CHD under CPB undergo further procedures with an acceptable operative risk. Unexpected re-operations are uncommon (2.2% of all patients in this series) and this facilitates the process of parental counselling pre-operatively. Late survival is encouraging.

**By Invitation*

TUESDAY MORNING, MAY 7, 2002

7:00 a.m. C. WALTON LILLEHEI RESIDENT FORUM SESSION

HALL C

Moderators: Ralph J. Damiano Jr. and Larry R. Kaiser

L1. Altered Pattern of Gene Expression in Ascending Thoracic Aortic Aneurysms and Abdominal Aortic Aneurysms

Tarck S. Absi*, Thoralf M. Sundt, Marc R. Moon*, William S. Tung*, Jason K. Lee*, Ralph J. Damiano, Jr., Robert W. Thompson*; St Louis, MO

OBJECTIVE: The purpose of this study was to characterize patterns of gene expression in ascending thoracic aneurysms (ATAAs) and abdominal aortic aneurysms (AAAs).

METHODS: Aortic wall tissues were obtained from patients undergoing surgical repair for either ATAAs or AAAs, with normal ascending aortas (ATNAs) or infrarenal aortas (NIAs) obtained from organ transplant donors as controls (n = 4 in each group). Total RNA was used to create array-specific [32P] -labeled cDNA probes by reverse transcription. Labeled cDNA probes were hybridized to a nylon microarray containing 1176 cDNA clones, and phosphoimaging and densitometry were used to standardize the expression level for each gene. Differential expression

was quantified using a statistical package designed specifically for cDNA microarray analysis (Gene Spring).

RESULTS: 105 of the 1176 genes (8.9%) were differentially expressed between ATAs and ATNAs, including upregulated expression of Cathepsin D (5.4fold;pO<.05), Cathepsin C (4.9 fold; p<0.05), and II.-1 beta (5.4 fold; p0<.05). There were 103 genes (8.7%) differentially expressed between AAAs and NIAs, including increased expression of Cathepsin H (4.5 fold; p0<.05), and Apolipoprotein E (14 fold; p<0.05). Gelatinase B/MMP-9 was markedly upregulated in both ATMs (8.64 fold; p<0.05) and A/Us (86.5 fold; p0<.05).

CONCLUSION: ATM and AM tissues exhibit distinct patterns of altered gene expression that appear to reflect the importance of chronic inflammatory responses and elevated production of extracellular matrix-degrading proteinases in these conditions. Further studies are needed to elucidate the pathophysiologic significance of individual gene products that exhibit differential levels of expression in ATAs and AMs.

³1994-96 Research Scholar

*By Invitation

L2. Induced Fibrillation is Equally Effective as Crystalloid Cardioplegia in the Protection of Fetal Myocardial Function

Sunil P. Malhotra*, Stephan Thelitz*. R. Kirk Riemer*. V. Mohan Reddy*, Sam Suleman*, Frank L. Hanley; New York, NY; San Francisco, CA

OBJECTIVE: Fetal cardiac intervention represents a potential advance in the treatment of congenital cardiac lesions that increase in complexity during development. Prenatal repair of a primary defect can prevent pathological blood flow patterns that result in hypoplasia of a cardiac chamber or great vessel. However, strategies to optimize protection of the fetal myocardium have not been studied. A biventricular working fetal heart preparation was used to evaluate the cardioprotective properties of induced fibrillation and crystalloid cardioplegia.

METHODS: Hearts from 16 fetal lambs, 115-125 days gestation, were harvested and perfused with Krebs-Henseleit (K-H) solution. To simulate the parallel circulation of the fetus, the descending aorta was ligated distal to the ductal insertion and the branch pulmonary arteries were ligated. Hearts were arrested for 30 minutes with normothermic electrical fibrillation (N=8) or hypothermic crystalloid (Plegisol) cardioplegia (N=8) before reperfusion with K-H. Baseline and postarrest myocardial function measurements were obtained from the analysis of pressure-dimension relationships.

RESULTS: Fetal myocardial protection was equivalent following fibrillatory and cardioplegic arrest. There was no statistically significant difference with either technique in recovery of biventricular systolic function, diastolic function, or overall contractility (Table). Myocardial water content was also unchanged in hearts arrested with fibrillation and cardioplegia (84±1.5% vs 83.7±0.9%, p=0.71).

	FIBRILLATION	CARDIOPLEGIA	p-value
% recovery of LV end-systolic elastance (HSK)	70±5.0	68±15	0.52
% recovery of RV.F.SK	68±4.5	65±5.0	0.26
% increase in LV diastolic stiffness	32±5.3	38±11	0.24
% increase in RV diastolic stiffness	25±3.3	27±2.1	0.46
% recover of I.V preload recruitable stroke work (PRSW)	77±3.6	75±13	0.82
% recovery of RV PRSW	81±9.1	79±4.5	0.69

CONCLUSION: Normothermic fibrillation and hypothermic crystalloid cardioplegia provide equal protection of the fetal myocardium. In contrast to cardioplegic arrest, fibrillation avoids the adverse effects of hypothermia, cardioplegia, and aortic clamping. In the setting of the limited myocardial reserve of the fetus, fibrillation may be preferable for in utero repairs of uncomplicated congenital heart defects.

*By Invitation

L3. Viral Gene Transfer of the Anti-Apoptotic Factor ARC Protects Against Post-Ischemic Heart Failure

Subhasis Chatterjee*, Lawrence T. Bish*, Vasant Jayasankar*, Allan S. Stewart*, Michael T. Crow*, H. Lee Sweeney*, Timothy J. Gardner; Philadelphia, PA; Bethesda, MI)

OBJECTIVE: Apoptosis after acute myocardial ischemia and subsequent ventricular remodeling is a pathologic mediator of heart failure. This study assessed whether a new anti-apoptotic factor ARC, a caspase Mocker, given after acute ischemia prevents late post-ischemic cardiomyopathy.

METHODS: 16 rabbits underwent 30 minutes regional ischemia by circumflex occlusion followed by reperfusion. In the experimental group (n=8), an adenovirus encoding for ARC was administered after the ischemic period directly into the LV cavity with 30 sec. transient aortic occlusion. A control group (n=8) received saline. Function was assessed by echocardiography and sonomicrocrystal fractional shortening of the borderzone compared to normal LV. After six weeks, histology with II & K, apoptosis by TUNEL assay, and gene expression with Western blot were evaluated.

RESULTS: ARC rabbits maintained higher ejection fractions at 4 and 6 weeks compared to controls (38% vs. 32%, 36% vs. 29%, p<0.05). There was greater preservation of borderzone fractional shortening (FS) at 6 weeks (70.81% vs. 46.49, p<0.05). Compared to normal rabbits, the control group had greater LV chamber dilatation (13.44 vs. 15.29 mm, p<0.05) and loss of wall thickness (3.61 vs. 2.71 mm, p<0.05) than the ARC group (13.59 and 3.48mm, p=NS). Reduced apoptosis (5.76% vs. 0.77%, p=0.001) was seen in the ARC group compared to controls.

CONCLUSION: Gene transfer of ARC after acute ischemia preserves LV function. This benefit results from a reduction in apoptosis with less subsequent ventricular remodeling to better preserve ventricular geometry. ARC administration offers a novel and effective strategy to prevent post-ischemic heart failure.

**By Invitation*

L4. Expression of Fas-Associating Death Domain Protein Induces Apoptosis in Lung Cancer Cells

Peter K. Kim*, James D. Luketich, Timothy R. Billiar*; Pittsburgh, PA

OBJECTIVE: Non-small cell lung cancers commonly develop resistance to radiation and chemotherapy, and often present at stages beyond surgical resectability. Because current treatment modalities are inadequate, novel therapies are necessary to reduce the effects of the increasing incidence in pulmonary neoplasms. FADD (fas-associating death domain protein) is a central mediator of death receptor-initiated apoptosis that directly activates the Caspase-8 protease in lung epithelium. We hypothesized that overexpression of the FADD gene would effectively eradicate lung cancer cells.

METHODS: We constructed a replication-deficient, adenoviral vector that expresses the wild-type murine FADD gene (Ad-FADD). Cultured A549 squamous cell carcinoma cells were exposed to increasing multiplicities of infection (MOI) of Ad-FADD or control virus (Ad-Psi5) for 4 hours. Twenty-four hours later cells were assessed for cell death by vital staining with trypan blue and microscopic analysis. Protein lysates were examined by Western blotting for expression of FADD and activated Caspase-8.

RESULTS: Adenoviral infection with Ad-FADD, but not Ad-Psi5 control virus, resulted in dose-dependent expression of the FADD protein and appearance of cleaved, activated Caspase-8 in A549 cells. (Figure) Increasing MOI of Ad-FADD, but not Ad-Psi5, was associated with increased cell death. Ad-FADD infection at MOI of 25 induced at least 10-fold increase in FADD levels, and cell death was detected in 83/127 (65%) of A549 cells.

CONCLUSION: Overexpression of FADD by adenoviral gene transfer induced dose-dependent cell death in A549 lung epithelial cancer cells. Expression of FADD results in activation of caspase-8, a hallmark of apoptosis. Delivery of Ad-FADD to lung cancer may be a novel method for therapy of non-small cell lung cancer.

**By Invitation*

L5. Intramyocardial or Intravenous Delivery of Exogenous Vascular Endothelial Growth Factor Increases Endogenous Production of Both VEGF and Basic Fibroblast Growth Factor in a Porcine Model of Hibernating Myocardium.

John E. Scarborough*, Patrick W. Domkowski*, Luis II. Diodato*, Shankha S. Biswas*, Anne Pippen*, Brian H. Annex*, Kevin P. Landolfo*; Durham, NC

OBJECTIVE: Although therapeutic angiogenesis is emerging as a potential therapy in patients with end-stage coronary artery disease, the myocardial tissue response to exogenous growth factor administration is largely unknown. The goal of this study was to examine the effects of intramyocardial (IM) and intravenous (IV) administration of exogenous vascular endothelial growth factor (VEGF) protein on endogenous myocardial growth factor levels and vascular density in a porcine model of chronic hibernating myocardium.

METHODS: Eighteen pigs underwent a 90% left circumflex (LCX) artery stenosis with subsequent documentation of hibernating myocardium by positron emission tomography and dobutamine stress echocardiography. Animals were then randomized to receive IM VEGF at 15 Mg/ml (n=6, 30 injections per animal), IV VEGF at 15 Mg/ml (n=6), or IM vehicle control (n=6). Six months after treatment, the animals were sacrificed and six 3X3mm samples were harvested from the LCX territory (hibernating myocardium) of each animal. VEGF and basic fibroblast growth factor (bFGF) protein levels were then determined using commercially available enzyme-linked immunosorbent assay kits. Vascular density analysis was also performed using an NIH Imaging Program. Statistical comparisons were performed using unpaired Student's t-test.

RESULTS: As shown in the table below, exogenous VEGF protein delivered either by direct injection into the myocardium or intravenously resulted in increased endogenous production of VEGF and bFGF protein, as well as increased vascular density, six months following treatment (protein concentrations expressed as pg/Jg of soluble protein).

	Control(n=6)	IM VEGF(n=6)	IV VEGF(n=6)
VEGF protein	352 ± 223	844 ± 379	921 ± 350
P Value vs. Control		< 0.05	<0.05
bFGF protein	242 ± 67	428 ± 197	309 ± 114
P Value vs. Control		5<0.0	<0.05
Vascular Density	840 ± 81	1730 ± 108	2145 ± 145
P Value vs. Control		<0.05	<0.05

CONCLUSION: These findings suggest that administration of a single exogenous angiogenic growth factor initiates a process that upregulates multiple endogenous angiogenic growth factors in hibernating myocardium.

*By Invitation

L6. IL-10 Gene Transfection of Donor Lungs Ameliorates Post-Transplant Cell Death by A Switch from Cellular Necrosis to Apoptosis

Stefan Fischer*, Marc dePerrot*, Jonathan A. Cardella*, Mix MacLean*, Yumiko Imai*, Michiharu Suga*, Mingyao Liu*, Shaf Keshavjee; Toronto, Ontario, Canada

OBJECTIVE: We have previously shown that cell death is a pathophysiologic consequence of inflammatory processes during ischemia and reperfusion in transplanted lungs. Apoptosis is predominant in transplanted lungs after 6 to 12 hrs of preservation with satisfying lung function and necrosis in lungs after 18 to 24 hrs of preservation with poor graft function. The aim of this study was to determine whether donor lung transfection with the gene that encodes for the anti-inflammatory cytokine IL-10 ameliorates cell death in transplanted lungs.

METHODS: 15 Lewis rats were divided into 3 groups (n=5). Group 1 received intratracheal administration of 5×10^9 pfu Ad5E1RSVhIL-10 (IL-10), group 2 5×10^9 pfu 'empty' vector (EV), and group 3 vector diluent (VD, 3% sucrose). After 24 hrs in vivo transfection, lungs were stored at 4 degree C for 24 hrs and then transplanted. After 2 hrs of reperfusion lungs were flushed with a trypan blue (TB) solution via the pulmonary artery in order to stain all dead cells in the lungs and then fixed in 10% formalin. For apoptosis detection the TUNEL technique was applied in combination with a propidium iodide stain, which stains all nucleated cells (dead + alive). TB+/TUNEL- cells were considered as necrotic cells, TB+/TUNEL+ cells were considered as apoptotic cells. This triple staining technique has previously been developed, validated and described by us.

The total number of cells in study lungs and the number of apoptotic and necrotic cells were counted in random high power fields of tissue sections.

RESULTS: The total number of dead cells was similar in the EV, VD and IL-10 group with 32.1 ± 3.3 , 30.2 ± 2.5 and 30.3 ± 3.8 , respectively. The amount of apoptosis, however, was the highest in IL-10 lungs (9.7 ± 1.9) compared to 2 ± 1.9 and 1.8 ± 2 in VD and EV lungs. Opposite to that, the number of necrotic cells was the lowest in the IL-10 group with 20.6 ± 5.7 vs. 28.3 ± 3.1 and 30.3 ± 4.2 in the VD and EV group. The differences between apoptosis and necrosis within the EV and VD group were $p < 0.001$. In IL-10 lungs apoptosis differed from necrosis also significantly ($p < 0.05$). Apoptosis and necrosis in IL-10 lungs were significantly different from numbers in EV and VD lungs (all $p < 0.05$).

CONCLUSION: Donor lung IL-10 gene transfection prior to transplantation ameliorates post-transplant cell death by decreasing the amount of necrotic cells and increasing the amount of apoptotic cells. It is possible that AdhIL-10, by decreasing pro-inflammatory cytokine production, leads to less overall injury which in turn preserves the ability of damaged cells to undergo the more quiescent and less tissue damaging mode of cell death - apoptosis - rather than necrosis. This study supports the role of gene therapy in lung transplantation and may help to make longer ischemic times and improved graft preservation possible in the future.

**By Invitation*

L7. A Possible Genetic Basis for Aortic Root Dilatation in Patients with Bicuspid Aortic Valve Malformations

Paul W.M. Fedak*, Subodh Verma*, Mauro de Sa*, Nafiseh Nili*, Pedram Kazemian*, Jagdish Butany*, Bradley H. Strauss*, Richard D. Weisel, Tirone E. David; Toronto, Ontario, Canada

OBJECTIVE: Bicuspid aortic valve (BAV) malformations are associated with aortic root (AR) dilatation, aneurysm formation, and dissection. Destruction of the extracellular matrix (ECM) may contribute to aortic dilatation given that increased matrix metalloproteinase (MMP) activity can promote aneurysm formation. Targeted under-expression of the fibrillin-1 gene induces ECM degradation and progressive AR dilatation similar to the Marfan aorta. We hypothesized that the vascular manifestations of BAV disease are induced by fibrillin-1 deficiency resulting in MMP-mediated ECM destruction and functional collapse of the vessel wall.

METHODS: AR samples were obtained from patients with normal and diseased tricuspid aortic valves (TAV, $n=17$) and congenital BAV ($n=22$). Echocardiography determined aortic dimensions. Indirect immunofluorescence and image analysis quantified fibrillin-1 and elastin. Differential hydroxyproline determination also quantified collagen and elastin. MMP gelatin zymography determined MMP-2 and MMP-9 activity.

RESULTS: Fibrillin-1 content was reduced in BAV aorta compared to TAV aorta (mean optical density+STD: 14.1 ± 8.2 vs. 24.5 ± 7.2 ; $p=0.02$). Fibrillin-1 content was not different between normal and diseased TAV aortas ($p=0.451$). Elastin and collagen content was similar (elastin (TAV vs BAV): 569 ± 169 vs. 513 ± 203 Mg/mgdry wt; $p=0.512$; collagen: 185 ± 97 vs. 203 ± 111 pg/mgdry wt.; $p=0.209$). Compared to TAV aorta, MMP-2 activity was 119% greater in BAV aorta (mean densitometric units: 20.61 ± 12.57 vs. 9.41 ± 5.57 ; $p=0.046$) and correlated positively with AR diameter ($r=0.75$, $p=0.05$). Pro-MMP-2 and MMP-9 levels were not significantly different.

CONCLUSION: With congenital BAV malformations, fibrillin-1 deficiency may promote MMP activation, which disrupts medial ECM components contributing to progressive AR dilatation and aneurysm formation. These data may influence surgical approaches to BAV disease.

**By Invitation*

L8. Targeting Matrix Metalloproteinases Alters the Course of Post-Myocardial Infarction Remodeling: Identification of Novel Therapeutic Strategies

William M. Yarbrough*, Rupak Mukherjee*, Theresa A. Brinsa*, Kathryn B. Dowdy*, Amelia A. Scott*, G. Patricia Escobar*, Cassandra Joffs*, David G. Lucas*, Fred A. Crawford, Jr., Francis G. Spinale*; Charleston, SC

OBJECTIVE: Objectives: Left ventricular (LV) remodeling often occurs following myocardial infarction (MI). This process is termed MI expansion and is associated with changes in the extracellular matrix (ECM). Matrix metalloproteinases (MMP) are a family of proteolytic enzymes that degrade the ECM and contribute to pathological remodeling such as occurs in inflammatory and neoplastic processes. Accordingly, the central hypothesis of this project is that modulation of myocardial MMP activity will alter MI expansion.

METHODS: Methods: Ligation of obtuse marginal coronary arteries and radio-opaque marker placement was performed in pigs (23 kg, MI size $21 \pm 2\%$). At 5 days post-MI, pigs were randomized to MMPi (MI+MMPi, PD166793, 20 mg/kg SID, n=9) or placebo (n=8). LV end-diastolic dimension (LVEDD, 2D targeted echocardiography) and regional MI geometry (marker area/fluoroscopy) were serially measured for up to 8 weeks.

RESULTS: Results: Regional MI expansion rate was diminished with MMPi (FIGURE), which translated into reduced LVEDD at 8 weeks post-MI (4.7 ± 0.1 vs 5.1 ± 0.1 cm, $p < 0.05$). This broad-spectrum MMPi attenuated post-MI LV remodeling. However, broad-spectrum inhibition, particularly that of the MMP-1 species, can cause undesirable systemic effects. Accordingly, selective targeting of myocardial MMPs has begun. A pharmacologic regimen (PGE 7113313, 20mg/kg TID) has been established that achieves steady-state plasma concentrations of 212 ± 11 ng/mL which inhibits MMP-2,9 and 13 while sparing MMP-1. This strategy is being deployed in a similar MI model.

CONCLUSION: Conclusions: This study for the first time demonstrated that increased MMP activation contributes to post-MI regional and global LV remodeling. Selective targeting of myocardial MMP species in the post-MI period holds significant therapeutic potential.

**By Invitation*

9:00 a.m. SCIENTIFIC SESSION

HALL C

Moderators: Timothy J. Gardner and Tirone E. David

9:05 a.m. THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION

David B. Skinner, President

HALL C

BASIC SCIENCE LECTURE *The Immune Response to Human Cancer: Lessons from the Molecular Analysis of Patients with a Dramatic Response to Immunotherapy*

Steven A. Rosenberg, Bethesda, Maryland

Introduced By: Timothy J. Gardner, Philadelphia, Pennsylvania

9:35 a.m.

35. A Comparative Analysis of Positron Emission Tomography and

Mediastinoscopy in Staging Patients with Non-Small Cell Lung Cancer

Gonzalo V. Gonzalez-Stawinski*, Anthony Lemaire*, Faisal M. Merchant*, Elizabeth K. O'Halloran*, David II. Harpole, Thomas A. D'Amico*; Durham, NC

Discussant: Douglas E. Wood

OBJECTIVE: Positron emission tomography (PET) has been demonstrated to improve the detection of distant metastases in patients with lung cancer. This study compares the efficacy of PET to mediastinoscopy in mediastinal staging of patients with non-small cell lung cancer (NSCLC).

METHODS: Between May 1995 and May 2000, PET was performed on 1,988 patients with suspected or proven NSCLC at a single institution. Cervical mediastinoscopy was subsequently performed in selected patients without demonstrable evidence of distant metastases. The accuracy of mediastinal staging was analyzed by comparing the prospective results of PET to the histopathologic results of mediastinoscopy, by nodal station.

RESULTS: In this study, 203 patients with NSCLC (125 men) underwent mediastinoscopy after PET. Of the 66 patients with a positive PET, only 25 patients had a positive mediastinoscopy in the corresponding nodal station (subsequently verified at thoracotomy). Of the 137 patients with a

negative PET, 16 patients were demonstrated to have N2 or N3 disease at mediastinoscopy. The sensitivity, specificity, positive and negative predictive values for PET were 61.0%, 74.7%, 37.9%, and 88.3%, respectively. Histologic findings in patients with NSCLC and a false-positive mediastinal PET included sarcoidosis, silicosis, and granulomatous inflammation.

CONCLUSION: PET neither confirmed nor excluded involvement of the mediastinum in this study. Cervical mediastinoscopy with lymph node biopsy remains the gold standard for mediastinal staging in patients with potentially resectable NSCLC.

**By Invitation*

36. Totally Endoscopic Mitral Valve Repair: Feasible, Reproducible and Durable

Filip P. Casselman*, Sam Van Slycke*, Helge Dom*, Dave Lambrechts*,
Yvette Vermeulen*, Hugo Vanermen*; Aalst, Belgium

Discussant: Delos M. Cosgrove

OBJECTIVE: To document the feasibility, safety and effectiveness of performing mitral valve repair by a totally endoscopic approach.

METHODS: Between February 1, 1997 and October 1, 2001, 187 patients underwent totally endoscopic mitral valve repair at our institution. The mean age was 60.7±13.2 years and 62% were male. Median preoperative functional class and degree of mitral regurgitation were 2 and 4, respectively. Data collection included an institutional protocol assessing procedure related pain, cosmesis and functional recovery. Statistical analysis included Kaplan-Meier and Cox regression methods. Mean follow-up was 18.8±15.2 months and was 100% complete.

RESULTS: Associated atrial procedures were performed in 8.6% (n=16) of the patients. Two patients required intra-operative conversion to sternotomy. Thoracoscopic reevaluation for suspected bleeding (n=19) was part of our aggressive postoperative management. One patient required sternotomy for control of bleeding. Hospital mortality included 1 patient (0.5%) and was not technology related. There were one early and five late reoperations, four of which were due to endocarditis. No risk factors for repair failure could be detected. Freedom from mitral valve reoperation at 4 years was 94.5±2.3%. Median degree of mitral regurgitation at follow-up was 0. Ninety two % of the patients were highly satisfied with either no or mild postoperative pain and 98.2% felt they had an esthetically pleasing scar.

CONCLUSION: Totally endoscopic mitral valve repair can be done safely with excellent results and a high degree of patient satisfaction. It is now our exclusive approach for isolated atrioventricular valve disease

10:15 a.m. INTERMISSION - VISIT EXHIBITS

HALL B

**By Invitation*

11:00 a.m. C. WALTON LILLEHEI RESIDENT FORUM AWARD PRESENTATION

HALL C

SCIENTIFIC SESSION

Moderators: Timothy J. Gardner and Tirone E. David

37. The Fontan Operation: Early and Late Results in 135 Adult Patients

Harold M. Burkhart*, Joseph A. Dearani*, Francisco J. Puga, Douglas D.
Mair*, Carole A. Warnes*, Gordon K. Danielson; Rochester, MN

Discussant: Thomas L. Spray

OBJECTIVE: The Fontan operation, usually performed in children, is used for the treatment of functional single ventricle. We reviewed our experience with the modified Fontan procedure performed in the adult patient.

METHODS: Between October 1973 and May 2001, the Fontan operation was performed on 135 adult patients (73 men, 62 women). Median age was 23 yr (range 18-53 yr). Diagnoses included tricuspid atresia in 36 patient (27%), double-inlet left ventricle in 50 (37%), and complex lesions in 49 (36%). The majority (84%) of patients had at least one prior palliative procedure; the most

common procedures were a Blalock-Taussig shunt in 91 patients and cavopulmonary anastomosis in 29.

RESULTS: Operations included an atriopulmonary connection in 76 patients, lateral tunnel in 27, intra-atrial conduit in 15, right atrium-right ventricle in 10, extra-cardiac conduit in 3, and other in 4. Overall early mortality was 9.6%. Mortality was 6.4% for operations performed after 1980; this compares favorably with a 7% mortality for 102 pediatric patients operated on during the same time interval. Six of the 7 early deaths since 1980 occurred in the complex lesion group. Morbidity included prolonged pleural effusion in 16 patients, permanent pacemaker in 14, tamponade in 6 and stroke in 2. Mean follow-up was 8.9 yr with a maximum of 19.1 yr. Actuarial survival was 82%, 68%, and 60% at 5, 10 and 15 yr, respectively. The majority (88%) of patients were NYHA class I or II at follow-up.

CONCLUSION: The Fontan operation can be performed in selected adult patients with an early mortality comparable to younger patients. Early mortality is more likely with complex lesions. The majority of late survivors have a good quality of life.

**By Invitation*

38. Surgical RadioFrequency Ablation of Both Atria for Atrial Fibrillation - Results of a Multi-Centre Trial

Jai Raman*, Susumu Ishikawa*, John M. Power*; Heidelberg and Prahran,
Victoria, Australia

Discussant: James L. Cox

OBJECTIVE: Surgical Radio-frequency Ablation (RFA) has been used in the treatment of Atrial Fibrillation (AF) with varying degrees of success. We report on the results of RFA in Australia and New Zealand, which began in March 2000 as a multi-centre registry based study.

METHODS: One hundred and three patients in 17 hospitals underwent RFA. Mean age was 63 years (range 41 to 63). Preoperatively, patients had chronic AF (69%), frequent episodic AF (29%) and flutter (2%). Surgical procedures were performed in conjunction with mitral valve surgery (43%), CABG (16%), CABG and valve surgery (11%), double valve procedures (7%), aortic valve replacement (7%), and others (16%). RFA was performed using the Cobra probe (EPT-Boston Scientific Corp) creating a standard set of lesions. Each lesion was effected at 80 to 85°C for 2 minutes. Left sided lesions were endocardial in mitral procedures, but epicardial in CABG or aortic valve procedures. Right atrial (RA) epicardial lesions were common to all groups. The procedure added an average of 20 minutes to the operative time. RA test epicardial lesions were created in 12 patients with the heart beating.

RESULTS: There were no major complications related to RFA. Follow-up was complete, with 9 patients over 12 months and 44 over 3 months. Five patients were defibrillated into sinus rhythm within 3 months post-operatively. The post-operative AF free rate was 90% at 3 months, 95% at 6 months and 100% at 12 months. Transmural changes were seen in 83% of test epicardial lesions.

CONCLUSION: Surgical RFA of both atria with a standardized lesion set is safe and effective, in a multi-centre setting. Longer follow-up is required to delineate the long-term results of this technique.

11:45 a.m. ADDRESS BY HONORED SPEAKER -

Beyond Flat land

†Marc K. de Leval, London, England

HALL C

Introduced By: Timothy J. Gardner, Philadelphia, Pennsylvania

12:15 p.m. ADJOURN FOR LUNCH - VISIT EXHIBITS

HALL B

12:30 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

ROOM 15

†1973-74 Graham Fellow

TUESDAY AFTERNOON, MAY 7, 2002

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A -ADULT CARDIAC SURGERY
HALL C

Moderators: W. Randolph Chitwood Jr., and O. Wayne Isom

39. Repair versus Replacement for Degenerative Mitral Valve Disease and Coronary Artery Disease

Cristiano Faber*, A. Marc Gillinov, Eugene H. Blackstone, Ramon Diaz*,
Delos M. Cosgrove; Cleveland, OH *Discussant: Tirone E. David*

OBJECTIVE: To determine whether mitral valve repair is superior to mitral valve replacement in patients with degenerative mitral valve disease and coexisting coronary artery disease (CAD).

METHODS: From 1973 to 1999, 707 patients (mean age 67±9 years, 73% men) with degenerative mitral valve disease and CAD underwent combined coronary artery bypass grafting and either mitral valve repair (64%) or replacement (36%). Multivariable logistic analysis was used to identify predictors of valve repair, and analysis of risk factors for death was conducted in the multi-phase hazard function domain, with adjustment for selection by propensity score. Mean follow-up was 5.2±3.6 years, with a total of 3,568 patient years of follow-up.

RESULTS: Patients were more likely to receive mitral valve repair if they had isolated posterior chordal rupture (P<.0001), were younger (P=.001), or had surgery more recently (P<.0001); patients with bileaflet prolapse were more likely to have mitral valve replacement (P<.0001). Unadjusted survival at 30 days, 1, 5, and 10 years was 98%, 92%, 78%, and 60% after repair and 94%, 90%, 69%, and 40% after replacement. However, after adjusting for comorbid factors, extent and impact of CAD, and propensity score, the survival benefit of repair became evident beyond 3 years, particularly in patients receiving mechanical prostheses (P=.01).

CONCLUSION: In patients with degenerative mitral valve disease and CAD, mitral valve repair confers a survival advantage that becomes manifest 3 years after surgery.

**By Invitation*

40. Adverse Effect of Nitroglycerin on Coronary Artery Bypass Flow Early after Myocardial Revascularisation

Beat H. Walpoth*, Dirk Springe*, Beat Kipfer*, Pascal Berdat*, Peter

Neidhart*, Jürgens Robe*, Otto M. Hess*, Thierry Carrel*; Bern, Switzerland

Discussant: Andrew S. Wechsler

OBJECTIVE: Coronary bypass flow is dependent on graft material, anastomosis, perfusion pressure and distal coronary vascular resistance. Nitroglycerin (NTG) is often used postoperatively for coronary dilation and flow improvement. The aim of this study was to assess flow and pressure changes during NTG-infusion in arterial and venous bypass grafts.

METHODS: 30 patients (28 M; 2 F; 62±8 yrs) who underwent elective CABG (3.5±1.0 grafts/patient) were studied. After weaning from cardiopulmonary bypass, coronary bypass flow was measured in the ITA and in one venous bypass graft (SVG) using the transit time technique

(CardioMed). Increasing doses of NTG were infused: 16, 32, 64, 128 mcg/min i.v.. Mean arterial pressure (MAP) and flow were assessed simultaneously in both grafts.

RESULTS: Baseline flow was significantly higher in the SVG than in the ITA grafts ($p < 0.01$). With maximum NTG dose MAP decreased by 21%, ITA flow by 27% and SVG flow by 15%, respectively. Coronary resistance did not change significantly in ITA and SVG grafts, but resistance was higher in the ITA than SVG grafts. ITA and SVG graft flow were significantly correlated to perfusion pressure ($r = 0.6$ for ITA and $r = 0.5$ for SVG). There was a curvilinear dose-response curve for both grafts.

Variable	Baseline	Nitroglycerin	p (ANOVA)
MAP (mmHg)	76±9	60±9	<0.05
ITA flow (ml/min)	37±17	27±12	<0.02
SVG flow (ml/min)	67±37	57±34	ns
ITA resistance (mmHg/ml/min)	2.6±1.7	2.9±1.9	ns
SVG resistance (mmHg/ml/min)	1.8±1.5	1.6±1.1	ns

CONCLUSION: Despite higher flow rates in SVG than ITA-grafts a similar dose-dependent response to nitroglycerin was observed. Unchanged coronary vascular resistance and a linear pressure-flow relationship during NTG-infusion suggest the absence of autoregulation during CABG. Nitroglycerin reduces pressure and flow at the same extent and, thus, should be avoided in the early postoperative phase.

**By Invitation*

41. Mid-Term Results of the Australian Randomized Radial Artery Study

Brian F. Buxton*, Jai Raman*, Alexander Rosalion*, George Matalanis*,
David L. Hare*; Heidelberg, Victoria, Australia *Discussant: Stephen E. Fremes*

OBJECTIVE: There had been a resurgence in the use of the Radial Artery (RA) as a conduit in coronary artery bypass graft (CABG) surgery. We report on the mid-term results of a randomized study evaluating the fate of RA aorto-coronary grafts.

METHODS: Between July, 1996 and October, 2001 427 patients undergoing primary multi-vessel CABG on pump, were enrolled in the Australian Randomized Radial Artery Study. All patients received a Left Internal Thoracic Artery (LITA) graft to the left anterior descending coronary artery (CA). The next biggest graftable CA was randomized to receive a study graft (SG) based on stratification and randomization. Complementary grafts to other CA were with saphenous vein (SV). Group 1 comprising patients under 70 years of age or diabetics under 60, were randomized to receive RA or Free Right Internal Thoracic Artery (FRITA) as the SG. Patients over 70 or diabetics over 60 years made up group 2, and received either RA or SV as SG. Patients were randomized to undergo angiography at 6 months, 2, 5, 7 or 10 years post-operatively.

RESULTS: There were 2 early deaths (0.4%) and 14 late deaths (3.2%), of which 3 were sudden. Seventy angiograms have been performed electively (50 patients) and for symptoms (20), at a mean of 420 and 521 post-operative days, respectively. All LITA and SG were patent. Seven non-study SV grafts that were occluded, six of which had intervention. There was a significantly lower incidence of harvest site infections in the RA (3.3%) compared to SV (10%) ($p < 0.05$).

CONCLUSION: The mid-term patency of the FRITA.RA and SV were equally good when grafted electively to adequate sized CA. Harvest site complications were less frequent with RA compared to SV. Longer follow-up will help delineate the longevity of the RA.

**By Invitation*

42. Late Outcomes of Mitral Valve Repair for Floppy Mitral Valve. Implications for Asymptomatic Patients

Tirone E. David, Joan Ivanov*, Susan Armstrong*, Harry Rakowski*; Toronto, Ontario, Canada

Discussant: Michael A. Acker

OBJECTIVE: To assess the rationale of mitral valve (MV) repair for mitral regurgitation (MR) due to floppy MV disease.

METHODS: 488 patients had MV repair, and have been followed prospectively. The mean follow-up was 5.7±3.6 years and 99.5% complete.

RESULTS: Patients mean age was 58±13 years, and 49% were in functional classes 3 and 4. Kaplan-Meier estimates for freedom from morbid events were as shown in the table below. The Cox regression risk ratio (RR) and 95% C.I. for predictors of late mortality were: age/5 year increment RR 1.21 (1.0, 1.4); functional classes 3 and 4 RR 3.04 (1.2, 7.9); ejection fraction <40% RR 4.69 (2.2, 9.8); and hypertension RR 1.86 (1.0, 3.4). Age and hypertension were predictors of thromboembolism. There were no predictors for recurrent MR or reoperation. Bootstrap validation of the mortality model (200 Cox regressions in randomly selected datasets, 400 patients each) showed that age/5 year increment (mean risk ratio 1.24), functional classes 3 and 4 (mean risk ratio 3.74) and ejection fraction <40% (mean risk ratio 4.48) appeared in 50% or more of the models. The 10-year survival of asymptomatic patients was identical to the general population matched for age and gender (91 % vs. 91 %) whereas it was reduced among symptomatic patients (76% vs. 88%, p <0.001).

	5 year	10 year	15 year
Death	92%±1%	80%±1%	57%±3%
Cardiac death	97%±1%	95%±2%	84%±7%
Thromboembolism	94%±1%	88%±2%	73%±9%
Moderate or severe MR	99%±1%	91%±3%	85%±7%
Reoperation	96%±1%	93%±2%	91%±3%

CONCLUSION: Patients with MR due to floppy MV should be operated on before development of symptoms if the MV is reparable.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

HALL B

**By Invitation*

3:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A -ADULT CARDIAC SURGERY

HALL C

Moderators: W. Randolph Chitwood, Jr., and O. Wayne Isom

43. Influence of Systemic Temperature on Paralysis after Complex Thoracoabdominal and Descending Thoracic Aortic Operations

Lars G. Svensson, Lev Khitin*, Edward M. Nadolny*, Wendy A. Kimmel*;
Burlington, MA

Discussant: Joseph S. Coselli

OBJECTIVE: Determine influence of systemic temperature on the incidence of paralysis after aortic surgery.

METHODS: We examined outcome according to either mild passive hypothermia (N=24), active cooling to 29-32°C (N=79) or profound hypothermia (N=29) in 132 patients. Of these patients, 51.5% (68/133) underwent thoracoabdominal (TAA) repairs and 48.5% (64/132) had descending repairs. Aortic dissection was present in 50.8% (67/132), 31% (41/132) had leaks or rupture, 29.5% (39/132) were re-operations on the descending or TAA and 40% (53/132) had concurrent arch ± ascending repairs.

RESULTS: A total of 3.8% (5/132) of patients had permanent neurological deficits. Of these, 4.2% (1/24) were with mild cooling, 3.8% (3/79) with active cooling, and 3.6% (1/29) with profound hypothermia, nonetheless, a further 7 of patients who had mild cooling had reversible neurological deficits (p=0.004). The multivariable independent predictors of neurologic deficit were: aortic clamp time (p=0.011), moderate or profound hypothermia (p=0.004) and CSF drainage (p=0.049), although the latter two modalities were protective. Interestingly, extent of repair, namely descending versus Crawford TAA types I, II, III, or IV did not significantly influence neurological deficit rate (p=0.1). The 30-day mortality rate was 8.3% (11/132). The only multivariable predictor of death was acuity of surgery (emergency versus urgent versus elective, p=0.058).

CONCLUSION: Active cooling or profound hypothermia resulted in significantly lower transient neurological deficits compared to mild passive hypothermia. Thus, we recommend active cooling with atrio-femoral bypass and CSF drainage for most patients and use of profound hypothermia for complex concurrent arch ± ascending repairs and re-operations.

**By Invitation*

44. Use of Unfractionated Heparin in Patients with Heparin-Induced Thrombocytopenia Requiring Cardiac Surgery: Presentation of a Novel Management Algorithm

Bassem N. Mora*, Michael N. D'Ambra*, Edwin G. Avery, IV*, Arvind K. Agnihotri*, Gary W. Akins, Alan D. Hilgenberg, Thomas E. MacGillivray*, Joren C. Madsen, ⁴Gus J. Vlahakes, Jennifer D. Walker*, David F. Torchiana;
Boston, MA

*Discussant: Michel Carrier**

OBJECTIVE: Heparin-induced thrombocytopenia (HIT) poses a formidable challenge in anti-coagulation for cardiopulmonary bypass (CPB). Alprostadil (PGE1) and dipyridamole reversibly inhibit heparin-induced platelet aggregation in vitro. We hypothesized that pre-treatment with alprostadil and dipyridamole would allow heparin anticoagulation for CPB and prevent subsequent thromboembolic complications.

METHODS: Between 1996-2001, 47 HIT-positive cardiac surgical patients, diagnosed by ELISA, were pre-treated with intravenous alprostadil intraoperatively prior to full heparinization with unfractionated heparin; 72% (34/47) also received oral dipyridamole preoperatively.

RESULTS: All patients had recent exposure to unfractionated heparin, which was discontinued a mean of 23.5 days preoperatively. 38% (18/47) had a preoperative IABP, 55% (26/47) had an EF \leq 30%, 70% (33/47) were in recent CHF, 21% (10/47) underwent emergent operations, and 21% (10/47) had preoperative thrombosis. Procedures performed were: CABG, 49% (23/47); CABG/valve replacement, 15% (7/47); heart transplantation, 15% (7/47); VAD implantation, 9% (4/47); valve replacement, 4% (2/47); other procedures, 9% (4/47). Perioperative mortality was 10.6% (5/47, 1 each following CABG, CABG on IABP, heart transplantation, recurrent post-infarct VSD, and homograft aortic root replacement). Severe coagulopathy contributed to 4 of 5 deaths. Hemorrhagic complications predominated, with excessive intraoperative bleeding in 38% (18/47) and mediastinal reexploration in 17% (8/47). Average blood transfusions are shown below. Chest tube output was 1225 and 1909 ml in the first 12 and 24 hours, respectively. Thromboembolic complications were rare: 2 patients, both with preoperative IABP, had lower extremity ischemia; one patient suffered a stroke.

	Packed RBC (Unit)	FFP (Unit)	Platelets (6-pack)
Intraop mean \pm st dev	5.1 \pm 5.5	4.1 \pm 7.0	4.0 \pm 9.9
Intraop median	4	2	1
Intraop range	0 - 27	0 - 42	0 - 47
Postop mean \pm st dev	7.0 \pm 13.8	6.0 \pm 13.1	4.1 \pm 17.0
Postop median	3	2	0
Postop range	0 - 79	0 - 61	0 - 108

CONCLUSION: HIT-positive patients represent a high-risk group of patients undergoing cardiac surgery, due to preoperative comorbidities and postoperative hemorrhagic and thromboembolic complications. Using this novel algorithm of pretreatment with dipyridamole and alprostadil, full heparinization with unfractionated heparin for CPB was possible. Hemorrhagic complications were frequent, while anticipated postoperative thromboembolic complications associated with HIT were gratifyingly uncommon.

⁴1988-90 Research Scholar

**By Invitation*

45. Continuous Insulin Infusion Reduces CABG Mortality in Diabetic Patients

Anthony P. Furnary*, Gary L. Grunkemeier*, Kathryn J. Zerr*, Guangqiang

Gao*, H. Storm Floten*, Albert Starr; Portland, OR

*Discussant: Vivek Rao**

OBJECTIVE: Diabetes is a risk factor for mortality following CABG. Its relative risk may be related to the level of perioperative hyperglycemia. We hypothesized that strict glucose control with a continuous insulin infusion (CII) over the first 2 postoperative days would reduce hospital mortality.

METHODS: All diabetic CABG patients (n=2871) were treated with either subcutaneous insulin (SQI) (1987-1990 or with CII (1992-2001) for hyperglycemia. Predicted and observed hospital mortality were compared using both internal and external (STS 1996) multivariable risk models.

RESULTS: Observed mortality with CII was significantly lower than with SQI. Likewise, mean glucose was significantly lower in the CII group (table). Internal comparison: Multivariable analysis showed that CII was independently protective against mortality (OR=0.55, p<0.04). Conversely, hyperglycemia (OR=1.01/mg/dl, p<0.04), congestive failure, reoperation, mediastinitis, emergency surgery, renal failure, and older age increased the risk of death. External comparison: STS predicted risk for both groups was not significantly different. Observed mortality with CII was significantly less than predicted by the model. CII added an independently protective effect on mortality (OR=0.43, p=0.015) to the constellation of risk factors in the STS risk model.

Group	Mean Glucose (mg/dl)	Observed Mortality	O/E Ratio (95% C.I.)	Observed vs. Expected
SQI	201±1.2	4.7%(37/787)	1.04(0.54, 1.54)	P=NS
CII	175±0.6	1.7%(36/2084)	0.48(0.21, 0.76)	P<0.0001
CIII vs. SQI	P<0.0001	P<0.0001	P = 0.05	

CONCLUSION: CII eliminates the incremental increase in CABG hospital mortality due to diabetes. The protective effect of CII may stem from the effective metabolic utilization of excess glucose to increase ATP-mediated myocardial inotropy.

5:00 p.m. EXECUTIVE SESSION (Members Only)

HALL C

7:00 p.m. ATTENDEE RECEPTION

SMITHSONIAN NATIONAL MUSEUM OF AMERICAN HISTORY

(Separate Subscription)

**By Invitation*

TUESDAY AFTERNOON, MAY 7, 2002

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

ROOM 20

Moderators: G. Alexander Patterson and Douglas E. Wood

46. Refining the Staging System for Esophageal Cancer

Thomas W. Rice, Eugene H. Blackstone, Sudish C. Murthy*, Malcolm M.

DeCamp*, Lisa A. Rybicki*, David J. Adelstein*, John R. Goldblum*;

Cleveland, OH

Discussant: Carolyn E. Reed

OBJECTIVE: Cancer staging is dynamic, reflecting accrual of knowledge and experience in treatment. The objective was to refine the current staging system for esophageal cancer.

METHODS: From 1983 through 11/2000, 480 patients underwent esophagectomy without induction therapy. T, N, and M descriptors, number of metastatic nodes, cell type and differentiation were subjected to (1) survival tree analysis of prognosis (Recursive Partitioning Analysis), (2) multivariable Cox and hazard function analysis, and (3) multivariable analysis of residual misclassification risk.

RESULTS: Refinement of descriptors: Depth of tumor invasion (T) was refined for T1: T1a is cancer confined to the intramucosal layers, and T1b is cancer confined to the submucosa. Regional lymph node status (N) was refined by number of metastatic nodes: N1 is one or two, N2 is three nodes or more. Cell type and differentiation minimally refined staging, and only for advanced stages. Refinement of staging: AJCC guidelines constrain staging groupings based on characteristics of the tumor and its spread, homogeneity, and monotonic, distinctive survival. The Table presents the current staging system, the best constrained system based on analysis (Constrained), an alternative system based on freeing these constraints (Free), and 5-year survival for the latter.

Stage	Current	Constrained	Free	5-yr % survival
0	HGD	HGD	-	-
I	T1N0	T1aN0	HGD, T1aN0	84-92
II	A) T2/T3N0	A) T1bN0, T1aN1	A) T1bN0, T1aN1	56
	B) T1/T2N1	B) T2N0	B) T2N0	50
III	T3N1	A) T3N0, T1b/T2N1	A) T3N0, T1b/T2N1	25
	T4any	B) T3N1, T4N0	B) T3N1, T4N0	17
		C) T4N1, TanyN2		
IV	A) M1a	A) M1a	A) T4N1, TanyN2	3
	B) M1b	B) M1b	B) M1a/M1b	0

CONCLUSION: A revision of the current staging system should include redefinition of T1 and N descriptors. Staged grouping within constraints of the AJCC guidelines produces less accurate prognosis than a free assignment based on survival data.

**By Invitation*

47. Mediastinal Metastases from Testicular Nonseminomatous Germ Cell Tumors: Patterns of Dissemination and Predictors of Long-Term Survival with Surgery

Kenneth A. Kesler, Jo Ann Brooks*, Karen M. Rieger*, Naomi S. Fineberg*,

Lawrence H. Einhorn*, John W. Brown; Indianapolis, IN

Discussant: Joe B. Putnam

OBJECTIVE: The treatment of nonseminomatous germ cell tumors of testicular origin (NSGCT) with cisplatin-based chemotherapy (CT) followed by aggressive surgical resection of residual disease, which not infrequently is located in the lungs and/or mediastinum, represents one of the most successful models for multimodality cancer therapy. The purpose of the study was to evaluate variables which may influence survival following mediastinal dissection in patients with metastatic NSGCT.

METHODS: From 1981 to 2000, 421 patients presented to our institution for extirpation of residual lung and/or mediastinal disease following CT for metastatic NSGCT. We retrospectively reviewed 268 of these patients who required removal of residual mediastinal disease, undergoing a total of 455 thoracic surgical procedures (126 or 47% required more than one thoracic surgical procedure for complete removal of residual disease). The mean age was 26.817.6 years and the majority presented at diagnosis with elevated serum tumor markers (71% AFP, 81% BHCG). All patients had undergone first line cisplatin-based CT and 36% subsequently received second-line CT prior to surgery. Pathology of resected mediastinal disease was necrosis (15%), teratoma (59%), persistent nonseminomatous germ cell cancer (15%), and non-germ cell carcinomatous degeneration (11%). Fifteen variables were evaluated by univariate analyses and four variables, significant at $p < 0.05$, were subsequently entered into a Cox regression model (older age at diagnosis, elevated preoperative BHCG, removal of malignant pulmonary metastases, and mediastinal pathology).

RESULTS: There were two operative deaths (<1%). Overall five and ten-year survival rates were 86±2 and 74±4%, respectively. By multivariate analysis, survival was negatively influenced by older age at diagnosis ($p = .005$), elevated preoperative BHCG ($p = .028$), and mediastinal pathology ($p = .006$). The subset of patients who pathologically demonstrated tumor necrosis or teratoma had a 78.3±4% ten-year survival.

CONCLUSION: Patients with residual mediastinal disease following CT for NSGCT of testicular origin have good to excellent long-term survival rates which justify an aggressive surgical approach, frequently including multiple procedures. Improved survival is predicted by a younger age at diagnosis, a lower preoperative BHCG, and more benign mediastinal pathology.

**By Invitation*

48. Feasibility and Safety of Extra-Anatomic Transbronchial Decompression for Emphysema

Erino A. Rendina, Tiziano De Giacomo*, Federico Venuta*, Giorgio Furio Coloni*, Bryan F. Meyers*, G. Alexander Patterson, Joel D. Cooper; Rome Italy; St. Louis, MO *Discussant: Scott J. Swanson**

OBJECTIVE: We have proposed that direct passages created between pulmonary parenchyma and large airways could take advantage of the extensive collateral ventilation present in emphysematous lungs to provide improvement in expiratory flow and respiratory mechanics. A crucial step in the safe performance of this procedure is to create passages through the airway wall into lung parenchyma while avoiding injury to adjacent blood vessels.

METHODS: The procedure consists of selecting a target site bronchoscopically, use of a doppler catheter (Broncus Technologies, Inc.) to detect and avoid peribronchial blood vessels, and creation of a passage through the airway wall with a radio frequency catheter (Broncus Technologies, Inc.). Ten patients were treated during prescheduled lobectomy for neoplasm. The procedure was done after thoracotomy, immediately before resection, and was confined to tissue identified for removal. To extend the procedure to emphysematous patients the procedure was subsequently performed in 3 patients undergoing lung transplantation for emphysema.

RESULTS: Twenty-nine passages (one to five per subject) were created in the lobectomy patients. Ten passages were created (three to four per subject) in the transplant patients. There were two instances of mild bleeding in the lobectomy patients and no bleeding in the transplant patients. Both instances were treated with suction and topical application of epinephrine and resolved without incident.

CONCLUSION: The results of this study confirm that passages can be made safely through the airways of human subjects. These clinical results support further investigation of the efficacy of the Transbronchial Decompression procedure in emphysema patients.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

HALL B

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - GENERAL THORACIC SURGERY

ROOM 20

Moderators: G. Alexander Patterson and Douglas E. Wood

49. A 25-Year Experience Supporting Transthoracic Paraesophageal Hiatal Hernia Repair in 247 Patients

Himanshu J. Patel*, Bethany B. Tan*, John Yee*, Mark B. Orringer, Mark D. Iannettoni; Ann Arbor, MI

Discussant: James D. Luketich

OBJECTIVE: The repair of paraesophageal hiatal hernias (PHH) is one the most technically challenging operations in thoracic surgery. With the recent enthusiasm for laparoscopic hiatal hernia repairs, the importance of establishing "benchmark" data based on the results of traditional open operations is evident. This report reviews a 25-year experience with 247 operations for the repair of PHH.

METHODS: A retrospective review of 247 consecutive primary transthoracic repairs for PHH from March 1977 to September 2001 was performed. 231 (94%) were type III, and 16 (6%) were type IV. There were 170 (69%) women and 77 (31%) men, whose average age was 65±12 years. Presenting symptoms were: abdominal/chest pain in 121 (49%); dysphagia in 60 (24%); reflux in 121 (49%); bleeding/anemia in 56 (23%). Repairs included: Collis-Nissen in 238 (96%); Collis-Belsey in 1 (0.5%); and a Nissen in 8 (3%).

RESULTS: There were 4 (1.6%) postoperative deaths. Mean hospital stay was 8 days. Mean follow was 28 months (range 1-221). Good to excellent results are reported in 181 patients (73%). Sixty patients (24%), (17 of whom had dysphagia preoperatively) required dilatations postoperatively for dysphagia. Twenty-one patients (8%) developed anatomic recurrence of their hernias. Ten patients (6.6%) required re-operation for recurrent hernia (8) or persistent dysphagia (2).

CONCLUSION: These data demonstrate the excellent results that can be obtained with open transthoracic repair of paraesophageal hiatal hernias and provide a standard against which minimally invasive approaches must be compared.

**By Invitation*

50. Radiofrequency Ablation of Primary and Metastatic Lung Tumors: Analysis of an Ablate and Resection Study

Stephen Yang*, Richard Whyte, Fred Askin*, Sharon Thomsen*, Pheroze Tamboli*, Gerald Berry*, Carmen Roig*, Joe Putnam; Baltimore, MD; Stanford, CA; Austin and Houston, TX

Discussant:

OBJECTIVE: Chemo- and radiation therapy provide limited responses to advanced lung neoplasms. Alternative local therapies are needed. Radiofrequency ablation (RFA) of liver lesions is an accepted treatment option; experience is limited for lung tumors. Therefore, the purpose of this study was to prospectively evaluate the feasibility, safety and efficacy of RFA on malignant lung neoplasms.

METHODS: This was a study conducted at three institutions ablating primary or metastatic lung tumors in patients undergoing thoracotomy for resectable lesions < 3 cm. After informed consent, malignancy was confirmed intraoperatively by needle biopsy (if none preoperatively). Following RFA, the tumors were resected by lobectomy or lesser resection. Extent of necrosis was examined by nicotinamide adenine dinucleotide stains.

RESULTS: Of 15 patients (six primary, nine metastatic), 13 (14 tumors) were treated by RFA. Two were excluded due to extensive calcification (metastatic sarcoma) and minor bleeding at the RFA site (primary tumor). Although median tumor kill was 70%, seven patients had 100% ablation.

The ablation margin extended beyond the tumor in 13 specimens. Despite certain study limitations (no image guidance for accurate intratumoral array deployment; complete tissue necrosis occurs 24-72 hours after thermal injury), 100% tumor kill was achieved in five of the last six cases.

CONCLUSION: RFA for lung tumors is feasible and safe. Complete ablation occurred in 54% of patients, most at study conclusion. Partial ablation is attributed to suboptimal device placement and "learning curve" issues. Though currently impractical to use by a surgical approach, RFA of lung malignancies will be more applicable for image-guided percutaneous techniques.

**By Invitation*

51. The Efficacy of Re-do Laparoscopic Fundoplication for Persistent/Recurrent Symptoms After Anti-Reflux Surgery

Woodrow W. Yeane*, Pavlos K. Pappas*, Fernando D. Hayetien*, Rodney

J. Landreneau, Richard H. Maley, Jr.*, Philip F. Caushaj*, Daniel J. Gagne*,

Robert J. Keenan; Pittsburgh, PA

Discussant: Claude Deschamps

OBJECTIVE: To determine the safety and effectiveness of re-do laparoscopic fundoplication (LF) in patients with persistent or recurrent symptoms.

METHODS: We retrospectively reviewed our experience with re-do LF. The symptom requiring re-operation, technical feasibility and success of performing a re-do LF, and operative findings were examined. Major symptoms were heartburn, dysphagia or gas/bloat. Using visual analog scores the outcome of re-do LF was graded as 0-worse, 1-no to mild improvement, 2-moderate improvement, 3-significant improvement. Statistical significance was determined by Chi-square analysis.

RESULTS: 50 re-do patients were identified. Of 805 pts undergoing LF at our institutions, 36 (4.5%) required re-operation. 14 pts, whose initial procedure was done elsewhere, underwent re-do LF; 7 initially had open procedures. Indications were heartburn (n=25), dysphagia (n=15) and gas/bloat (n=10). Average time from initial to re-do surgery was 25.1 ±41.5 months (range: 12.3 months gas/bloat - 31.8 months heartburn). 47/50 (94%) operations were completed laparoscopically including 4/7 pts who had previous open fundoplications. Length of stay averaged 2.6±2.0 days. Technical problems (disrupted or slipped wrap) were found in 68% of pts with heartburn and 40% with dysphagia. No failure was found in 9/10 gas/bloat pts. Symptom scores were obtained 20.5±1.3-4 months after re-do surgery. All symptom categories experienced significant improvement (table) with heartburn patients obtaining the greatest relief.

	SYMPTOM		
	Heartburn	Dysphagia	Gas/Bloat
Chi-Square	0.0001	0.004	0.04
Post-op score 2-3 (%)	83%	62%	44%

CONCLUSION: Significant improvement after re-do LF can be achieved for all symptom categories. Re-do LF can safely and effectively be performed even with an open initial operation. Technical failures are most frequently found in heartburn pts.

5:00 p.m. EXECUTIVE SESSION (Members Only)

HALL C

7:00 p.m. ATTENDEE RECEPTION

SMITHSONIAN NATIONAL MUSEUM OF AMERICAN HISTORY

(Separate Subscription)

**By Invitation*

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

ROOM 31

Moderators: Roger B. B Mee and Richard A. Jonas

52. Primary Thoracoscopic Treatment of Empyema in Children

Gordon A. Cohen*, Vibeke E. Hjortdal*, Marco Ricci*, Colin Wallis*. Robert Dinwiddie*, Martin J. Elliott*, ⁵Marc R. de Leval; London, United Kingdom

Discussant: Douglas E. Wood

OBJECTIVE: The optimal treatment of pediatric empyema remains controversial. The objective of this study is to compare the use of conventional treatment (CT) versus primary thoraco-scopic drainage and decortication (TDD) in children with empyema.

METHODS: CT consisted of chest drain insertion under general anesthesia plus intravenous (IV) antibiotics. TDD consisted of primary thoracoscopic drainage and decortication plus IV antibiotics. The clinical course of 54 CT patients treated between 1989 and 1997 was compared to 21 TDD patients treated between September 2000 and September 2001.

RESULTS: Results are summarized in Table 1. Length of antibiotic therapy, chest tube drainage and hospital stay were significantly shorter in the TDD group ($p < 0.001$). In addition, whereas no patients required open thoracotomy and decortication in the TDD group, this was necessary in 21/54 (39%) patients in the CT group

		TDD(n=21)	CT (n=54)	
	Mean age (months)	70.7±14.6	70.8±46.8	
Invasive interventions	Primary chest drain	0	47/54 (87%)	
	VATS	21/21 (100%)	0	
	Thoracotomy	0	21/54(39%)	
	Procedure/patient	1.0	1.26	
Length of IV antibiotics (days)	Mean	7.6±1.2	18.2±7.5	p<0.001
Length of chest tube drainage (days)	Mean	4.0±0.5	10.2±6.1	p<0.001
Length of hospital stay (days)	Mean	7.4±0.8	15.4±7.4	p<0.001

CONCLUSION: Although the two groups were not prospectively randomized and they were treated in different time periods, the results of this study support the use of thoracoscopic surgery as the primary therapeutic modality in children presenting with pleural empyema. This strategy appears to offer significant benefits over conventional treatment in terms of duration of treatment and the need for more invasive surgery.

⁵1973-74 Graham Fellow

**By Invitation*

53. Is Surgery Still Indicated in Recurrent Aortic Arch Obstruction

Joy Zoghbi*, ⁶Alain Serraf*, Siamak Mohammadi*, Francois Lacour Gayet,

Emre Belli*, Regine Roussin*, Claude Planche; Paris, France; Hamburg,

Germany

Discussant: Carl L. Backer

OBJECTIVE: Although balloon dilatation has become the standard treatment for recurrent aortic arch obstruction (RAAO), patients failing or not amenable to balloon angioplasty should be managed surgically. We analyse the different approaches and results of the reoperations of the aortic arch.

METHODS: Since 1983, 63 patients underwent reoperation for recoarctation, 17 had associated arch hypoplasia, 8 had unsuccessful balloon dilatation. Mean age at reoperation was $56,6 \pm 53$ months and mean delay after repair $45,5 \pm 61$ months. Mean gradient before reoperation was $56 \pm 18,8$ mmHg. Indication for reoperation was systemic hypertension in 41 cases, heart failure in 9 patients and major gradient for 13. Surgical approach was oriented towards an anatomical reconstruction of the aortic arch with resection of the recoarctation. It could be performed through a left thoracotomy with extended end to end anastomosis in 34 pts, subclavian flap in 9, conduit insertion in 7 and patch enlargement in 7. More recently, an anterior approach with cardio pulmonary bypass and selective cerebral perfusion was applied in 6 pts with proximal aortic arch hypoplasia to patch enlarge all the aortic arch.

RESULTS: There was one early and no late deaths. None of the patients disclosed any neurological impairment. At a mean follow up of $11,8 \pm 3,3$ y, the mean gradient was of $14,2 \pm 8,4$ mmHg. Systemic hypertension normalized in all but 5 patients. One patient was reoperated for recoarctation.

CONCLUSION: Reoperation for RAAO can be performed safely with low rates of mortality and morbidity. This approach should be still considered versus balloon angioplasty

⁶1993-1994 Graham Fellow

**By Invitation*

54. Combined Arterial Switch and Senning Operation for Congenitally Corrected Transposition of the Great Arteries: Patient Selection and Early Results

Eric J. Devaney*, John R. Charpie*, Richard G. Ohye*, Edward L. Bove; Ann Arbor, MI

Discussant: Roger B. B. Mee

OBJECTIVE: The combined arterial switch and Senning (double switch) operation may improve the long term survival of patients with congenitally corrected transposition of the great arteries (CCTGA) by establishing atrioventricular and ventriculoarterial concordance. In this report, we review patient selection and early results following double switch operation for CCTGA.

METHODS: Since 1993, we have evaluated 34 patients with CCTGA who had two ventricles of adequate size and no valvar pulmonary stenosis and who were potential candidates for a double switch operation. Twelve patients were either clinically well or not yet referred for further treatment. The hospital records of the remaining 22 patients were reviewed retrospectively and follow up data was obtained through clinic notes and contact with the patients, their families, or the treating cardiologists.

RESULTS: The 22 patients were judged to be candidates for double switch due to right ventricular dysfunction and or tricuspid regurgitation (n=15) or associated uncorrected defects (n=7). To date, sixteen patients have undergone double switch operation. There has been no early or late mortality, although one patient required cardiac transplantation for progressive left ventricular failure. Ventricular function and tricuspid regurgitation ultimately remained stable or improved in all patients, with the exception of the patient requiring transplantation. All patients are alive at a mean follow up of 36 months (range, 1 month to 8 years).

CONCLUSION: CCTGA with a normal pulmonary valve and two adequate ventricles can be managed with combined arterial switch and Senning operation with excellent early results. Late follow up will be necessary to determine whether this management strategy provides a survival advantage for these patients.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

HALL B

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - CONGENITAL HEART DISEASE

ROOM 31

Moderators: Roger B. B. Mee, and Richard A. Jonas

55. Induction of Pulmonary Angiogenesis by Adenoviral Mediated Gene Transfer of Vascular Endothelial Growth Factor in a Fetal Model of Pulmonary Artery Hypoplasia.

Virginia Lambert*, Patricia Lemarchand*, RenÃ© Michel*, Guy-Michel Mazmanan*, Elisabeth Dulmet*, Andre Capoerou.x*, Philippe Herve*, Claude Planche, ⁶Alain Serraf*; Paris, France; Montreal, Quebec, Canada

Discussant: Irving L. Kron

OBJECTIVE: Gene transfer of vascular endothelial growth factor(VEGF)induces myocardial and peripheral angiogenesis. The effects of transbronchial adenoviral mediated gene transfer of VEGF on pulmonary arteries (PA) angiogenesis and growth in a fetal model of PA hypoplasia are unknown.

METHODS: Thirteen fetal lambs had left PA banding in utero at 106 days of gestation. Following birth, 3 groups were divided: VEGF group (n=5) received an adenoviral vector encoding for human VEGF 165, Betagal group (n=4) an adenoviral vector encoding for Galactosidase A and Control group (n=4) had neither gene nor virus. Viral suspensions were selectively instilled at 6.5 days of age in the left bronchus. A fourth group (n=5) of non operated lambs were considered as normal. Euthanasia was performed at 30 days of age. The lungs were fixed in 10% buffered formalin instilled at 20 mmHg for histomorphometric evaluation. All groups were compared with ANOVA test and paired test was used to compare right and left lung in each animal.

RESULTS: Gene transfer was assessed by blue staining of left lung obtained with XGal reagent in an additional experiment. Left PA and lung hypoplasia present in all operated groups were significantly less pronounced in VEGF group ($p<.05$). Left lung arterial density was higher in VEGF group versus others ($p<.05$). The percentage of parenchyma of left lung was lower in Betagal group versus normal and control groups ($p<.05$), partial recovery was found in VEGF group.

CONCLUSION: Transbronchial adenoviral mediated VEGF gene transfer induces PA angiogenesis and proximal PA growth in this model.

1993-1994 AATS Graham Fellow

**By Invitation*

56. Myocardial Apoptosis is Triggered by Cardioplegic Arrest in the Neonatal Lamb

James M. Hammel*, Timothy L. Van Natta*, Wei-Gen Li*, Thomas D. Scholz*, Jeffrey L. Segar*, Douglas M. Behrendt, Christopher A. Caldarone*; Iowa City, IA

Discussant: Ross M. Ungerleider

OBJECTIVE: Cardioplegic arrest in the neonate is associated with a period of myocardial depression typically occurring 8-12 hours after separation from cardiopulmonary bypass. Because previous reports demonstrate that the neonatal heart is in a pro-apoptotic state, we hypothesize that apoptosis-related pathways are triggered after Cardioplegic arrest in neonatal myocardium and can, therefore, contribute to postoperative myocardial depression.

METHODS: Hypothermic cardiopulmonary bypass was established in 6- to 8-day-old lambs (n=4, non-operated controls n=5). Cold crystalloid cardioplegia was administered at 20 minute intervals. Total crossclamp time: 70 minutes; total bypass time: 90 minutes. After a six hour recovery period, hearts were excised and myocardium examined using Tdt-mediated dUTP nick-end labeling (TUNEL), radiolabeled DNA electrophoresis, fluorimetric caspase 3 activity assay, Bcl-2 protein expression (Western), and mRNA microarray.

RESULTS: 2.58% of cardiomyocytes were TUNEL-positive after Cardioplegic arrest compared to 0.03% in controls ($p<0.05$). Electrophoresis of DNA revealed specific internucleosomal cleavage

typical of apoptosis (DNA laddering) in all bypass animals which was absent among controls. Caspase 3 activity (an apoptosis-specific protease) increased 1.7-fold ($p=0.076$). Bcl-2 protein (anti-apoptotic), Bcl-x mRNA (anti-apoptotic), and Bax mRNA (pro-apoptotic) were upregulated.

CONCLUSION: In a clinically relevant model of neonatal cardiopulmonary bypass and Cardioplegic arrest, apoptotic cell death is present and apoptosis-related regulatory pathways are activated within six hours after reperfusion. Because the effector mechanisms of apoptosis involve mitochondrial dysfunction, the degree of postoperative myocardial depression likely exceeds the fraction of cardiomyocytes undergoing apoptotic cell death. Therefore, activation of apoptosis-related pathways may contribute significantly to postoperative myocardial dysfunction in the neonate.

**By Invitation*

57. Ventricular Energetics After Fontan Operation: Contractility-Afterload Mismatch

Gabor Szabo*, Terezia Andrasi*, Christian F. Vahl*, Siegfried Hagl*;

Heidelberg, Germany; Budapest, Hungary

Discussant: ¹Marc R de Leval

OBJECTIVE: Both pulmonary and systemic circulation must be maintained by a single pump in Fontan circulation. This unique property may be related to impaired hemodynamics and decreased exercise tolerance often observed in these patients. The present study investigated cardiac performance after Fontan operation by using ventricular-vascular coupling framework analysis.

METHODS: In 7 anesthetized open-chest dogs, left ventricular hemodynamic variables were measured by a combined pressure-volume-conductance catheter. Additionally, aortic flow and pressure was recorded continuously. Ventricular contractility was quantified by the load-independent slope of the end-systolic pressure-volume relation (Ees). Arterial system properties were quantified by the end systolic pressure-stroke volume ratio (Ea). The coupling between left ventricle and arterial system was expressed by the Ea/Ees ratio. Additionally external (stroke) work (SW), total mechanical energy (TE) and mechanical efficiency ($Eff=SW/TE$) were calculated. Systemic and pulmonary impedance was determined by Fourier analysis. Fontan circulation was established by using cavopulmonary anastomosis.

RESULTS: Ees (4.7 ± 0.6 vs. 7.4 ± 0.8 mmHg/ml, $p<0.05$) was decreased and the Ea (4.2 ± 0.4 vs. 3.7 ± 0.5 mmHg/ml, n.s.) was increased. The Ea/Ees ratio was significantly higher after Fontan circulation (0.89 ± 0.05 vs. 0.50 ± 0.05 , $p<0.05$). Simultaneously, SW (1865 ± 276 vs. 1087 ± 1243 mmHgxml, $p<0.05$) and Eff (0.82 ± 0.09 vs. 0.56 ± 0.05) were significantly reduced.

CONCLUSION: Fontan circulation leads to contractility-afterload mismatch by increased impedance due to additional connection of the pulmonary vascular bed to the systemic vasculature and by deterioration of myocardial contractility. The increased ventriculo-arterial coupling ratio and reduced mechanical efficiency predict limited cardiac functional reserve after Fontan operation.

⁷1973-74 Graham Fellow

**By Invitation*

58. Cardioprotective Effects and the Mechanisms of Terminal Warm Blood Cardioplegia in Pediatric Cardiac Surgery

Yoshiya Toyoda*, Masahiro Yamaguchi*, Naoki Yoshimura*, Shigeteru Oka*,
Yutaka Okita; Kobe, Japan

Discussant: Bradley S. Allen

OBJECTIVE: Terminal warm blood cardioplegia (TWBCP) has been shown to enhance myocardial protection in coronary artery bypass surgery. However, the cardioprotective effects and the mechanism of TWBCP in pediatric cardiac surgery were unknown.

METHODS: One hundred and three consecutive patients undergoing open heart surgery for congenital heart diseases including ventricular septal defect, atrial septal defect, tetralogy of Fallot, and pulmonary atresia were prospectively randomized to one of two groups. In the control group (C: n=52), myocardial protection was achieved using intermittent hyperkalemic (K⁺=18mEq) antegrade cold (4 °C) blood cardioplegic solution. In the TWBCP group (T: n=51), this was supplemented with a 3-minute exposure to hyperkalemic warm (35 °C) blood cardioplegia prior to declamping the aorta. Myocardial energy metabolism and tissue injury was monitored using a catheter placed in the coronary sinus and aortic root.

RESULTS: There were no significant differences between the two groups in age (5.6±0.5 in T vs 5.5±0.6 in C), body weight (19.8±1.7kg in T vs 17.2±1.4kg in C), percentage of cyanotic heart diseases (45.1 % in T and 44.2% in C), number of patients required right ventriculotomy (39±7% in T vs 33±7% in C), cardiopulmonary bypass time (177±9 min in T vs 194±12 min in C), aortic cross-clamp time (82±5 min in T vs 83±6 min in C), minimal rectal temperature (28.1 ±0.3 °C in T vs 27.4±0.3 °C in C), and minimal myocardial temperature (9.6±0.7 °C in T vs 9.6±0.6 °C in C). Electrical defibrillation was required to obtain self beat in 20.0% in T group, which was significantly (p=0.033) lower than 38.5% of C group. The lactate extraction rate at 60-minute reperfusion was significantly (p=0.008) lower in C group (-3.3±2.4%) than T group (9.0±2.8%). The post-reperfusion values of troponin I (7.6±0.9 vs 11.2±1.4ng/mL at 6 hours) and fatty acid binding protein (137±28 vs 240±30 ng/mL at 2 hours; 88±19 vs 162±26 ng/mL at 3 hours) were significantly lower in T (p<0.05 vs C).

CONCLUSION: TWBCP reduces arrhythmogenesis during early reperfusion, accelerates recovery of myocardial energy metabolism and provides less myocardial damage in pediatric cardiac surgery.

5:00 p.m. EXECUTIVE SESSION (Members Only)
HALL C

7:00 p.m. ATTENDEE RECEPTION

SMITHSONIAN NATIONAL MUSEUM OF AMERICAN HISTORY

(Separate Subscription)

**By Invitation*

WEDNESDAY MORNING, MAY 8, 2002

7:00 a.m. EMERGING TECHNOLOGIES AND TECHNIQUES

HALL C

Moderators: Michael J. Mack and Freidrich W. Mohr

T1 Clinical and Six-Month Angiographic Evaluation of Coronary Arterial Graft Interrupted Anastomoses using a Self-Closing Clip Device - A Multi-Center Prospective Clinical Trial

Randall K. Wolf, Edwin L. Alderman*, Michael P. Caskey*, Allen R. Raczkowski*, Mercedes K. Dullum*, Dwight C. Lundell*, Arthur C. Hill*, Nan Wang*, Michael A. Daniel*; Columbus, OH.; Stanford and Palo Alto, CA; Phoenix and Mesa, AZ. Washington, D.C.

Discussant: Erik W. L. Jansen

OBJECTIVE: To evaluate the safety and effectiveness of a self-closing surgical clip (Coalescent Surgical U-CLIP™ anastomotic device) using an interrupted technique in LITA to LAD bypass grafting.

METHODS: Eighty-one patients were enrolled (February, 2000 and August, 2001), into a prospective, multi-center, trial. LITA to LAD anastomoses were performed in 62 OPCAB (76.5%), 13 conventional CABG (16%) and 6 MIDCAB (7.5%) procedures. Angiograms (21-349 days; mean 196 days) were completed on 52 patients (64%). Qualitative and quantitative angiographic assessment was performed by an independent core laboratory.

RESULTS: The U-CLIP was used for 81 LITA to LAD interrupted anastomoses without the requirement for knot tying or primary suture management. Mean LITA to LAD anastomosis time was 12.5 minutes (3-35 minutes). There was one peri-operative and one late death (both non-cardiac) and one re-exploration for bleeding unrelated to the anastomotic sites. FitzGibbon Grades were; A (N=49; 94.2%), B (N=3; 5.8%) including 1 kinked LITA and Grade 0 (Occluded; N=0). Quantitative analysis (N=52) showed mean lumen diameters of LITA proximal to the anastomosis of 2.0mm, at anastomosis of 1.9mm, and in the LAD distal to the anastomosis of 1.7mm. The average ratio of the anastomosis to the LAD diameter was 1.13 (0.45 to 1.93). Anastomotic stenosis as a percentage of average LITA/LAD diameter was 7.5%. This result compares favorably with the 23 and 24% reported in the Patency Outcomes Economics (POEM) study.

CONCLUSION: The interrupted technique, facilitated by a self-closing anastomotic clip, yields favorable 6-month angiographic results when compared with other published studies.

**By Invitation*

T2 Automated Distal Coronary Bypass Using a Novel Magnetic Coupler

⁷David H. Adams, Farzan Filsoufi*, ⁸Lishan Aklog*, R. Saeid Farivar*, Curtis A. Anderson*, Raymond H. Chen*, Samuel Lichtenstein*, Ji Zhang*; Boston, MA; Vancouver, British Columbia, Canada

*Discussant: Anno Diegeler**

OBJECTIVE: To assess the feasibility of performing distal coronary artery bypass anastomoses using a novel magnetic coupling device.

METHODS: From May 2000 to April 2001, 39 pigs (35-60 kg) underwent side to side coronary artery bypass grafting on a beating heart without the use of stabilizers, shunts, or perfusion bridges. Seventeen right internal mammary artery (IMA) to right coronary artery and 22 left IMA to left anterior descending artery anastomoses were created using the magnetic vascular positioner system (MVP™). This device includes two pairs of elliptical magnets which form anastomotic docking ports on the graft and the target vessels respectively. The automated anastomosis is created by approximating the two docking ports which magnetically couple. Anastomotic patency was evaluated by angiography during the first post-operative week and at one month. Histologic studies were performed at selected time points including 1, 3, and 6 months.

RESULTS: The self-adherent and self-aligning properties of the implants facilitated immediate and secure distal anastomoses in all animals. Five non-device related deaths occurred postoperatively. Angiography revealed 100% (35/35) patency at one week and 97% (33/34) patency at one month. Histologic studies as late as 6 months demonstrated neointimal coverage of the magnets, without any significant luminal obstruction.

CONCLUSION: The MVP™ anastomotic system employs magnetic force to create rapid and secure distal coronary artery anastomoses, which may facilitate minimally invasive and totally endoscopic coronary artery bypass surgery.

⁷1992-94 Research Scholar

⁸1998-99 International Traveling Fellow

*By Invitation

T3 Transcoronary Implantation of Bone Marrow Stromal Cells Ameliorates Cardiac Function After Myocardial Infarction

Takayuki Saito*, Jin-Qiang Kuang*, Charles Lin*, Ray C-J Chiu; Montreal, Quebec, Canada

Discussant: Todd K. Rosengart

OBJECTIVE: Bone marrow stromal cells (MSCs) are capable of differentiating into cardiomyogenic cells. We tested the hypothesis that transcoronary implantation of MSCs may regenerate infarcted myocardium and restore cardiac function.

METHODS: Isolated MSCs from the isogenic donor rats were transfected with LacZ reporter gene for cell labeling. In order to stimulate cardiomyogenic differentiation, the MSCs were treated with 5-azacytidine before implantation. Two weeks after left coronary ligation, these cells (1x10⁶ in 150 µL) were infused into the briefly distally occluded ascending aorta of the recipient rats (n=14). Control animals were infused with cell-free medium (n=14). Cardiac function was evaluated by echocardiography at pre-, 4, and 8 weeks post-implantation. The hearts were then immunohistochemically studied to identify phenotypic changes of implanted MSCs.

RESULTS: Immediately after cell infusion, the MSCs were trapped within coronary vessels in both infarcted and noninfarcted areas. However, after 8 weeks, most of the cells were identified within myocardial scar expressing cardiomyocyte specific proteins, troponin I-C and sarcomeric myosin heavy chain. Some MSCs expressed alpha-smooth muscle cell actin and were integrated in

vessels. Two-way repeated-measures ANOVA revealed significant improvement in FS, LVEDd, and LVESd ($p=0.0214$, 0.0078 , 0.0012 , respectively) in the rats infused with MSCs.

CONCLUSION: Although MSCs had been reported to improve cardiac function when injected directly into the myocardial scar, this study demonstrated for the first time that MSCs can simply be delivered via the coronary artery, as they are capable of targeted migration and differentiation into cardiomyocytes in the scar tissue to improve cardiac function.

**By Invitation*

T4 In Vivo Resistance to Calcification of Syner Graft Tissue Engineered Heart Valve Crafts

Ronald C. Elkins, Steven Goldstein*, Steven P. Walsh*, Kirby S. Black*;
Oklahoma City, OK; Kennesaw, GA

Discussant: John E. Mayer

OBJECTIVE: Calcification of bioprosthetic heart valves is a significant cause of graft failure. While post-fixation calcification mitigation treatments may be effective, these valves are non-viable and are incapable of recellularization. Because tissue calcification may stem from cell remnants within tissues, we evaluated the calcification of decellularized/non-glutaraldehyde fixed tissue grafts, which are essentially free of histologically demonstrable cell content.

METHODS: Porcine composite aortic valves ($n=11$) or pulmonary valves ($n=13$) comprised of decellularized tissue were implanted in the sheep right and left ventricular outflow tracts, respectively. Tissues were explanted between 90 and 164 days post-surgery and were analyzed for calcium content by atomic absorption spectrometry or calcium deposition by standard histologic techniques.

RESULTS: At 150 days, aortic valve leaflet calcium content was 2.48 ± 5 mg/g dry weight, not different from pre-implantation tissue. Similarly, pulmonary valve leaflet calcium ranged between 0.7 - 5.6 mg/g. Calcification of pulmonary artery was localized with unmineralized regions of conduit displaying in vivo recellularization with interstitial cells. Conduit calcium content was lower than the ovine aortic control tissue (121 ± 13 mg/g) in both the decellularized aorta (82 ± 28 mg/g) and in discrete regions of decellularized pulmonary artery (57 mg/g at 164 days post-implantation).

CONCLUSION: Tissue engineering is a powerful approach to improvement of valve durability. The combination of lower calcium and tissue viability observed with these decellularized/unfixed tissue matrices provides a potential solution to calcific structural valve deterioration.

**By Invitation*

T5 Global Surgical Experience with the Acorn Cardiac Support Device

⁹Mehmet C. Oz, Wolfgang F. Konertz*, Franz X. Klebef, Friedrich W. Mohr, Jan F. Gummert*, Jorg Ostermeyer*, Michael Lass*, Jai Raman*, Michael A. Acker, Nicholas Smedira*; New York, NY; Berlin, Leipzig, and Hamburg, Germany

OBJECTIVE: Remodeling the dilated left ventricle (LV) associated with heart failure (HF) is an attractive surgical option. Providing end-diastolic support with an innovative mesh-like cardiac support device (CSD) has been demonstrated in animal models to reduce mechanical stress, improve cardiac function, and reverse the remodeling process with no safety issues.

METHODS: The CSD (Acorn CorCap) has been implanted worldwide in 91 patients with dilated cardiomyopathy (25 patients with the CSD only; 66 patients with concomitant cardiac surgery). The CSD is positioned around the ventricles and custom fit to produce a slight reduction in LV end-diastolic dimension (LVEDD).

RESULTS: Average CSD implant time was 25 minutes (15 to 35 min). Mean intra-operative LVEDD reduction was $3.6 \pm 1.4\%$. There were no device related intraoperative complications. There were 9 early deaths and 7 late deaths, an overall crude 2-year mortality of 18%. There was no difference between CSD only or concomitant surgery patients. Long-term follow-up is available for 49 patients (34 patients with CSD plus concomitant surgery, 15 CSD only). At implant, 11 patients were in NYHA Class II, 34 in III and 4 in IV. No adverse events were device related with no evidence of constrictive disease. Six and twelve-month follow-up showed a decreased LVEDD and improved EF and NYHA Class (see Table). CSD-only patients showed similar improvement at 6, 12, and 24-month follow-up compared with patients having concomitant surgery.

*p<0.05 pre vs. follow-up	Pre (n)	6 Mo. (n)	12 Mo (n)
LVEDD (mm)	7.1±1.1(44)	65.2±1.9*(27)	63.1±2.3*(22)
LVEF (%)	22.4±1.3(44)	29.8±2.2(28)	31.1±2.3*(23)
NYHA	2.9±0.1(49)	1.7±0.1*(30)	1.6±0.1*(23)

CONCLUSION: Based on these encouraging results, randomized clinical trials are currently underway in Europe, Australia, and North America.

⁹1994-96 Research Scholar

**By Invitation*

T6 Left Ventricular Reshaping: CardioClasp Enhances Contractility and Response to Increased Afterload

Abul Kashem*, Satoshi Furukawa*, David B. Melvin*, William P. Santamore*, Sarmina Hassan*, Deborah L. Crabbe*, Kenneth B. Margulies*, Bruce Goldman*, Philadelphia, PA; Cincinnati, OH

OBJECTIVE: A hallmark of heart failure is the steep decline in cardiac output (CO) with increasing afterload. We tested whether the CardioClasp (Clasp) device by acutely reshaping the left ventricle (LV) and reducing the LV wall stress (LVWS), would improve the response to increased afterload.

METHODS: In dogs (n=9), 4-weeks of ventricular pacing (210 to 240 ppm) induced severe heart failure. LV function was evaluated before and after placing Clasp, which employs two indenting bars to reshape LV. Hemodynamics, echocardiography, and Sonometrics (®) crystals dimension were measured at steady state and during inferior vena cava occlusion.

RESULTS: Clasp decreased the LV end-diastolic anterior-posterior dimension by $27.8 \pm 2.6\%$, decreased LVWS from 116.5 ± 10.6 to 66.0 ± 7.6 g/cm², and increased fractional area of contraction (FAC) from 18.9 ± 8.7 to $31.1 \pm 15.2\%$. Clasp did not alter LVEDP, LVP, LV dp/dts,

and CO. Clasp increased the slopes of end-systolic pressure-volume relationship by $66.8 \pm 21.6\%$. At normal afterload, stroke volume vs. end-diastolic pressure (EDP) relationship was unaltered by the Clasp. However, with increased afterload (mean systolic pressure 90.7 ± 5.6 to 76.2 ± 6.8 mmHg as control), stroke volume decreased significantly less with Clasp compared to control ($p < 0.01$).

creased FAC by reshaping the LV. Clasp was able to maintain CO and arterial pressure while increasing contractility. Clasp prevented the steep decline in CO with increasing afterload. In severe heart failure patients with increased afterload, CardioClasp device may be effective in long-term clinical application.

**By Invitation*

T7 Video-Assisted Thoracoscopic Direct Left Ventricular Lead Placement in Patients with Congestive Heart Failure Who Fail Transvenous Lead Insertion for Cardiac Resynchronization

Omar M. Lattouf*, Amar M. Jayawant*, Angel Leon*, David DeLurgio*, Kathryn Glas*, John D. Puskas*, Robert A. Guyton; Atlanta, GA

*Discussant: Randall K. Wolf**

OBJECTIVE: The MIRACLE and MUST1C studies have shown that biventricular pacing (BVP) improves symptoms, quality of life, and ejection fraction in selected patients. However, transvenous insertion of the left ventricular (LV) pacing lead via the coronary sinus fails in up to 10% of patients. Early experience with thoracotomy for LV lead implantation resulted in a high mortality and morbidity. We describe a novel, minimally invasive approach to LV lead placement using video-assisted thoracoscopy (VAT).

METHODS: Eight patients underwent VAT LV lead placement. Patients had NYHA Class III and IV CHF, with a mean LV ejection fraction of $19\% \pm 10\%$. VAT provided access to the left ventricular wall anterior to the phrenic nerve. Two epicardial active fixation leads were inserted. Intra-operative transesophageal echocardiography (TEE) was used to guide lead positioning and assess ventricular function. Leads were tunneled subcutaneously and connected to a previously implanted tri-chamber transvenous pacemaker.

RESULTS: VAT LV lead implantation succeeded in all cases. Acute thresholds measured $1.43 \pm 0.9V$, with R-wave amplitude of 11.90 ± 7.4 mV and impedance of 708.50 ± 260.8 Ohms. TEE was important for site selection and confirming LV resynchronization and reduced mitral regurgitation during BVP. There were no deaths, MI, bleeding, or reoperation. Mean operative times and hospital stay were 219 ± 30 min and 3.4 ± 1 days, respectively.

CONCLUSION: VAT LV lead placement is safe and effective when transvenous implantation fails. Further experience will define the role of the VAT technique in biventricular pacing.

**By Invitation*

T8 Can Participate Extraction from the Ascending Aorta Reduce Neurologic Injury in Cardiac Surgery?

Christoph Schmitz*, Armin Welz*; Bonn, Germany

Discussant: Robert W. Emery

OBJECTIVE: This study examined whether extraction of paniculate emboli using intraaortic filtration could improve neurologic outcomes.

METHODS: Patients were enrolled in a prospective, controlled study. The therapy arm received intraaortic filtration, while control patients did not. Preoperative, procedural, and postoperative data were collected. Neurologic examinations included NIH Stroke scale and Glasgow Coma scale evaluations, as well as a battery of neurologic, neurocognitive, and neuropsychologic tests. Investigators administering neurologic tests were blinded to each patient's study arm.

RESULTS: 490 filter patients were enrolled: 261 in the filter arm, and 229 in the control arm. Patient histories and demographics showed no major differences between groups (Filter vs. control: mean age = 66 vs. 67 years; % male = 78 vs. 69; prevalence of hypertension = 77% vs. 79%; unstable angina = 17% vs. 13%; prior stroke 7% vs. 6%; prior TIA 14% vs. 13%). Procedures performed in each group were also similar (Filter vs. control: CABG = 85% vs. 84%; valve = 2.9% vs. 3.4%; combined CABG/valve 9.8% vs. 12.4%). Filter patients experienced a lower incidence of adverse neurologic outcomes than control patients. Filter patients experienced less stroke (0.4% vs 2.2%), TIA (0% vs. 1.8%), delirium (2% vs. 7.4%), coma (0% vs. 0.4%), memory deficit (1.6% vs. 7.5%). Neurocognitive and neuropsychological results supported the gross clinical findings. Patients receiving filtration exhibited higher postoperative scores when compared to their preoperative scores than control patients.

CONCLUSION: Patients in whom paniculate emboli are extracted using intraaortic filtration experience improved neurologic outcomes.

**By Invitation*

9:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY - PLENARY

HALL C

Topic: Peer Review is the Best Way to Insure Quality Patient Care; Public Reporting of Surgical Results is Unnecessary (and Misleading)

Pro: William C. Nugent

Lebanon, New Hampshire

Con: Mark Chassil

New York, New York

Moderator: James L. Cox

Marco Island, Florida

CONTROVERSIES IN CARDIOTHORACIC SURGERY - ACQUIRED

CARDIAC CONTROVERSIES

HALL C

10:30 a.m. Topic: Arterial Conduits Only Should Be Used in Coronary Artery Bypass Surgery

Pro: Brian F. Buxton

Heidelberg, Victoria, Australia

Con: Paul T. Sergeant

Bertem, Belgium

Moderator: Bruce W. Lytle

Cleveland, Ohio

11:15 a.m. Topic: Optimal Management of Postinfarction Left Ventricular Failure is Surgical Remodeling

Pro: Gerald D. Buckberg

Los Angeles, California

Con: Philippe Menasche

Paris, France

Moderator: D. Craig Miller

Stanford, California

12:00 p.m. ADJOURN

WEDNESDAY MORNING, MAY 8, 2002

CONTROVERSIES IN CARDIOTHORACIC SURGERY - GENERAL THORACIC CONTROVERSIES

ROOM 20

10:30 a.m. Topic: Randomized Clinical Trials Should be the Gold Standard in Clinical Studies for Thoracic Surgery

Pro: Larry R. Kaiser

Philadelphia, Pennsylvania

Con: Thomas W. Rice

Cleveland, Ohio

Moderator: Andrew S. Wechsler

Philadelphia, Pennsylvania

11:15 a.m. Topic: Resection for T-4 Lung Cancers: The Risks Outweigh the Benefits

Pro: Robert J. Ginsberg

Toronto, Ontario, Canada

Con: Philippe G. Dartevelle

Paris, France

Moderator: Douglas E. Wood

Seattle, Washington

12:00 p.m. ADJOURN

**CONTROVERSIES IN CARDIOTHORACIC SURGERY -
CONGENITAL HEART CONTROVERSIES
ROOM 31**

10:30 a.m. Topic: The Asymptomatic Young Patient with Ebstein's Anomaly Should have Surgery

Pro: ¹⁰Christopher J. Knott-Craig*

Oklahoma City, Oklahoma

Con: Joseph Dearani*

Rochester, Minnesota

Moderator: John J. Lamberti

New York, New York

11:15 a.m. Topic: Anatomic Correction is Better than Physiologic Correction for Congenitally Corrected Transposition of the Great Arteries

Pro: Roger B.B. Mee

Cleveland, Ohio

Con: Constantine Mavroudis

Chicago, Illinois

Moderator: Frank L. Hanley

San Francisco, California

12:00 p.m. ADJOURN

¹⁰1989-90 Graham Fellow

*By Invitation

GRAHAM EDUCATION AND RESEARCH FOUNDATION

13 Elm Street, Manchester, Massachusetts 01944, (978) 526-8330

President Tirone E. David, M.D.

Toronto, Ontario, Canada

Vice President Richard A. Jonas, M.D.

Boston, Massachusetts

Secretary-Treasurer William T. Maloney

Manchester, Massachusetts

Director Edward D. Verrier, M.D.

Seattle, Washington

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWSHIP

The Evarts A. Graham Memorial Traveling Fellowship was established in 1951 by The American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young surgeons from abroad who have completed their formal training in general, thoracic, and cardiovascular surgery. The award allows the recipient to study a year to intensify his training in a program of special interest and to travel to several sites to broaden his overall training and increase his contacts with thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 49 young surgeons from 26 countries have completed their training at thoracic surgical centers.

1st	1951-52	L. L. Whytehead Winnipeg, Manitoba, CANADA
2nd	1953-54	W.B. Ferguson Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley London, ENGLAND
4th	1955-56	Raymond L. Hurt Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth London, ENGLAND
6th	1957-58	Peter L. Brunnen Aberdeen, SCOTLAND
7th	1958-59	N.G. Meyne Amsterdam, HOLLAND

8th	1960-61	Godrej S. Karai Calcutta, INDIA
9th	1961-62	Fritz Helmer Vienna AUSTRIA
10th	1962-63	Theodor M. Scheinin Helsinki, FINLAND
11th	1963-64	Masahiro Saigusa Tokyo, JAPAN
12th	1963-64	Adarl. Hallen Uppsala, SWEDEN
13th	1964-65	Stuart C. Lennox London, ENGLAND
14th	1964-65	Elias Carapistolis Thessaloniki, GREECE
15th	1965-66	Gerhard Frichs Graz, AUSTRIA
16th	1965-66	Ary Blesovsky London, ENGLAND
17th	1966-67	C. Peter Clarke Fitzroy, AUSTRALIA
18th	1966-67	G.B. Parulkar Bombay, INDIA
19th	1967-68	Claus Jessen Copenhagen, DENMARK
20th	1969-70	Peter Brucke Linz-Puchenau, AUSTRIA
21st	1970-71	Michel S . Slim New York, NY, USA
22nd	1971-72	Severi Pellervo, Mattila Kaunianen, FINLAND
23rd	1972-73	Yasuyuki Fujiwara

		Tokyo, JAPAN
24th	1973-74	Marc Roger de Leval London, ENGLAND
25th	1974-75	J. J. DeWet Lubbe Cape Town, SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner Gdansk, POLAND
27th	1976-77	Bum Koo Cho Seoul, KOREA
28th	1977-78	Alan William Gale Sydney, AUSTRALIA
29th	1978-79	Eduardo Otero Coto Valencia, SPAIN
30th	1980-81	Richard K. Firmin Leicester, ENGLAND
31st	1981-82	Claudio A. Salles Belo Horizonte, MG, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki Osaka, JAPAN
33rd	1983-84	Georg S . Kobinia Klagenfurt, AUSTRIA
34th	1984-85	Aram Smolinsky Tel Hashomer, ISRAEL
35th	1985-86	Florentine J. Vargas Buenos Aires, ARGENTINA
36th	1986-87	Ari L. J. Harjula Helsinki, FINLAND
37th	1987-88	Byung-Chul Chang Seoul, KOREA
38th	1988-89	Wang Cheng Beijing, CHINA

39th	1989-90	Christopher John Knott-Craig Cape Town, SOUTH AFRICA
40th	1991-92	Ko Bando Okayama, JAPAN
41st	1992-93	Timothy E. Oaks Hershey, PA, USA
42nd	1993 -94	Alain E. Serraf Le Plessis Robinson, FRANCE
43rd	1995-96	Cornelius McKown Dyke Richmond, VA, USA
44th	1996-97	Monica Robotin-Johnson Sydney, AUSTRALIA
45th	1997-98	Jun Wang Beijing, CHINA
46th	1998-99	Christian Kreutzer Buenos Aires, ARGENTINA
47th	1999-00	Andes Franco-Cereceda Stockholm, SWEDEN
48th	2000-01	Albertus Scheule Tuebingen, GERMANY
49th	2001-02	Anna Maria Ciccone Rome, ITALY
50th	2002-03	Cliff K.C. Choong Auckland, NEW ZEALAND

**AMERICAN ASSOCIATION
FOR THORACIC SURGERY**

RESEARCH SCHOLARSHIP

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

EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

1986-1988 Mark K. Ferguson

University of Chicago, Department of Surgery

ALFRED BLALOCK RESEARCH SCHOLARSHIP

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

1988-1990 Gus J. Vlahakes

Massachusetts General Hospital and Harvard Medical School

JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

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

1990-1992 Donald D. Glover

Duke University Medical Center

ALTON OCHSNER RESEARCH SCHOLARSHIP

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

1992-1994 David H. Adams

Brigham and Women's Hospital

ROBERT E. GROSS RESEARCH SCHOLARSHIP

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

1994-1996 Mehmet C. Oz

Columbia-Presbyterian Medical Center

"Development of a Model of Chronic Pulmonary Rejection in Miniature Swine"

1994-1996 Thoralf Mauritz Sundt, III

Washington University School of Medicine

JOHN ALEXANDER RESEARCH SCHOLARSHIP

"Strategies to Prevent Hyperacute Rejection of the Pig Lung by Human Blood"

1996-1998 Richard Norris Pierson, 111

Vanderbilt University Medical Center

ANDREW G. MORROW RESEARCH SCHOLARSHIP

"The Detection of Telomerase Activity in Patients with Non-Small Cell Lung Cancer"

1997-1999 Stephen C. Yang

Johns Hopkins University School of Medicine

DWIGHT HARKEN RESEARCH SCHOLARSHIP

"Chimeric Hearts Test the Role of Antigen Presenting Cells in Rejection and Tolerance"

1998-2000 Bruce Rosengard

The University of Pennsylvania

SECOND EDWARD D. CHURCHILL RESEARCH SCHOLARSHIP

"The Role of Respiratory Muscle Adaptation in Lung Volume Reduction Surgery"

1999-2001 Joseph B. Shrager, M.D.

The University of Pennsylvania

SECOND ALFRED E. BLALOCK RESEARCH SCHOLARSHIP

"CD-4 Lymphocytes and Cardiac Allograft Vasculopathy"

2000-2002 Abbas Ardehali

UCLA School of Medicine

"Monocyte-Endothelial Cell Interactions in Delayed Xenograft Rejection"

2000-2002 Thomas K. Waddell

University of Toronto and Toronto General Hospital

SECOND JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

"Adjuvant Immunotherapy in the Treatment of Esophageal Cancer"

2001 -2003 Richard J. Battafarano, M.D.

Washington University School of Medicine

"B-Adrenergic Receptor Based Gene Therapy"

2001-2003 Carmelo A. Milano, M.D.

Duke University Medical Center

SECOND ALTON OCHSNER RESEARCH SCHOLARSHIP

"Nonablative Approches to Chimerism-Induced Transplantation Tolerance"

2002-2004 Yolonda Lorig Colson, M.D. Brigham & Women's Hospital

"Alveolar Macrophage in Reperfusion Injury of Lung"

Michael S. Mulligan, M.D.

University of Washington School of Medicine

**Charter Member*

+Board of Directors

†Director Ementus

**THE AMERICAN ASSOCIATION FOR
THORACIC SURGERY**

SCIENTIFIC ACHIEVEMENT AWARD

The American Association for Thoracic Surgery Scientific Achievement Award was established by the Association in 1994. The award serves to honor individuals who have achieved scientific contributions in the field of thoracic surgery worthy of the highest recognition the Association can bestow. Honorees receive a Medallion for Scientific Achievement from the Association presented by the president at the Annual Meeting and the honoree's name and biography is printed in the Journal of Thoracic and Cardiovascular Surgery.

**SCIENTIFIC ACHIEVEMENT AWARD
RECIPIENTS**

1995 John W. Kirklin, Birmingham, Alabama

1998 Norman E. Shumway, Stanford, California

1999 Michael E. DeBakey, Houston, Texas

2000 Denton A. Cooley, Houston, Texas

INTERNATIONAL TRAVELING FELLOWSHIP

The AATS Traveling Fellowship was established in 1997 by the American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young North American Cardiothoracic Surgeons who are within two years of the completion of their formal cardiothoracic surgery training. The award allows the recipient to study abroad for one year to intensify training in different disciplines and to travel to several sites to broaden the overall training and increase contacts with thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having potential for later international thoracic surgical leadership.

1998-99 Lishan Aklog, West Roxbury, MA

**AMERICAN ASSOCIATION
FOR THORACIC SURGERY**

**2001
NECROLOGY**

William E. Bloomer, M.D., Long Beach, California
David A. Blumenstock, M.D., Middlefield, Connecticut
Francis H. Cole, M.D., Memphis, Tennessee
John R. Derrick, M.D., Galveston, Texas
Charles Fineberg, M.D., Philadelphia, Pennsylvania
Charles J. Hahn, M.D., Arzier, Switzerland
Lucius D. Hill, M.D., Seattle, Washington
George H. Humphreys, M.D., West Dover, Vermont
R. Leonard Kemler, M.D., Hartford, Connecticut
Richard King, M.D., Atlanta, Georgia
G. Hugh Lawrence, M.D., Friday Harbor, Washington
Montague S. Lawrence, M.D., Cedar Rapids, Iowa
E B\$achley Main, M.D., Anaheim, California
Joe D. Morris, M.D., Ann Arbor, Michigan
William E. Neville, M.D., Santa Rosa, California
Melvin M. Newman, M.D., Pasadena, California
Alton Ochsner, Jr., M.D., Metairie, Louisiana
Beatty H. Ramsay, M.D., Pebble Beach, California
Herbert Ransdell, M.D., Louisville, Kentucky
Jefferson F. Ray, M.D., Key West, Florida
Keith Reemtsma, M.D., New York, New York
Morris Rubin, M.D., Rancho Mirage, California
Masahiro Saigusa, M.D., Tokyo, Japan
Will C. Sealy, M.D., Greenville, North Carolina
Ake Senning, M.D., Zurich, Switzerland
Zwi Steiger, M.D., Detroit, Michigan
Warren J. Taylor, M.D., Franconia, New Hampshire
Joe R. Utley, M.D., Spartanburg, South Carolina
John L. Wilson, M.D., Palo Alto, California

**Senior Member*

**AMERICAN ASSOCIATION
FOR THORATIC SURGERY**

2001 - 2002

GEOGRAPHICAL ROSTER

UNITED STATES

ALABAMA
Birmingham

CALIFORNIA
Alameda

Los Angeles
Benfield, John R

Holman, William L
Kahn, Donald R
Kirklin, James K
Kirklin, John W
McGriffin, David C

Montgomery

Simmons, Earl M

ARIZONA

Green Valley

McClenathan, James E
Paradise Valley
Nelson, Arthur R

Pheonix

Cornell, William P

Scottsdale

Fisk, R Leighton
Pluth, James R
Shields, Thomas W
Trastek, Victor F
Vaughn, Cecil C

Sun City

Read, C Thomas

Tucson

Copeland, Jack G, III
Sanderson, Richard G
Sethi, Gulshan K

ARKANSAS

Little Rock

Campbell, Gilbert S
Read, Raymond C

Ecker, Roger R
Arcadia
Lindsmith, George G
Bonita
Gonzalez-Lavin, Lorenzo

Burlingame

Ullyot, Daniel J

Capistrano Beach

Flynn, Pierce J

Chico

Becker, Ronald M

Coronado

Silver, Arthur

El Macero

Andrews, Neil C

Flintridge

Penido, John R F

Fresno

Evans, Byron H

Indian Wells

Salyer, John M

Inglewood

Lee, Myles E

La Canada

Meyer, Bertrand W

La Jolla

DeLaria, Giacomo A
Hutchin, Peter
Moreno-Cabral, Ricardo J

Loma Linda

Bailey, Leonard L
Razzouk, Anees J
Wareham, Ellsworth E

Long Beach

Stemmer, Edward A

Blanche, Carlos
Buckberg, Gerald D
Cohen, Robbin G
Davis, Lowell L
DeMeester, Tom R
Holmes, E. Carmack
Kay, Jermome H
Khonsari, Siavosh
Laks, Hillel
Longmire, William P, Jr
Maloney, James V, Jr
Matloff, Jack M
McKenna, Robert J, Jr
Mulder, Donald G
Sintek, Colleen F
Starnes, Vaughn A
Trento, Alfredo
Wells, Winfield J

Los Osos

Aronstam, Elmore M

Martinez

Guernsey, James M

Middletown

Turnley, Kevin

Montebello

Lui, Alfred H F

Oakland

Iverson, Leigh I G

Orange

Connolly, John E
Cazzaniga, Alan B
Milliken, Jeffrey C
Ott, Richard A

Palm Desert

Fosburg, Richard G

Palm Springs

Gundry, Steven R

Palo Alto

Jamplis, Robert W
Peters, Richard M

Palos Verdes Estates

Nelson, Ronald J
Stiles, Quentin R

Pasadena

Hughes, Richard K

Pebble Beach

Ebert, Paul A
Miller, George E. Jr

Portola Valley

Fogarty, Thomas J

Rancho Palos Verdes

Mandal, Ashis K

Sacramento

Berkoff, Herbert A
Follette, David M
Harlan, Bradley J
Hurley, Edward J
Young, J Nilas

San Bernardino

Misbach, Gergory A

San Diego

Baronofsky, Ivan D
Daily, Pat O
Dembitsky, Walter P
Jamieson, Stuart W
Miller, Fletcher A
Mountain, Clifton F

Sausalito

Zaroff, Lawrence I

Sonoma

Richards, Victor

Spring Valley

Long, David M, Jr

Stanford

Hanley, Frank L
Mark, James B. D
Miller, D. Craig
Mitchell, R. Scott
Oyer, Philip E
Reitz, Bruce A
Robbins, Robert C
Shumway, Norman E
Whyte, Richard I

Tiburon

Heydorn, William H

Torrance

Carey, Joseph S
Cukingnan, Ramon A
Moore, Thomas C
State, David

Ventura

Dart, Charles H, Jr

Victorville

Juardo, Roy A

CONNECTICUT

Bridgeport

Rose, Daniel M
Sanchez, Juan A

Essex

Jaretzki, Alfred, III

New Haven

Elefteriades, John A
Hammond, Graeme L
Kopf, Gary S

Norwalk

Okinaka, Arthur J

Wilton

Pool, John L

Woodbridge

Lindskog, Gustaf E
Stern, Harold

DELAWARE

Newark

Lemole, Gerald M

Wilmington

Norwood, William I
Pecora, David V

DISTRICT OF COLUMBIA

Washington

Katz, Nevin M
Keshishian, John M

Trummer, Max J
San Francisco
Ellis, Robert J
Hill, J. Donald
Karl, Tom R
Reddy, Vadiyala M
Thomas, Arthur N
Yee, Edward S

San Jose
Oakes, David D

San Marino
Tsuji, Harold K

San Rafael
Roe, Benson B

Santa Ana
Pratt, Lawrence A
Wakabayashi, Akio

Santa Barbara
Jahnke, Edward J
Love, Jack W

Santa Cruz
Fishman, Noel H

Santa Monica
Chaux, Aurelio
Fonkalsrud, Eric W
Morton, Donald L
Robertson, John M

Jacksonville
Edwards, Fred H
Koster, J Kenneth, Jr
McBride, Lawrence R
Stephenson, Sam Jr

Jupiter
Gerbasi, Francis S

Lady Lake
Luller, Josiah

Lakeland
Brown, Ivan W, Jr

Largo
Wheat, Myron W, Jr

Marco Island
Conrad, Peter W
Cox, James L

Miami
Bello, Alexis G
Bolooki, Hooshang
Daughtry, Dewitt C
Greenberg, Jack J
Jude, James R
Kaiser, Gerard A
Papper, Emanuel M
Pham, Si Mai
Ripstein, Charles B
Salerno, Tomas A
Subramanian, S
Thurer, Richard J
Wilder, Robert J

Miami Beach
Reis, Robert L
Spear, Harold C

Naples
Barrersby, James S
Gonzalez, Luiz L
Linberg, Eugene J
MacGregor, David C
Mundth, Eldred D
Smyth, Nicholas P D

Walnut Creek
May, Ivan A

COLORADO

Beulah
Bartley, Thomas D

Denver
Campbell, David N
Clarke, David R
Eiseman, Ben
Grover, Frederick L
Harken, Alden H
Hopeman, Alan R
Paton, Bruce C
Pomerantz, Marvin
Rainer, W. Gerald

Englewood
Kovarik, Joseph L

Littleton
Pappas, George

Snowmass
Karp, Robert B

Snowmass Village
Mills, Lawrence J

Winter Haven
Maurer, Elmer P R

Winter Park
Sherman, Paul H

GEORGIA

Atlanta
Craver, Joseph M
Gott, John P
Guyton, Robert A
Hatcher, Charles R, Jr
Hopkins, William A
Jones, Ellis L
Kanter, Kirk R
Kessler, Charles R
Lee, Arthur B, Jr
Mansour, Kamal A
Miller, Joseph I, Jr
Rivkin, Laurence M
Symbas, Panagiotis
Williams, Willis H

Augusta
Ellison, Robert G
Zumbro, G. Lionel, Jr,
Chickamauga
Hall, David P

Macon
Dalton, Martin L, Jr
Van De Water, Joseph M

Savannah
Yeh, Thomas J
St Simons Island
Taylor, Frederick H

HAWAII

Honolulu
Ching, Nathaniel P
Gebauer, Paul W
McNamara, J. Judson

Kihei
Smeloff, Edward A

IDAHO

Midgley, Frank M
Simmons, Robert L

FLORIDA

Altlanic Beach
Stranahan, Allan

Aventura
Bregman, David

Bal Harbour
Grondin, Pierre R

Bellair
Lasley, Charles H

Coconut Grove
Center, Sol

Coral Gables
Cooke, Francis N

Delray Beach
Shumacker, Harris B, Jr

Gainesville
Alexander, James A
Spotnitz, William D

Barker, Walter L
Breyer, Robert H
Campbell, Charles D
Faber, L. Penfield
Ferguson, Mark K
Fullerton, David A
Geha, Alexander S
Goldin, Marshall D
Hanlon, C. Rollins
Head, Louis R
Ilbawi, Michel N
Jeevanandam, Valluvan
Kittle, C Frederick
Lo Cicero, Joseph III
Massad, Malek G
Marvoudis, Costantine
Michaelis, Lawrence
Montoya, Alvaro
Najafi, Hassan
Raffensperger, John
Replogle, Robert L
Snow, Norman J
Tatooles, C. J
Thomas, Paul A, Jr
Vanecko, Robert M
Warren, William H
Zajtchuck, Rostik
Elk Grove Village
Sullivan, Henry J

Evanston
Rosengart, Todd K

Glencoe
Rubenstein, L H

Maywood
Pifarre, Roque

Oak Brook
Javid, Hushang
Jensik, Robert J
Mason, G. Robert
Nigro, Salvatore L

Ponte Verda Beach
Barnhorst, Donald A
Gilbert, Joseph, Jr
Punta Gorda
Taber, Rodman E
St Petersburg
Daicoff, George R
Tallahassee
Kraeft, Nelson H
Tamarac
Mendelssohn, Edwin
Tampa
Angell, William W
Robinson, Lary A

Boise
Herr, Rodney H
ILLINOIS

Burr Ridge
Blakeman, Bradford P
Chicago
Amato, Joseph J
Backer, Carl L

Oak Lawn
Allen, Bradley S
Park Ridge
Weinberg, Milton Jr
Peoria
DeBord, Robert A
Springfield
Hazelrigg, Stephen R
Willowbrook
Leininger, Bernard J
Winnetka
Fry, Willard A
Hunter, James A
Mackler, S Allen

INDIANA

Bloomington
O'Neill, Martin J, Jr
Fort Wayne
Ladowski, Joseph S
Indianapolis
Allen, Keith B.
Brown, John W
Lesler, Kenneth A
King, Harold
King, Robert D
Mahomed, Yousuf
Mandelbaum, Isidore
Siderys, Harry

IOWA

Cedar Rapids
Levett, James M
Council Bluffs
Sellers, Robert D
Des Moines
Dorner, Ralph A
Zeff, Robert H
Iowa City
Behrendt, Douglas M
Ehrenhaft, Johann L
Richenbacker, Wayne E
Rossi, Nicholas P
Stanford, William

KANSAS

Cunningham
Allbritten, Frank F, Jr
Kansas City
Piehler, Jeffrey M
Reed, William A
Lawrence
Miller, Don R
Mission Hills
Ashcraft, Keith W
Prairie Village
Holder, Thomas M
Shawnee Mission
Adelman, Arthur
Killen, Duncan A
Padula, Richard T
Wichita
Tocker, Alfred M

KENTUCKY

Lexington
Cruthcher, Richard R
Mentzer, Robert M, Jr
Todd, Edward P
Louisville
Austin, Erle H, III
Dowling, Robert D
Gray, Laman A, Jr
Mahaffey, Daniel E

LOUISIANA

Baton Rouge
Berry, B Eugene
Beskin, Charles A

Campiti

Bloodwell, Robert D
New Orleans
Blalock, John B
DeCamp, Paul T
DeLeon, Serafin Y
Ferguson, T. Bruce, Jr
Harrison, Lynn H, Jr
Hartz, Renee S
Hewitt, Robert L
Lindsey, Edward S
McFadden, P Lichael
Mills, Noel L
Moulder, Peter V
Ochsner, John L
Schramel, Robert J
VanMeter, Clifford H
Webb, Watts R

Shreveport

Macini, Mary C

MAINE

Portland

Bredenberg, Carl E
Morton, Jeremy R

Windham

Hiebert, Clement

MARYLAND

Baltimore
Attar Safuh
Baker, R. Robinson
Baumgartner, William A
Cameron, Duke Edward
Gott, Vincent L
Greene, Peter S
Haller, J. Alex, Jr
Hankins, John R
Krasna, Mark J
McLaughlin, Joseph S
Michelson, Elliott

Bethesda

Schrump, David S
Swain, Julie A

Glen Arm

Turney, Steven Z

Lutherville

Salomon, Neal W

Reisterstown

Heitmiller, Richard F

Worton

Walkup, Harry E

MASSACHUSETTS

Boston

Akins, Cary W
Aranki, Sary F
Austen, W. Gerald
Bougas, James A
Burke, John F
Byrne, John G
Cohn, Lawrence H
Collins, John J, Jr
Couper, Gregory S
Daggett, Willard M
Daly, Benedict D T
De Nido, Pedro J
Ellis, F. Henry, Jr
Folkman, M Judah
Grillo, Hermes C
Hilgenberg, Alan D
Jonas, Richard A
Lazar, Harold L
Levitsky, Sidney
Madsen, Joren C
Mathisen, Douglas J
Mayer, John E, Jr
Mentzer, Steven J
Moncure, Ashby C
Sellke, Frank W
Shemin, Richard J
Sugarbaker, David J
Thurer, Robert L
Torchiana, David F
Vlahkes, Gus J
Wain, John C, Jr
Weintraub, Ronald M
Wright, Cameron

Bolyston

Moran, John M
Okike, Okike N

Brookline

Berger, Robert L
Frank, Howard A

Watkins, Levi, Jr

Burlington

Shahain, David M

Cambridge

Malcolm, John A

Centerville

Lefemine, Armand A

Chestnut Hill

Laforet, Eugene G

Concord

Norman, John C

Dover

Black, Harrison

Falmouth

McElvein, Richard B

Framingham

Bernhard, William F

Hopkinton

Schuster, Samuel R

Medford

Desforges, Gerard

North Andover

Cook, William A

Osterville

Buckley, Mortimer J

Springfield

Engelman, Richard M

Rousou, John A

Vineyard Haven

Malm, James R

Wellesley Hills

Cleveland, Richard J

West Newton

Neptune, Wilford B

West Roxbury

Barsamian, Ernest M

Khuri, Shukri F

Weston

Rheinlander, Harold F

Westport Harbor

Findlay, Charles W

Westwood

Scannell, J. Gordon

Williamstown

Wilkins, Earle W

Worcester

Lahey, Stephen J

Vander Salm, Thomas J

Ferguson, Thomas B

Fiore, Andrew C

Fly, M. Wayne

Gay, William A, Jr

Huddleston, Charles B

Johnson, Frank E

Johnson, Robert G

Kouchoukos, Nicholas T

Lewis, J Eugene, Jr

Naunheim, Keith S

Pasque, Michael K

MICHIGAN

Ann Arbor

Bartlett, Robert H

Bolling, Steven F

Bove, Edward L

Deeb, G. Michael

Gago, Otto

Greenfield, Lazar J

Iannettoni, Mark D

Irsh, Marvin M

Neerken, A John

Orringer, Mark B

Pass, Harvey I

Prager, Richard L

Sload, Herbert E

Beverly Hills

Timmis, Hilary H

Detroit

Arbulu, Agustin

Baciewicz, Frank A, Jr

Delius, Ralph E

Silverman, Norman A

Stephenson, Larry W

Walters, Henry L, III

Wilson, Robert F

Grand Rapids

Harrison, Robert W

Neirotti, Rodolfo

Rasmussen, Richard A

Tomatis, Luis A

St Joseph

Levine, Frederick H

West Bloomfield

Arciniegas, Eduardo

MINNESOTA

Coon Rapids

Gannon, Paul G

Mendota Heights

Dennis, Clarence

Minneapolis

Arom, Kit V

Bolma, R. Morton, III

Emery, Robert W

Foker, John E

Garamella, Joseph J

Helseth, Hovald K

Joyce, Lyle D

Maddaus, Michael A

Molina, J. Ernesto

Nicoloff, Demetre M

Park, Soon J

Shumway, Sara J

Ward, Herbert B

NEW JERSEY

Alpine

Holswadem George R

Basking Ridge

Lewis, Ralph J

Belleville

Gerard, Franklyn P

Browns Mills

McGrath, Lynn B

Camden

Mamishion, Rudolph C

Rochester

Allen, Mark S

Bernatz, Philip E

Daly, Richard C

Danielson, Gordon K

Deschamps, Claude

McGregor, Chirstopher G A

Miller, Daniel L

Mullany, Charles J

Olsen, Arthur M

Orszulak, Thomas A

Pairolero, Peter C

Puga, Francisco J

Schaff, Hartzell V

Sundt, Thoralf M

Shorewood

Kiser, Joseph C

Stillwater

Kaye, Michael P

Waubun

DeNiro, Richard N

MISSISSIPPI

Cathage

Logan, William D, Jr

Jackson

Johnson, J. Harvey, Jr

Madison

Hardy, James D

MISSOURI

Chesterfield

Bergmann, Martin

Columbia

Curtis, Jacj J

Jones, James W

Silver, Donald

Walls, Joseph T

Fontenac

Penkiske, Patricia A

Strevey, Tracy E, Jr

Kansas City

Borkon, A Michael

Mayer, John H, Jr

VanWay, Charles W, III

St Charles

Codd, John E

St Louis

Barner, Hedrick B

Connors, John P

Cooper, Joel D

Damiano, Ralph J, Jr

NEW MEXICO

Albuquerque

Dietl, Charles A

Edwards, W. Sterling

Wernly, Jorge A

Alto

Sutherland, R. Duncan

Buena Vista

Thal, Alan P

Santa Fe

Davila, Julio C

Patterson, G. Alexander
Roper, Charles L
Sasser, William F
Webster Groves
Kaiser, George C
Willman, Vallee L

MONTANA

Columbia Falls
Myerowitz, P. David
Missoula
Duran, Carlos Gomez
Oury, James H

NEBRASKA

Omaha
Fleming, William H
Schultz, Richard D

NEVADA

Las Vegas
Carter, P Richard
Little, Alex G

NEW HAMPSHIRE

Center Harbor
Aaron, Benjamin L

Hanover
Baldwin, John C

Lebanon
Nugent, William C
Plume, Stephen K
Sanders, John H, Jr

Petersborough
Glenn, William W. L

Statham
Gaensler, Edward A

Windham
Burbank, Benjamin

DelRossi, Anthony J
Englewood
Ergin, M. Arisan
Fort Lee
Conklin Edward F

Hackensack
Hutchinson, John E, III

Jersey City
Demos, Nicholas J

Livingston
Hochberg, Mark S

Millburn
Parsonnet, Victor

Moorestown
Fernandez, Javier
Morse, Dryden P

Morristown
Parr, Grant V. S

Neptune
Roberts, Arthur J

New Brunswick
MacKenzie, James W
Scholz, Peter M

Newark
Donahoo, James
Swan, Kenneth G

Pittstown
Garzon, Antonio A

So. Orange
Gielchinsky, Issac

Tenafly
Gerst, Paul H
Wallsh, Eugene

Wyckoff
Adler, Richard H

Santa Teresa
Glass, Bertram A
Silver City
Waddell, William R

NEW YORK

Albany
Canver, Charles C
Moore, Darroch, W. O

Bay Shore
Ryan, Bernard J

Bellport
Finnerty, James

Bronx
Attai, Lari A
Ford, Joseph M
Frater, Robert W M
Gold, Jeffrey P
Hirose, Teruo
Veith, Frank J

Brooklyn
Acinapra, Anthony J
Cunningham, Joseph N, Jr
Levowitz, Bernard S
Sawyer, Philip N

Buffalo
Bhayana, Joginder N
Guiraudon, Gerard M
Hoover, Eddie L
Lajos, Thomas Z

Chappaqua
Fell, Stanley C

Dewitt
Parker, Frederick B, Jr

East Amherst
Anderson, Murray N

East Quogue
McCormack Patricia M

Fayetteville
Bugden, Walter F
Effler, Donald B

Young, W. Glenn, Jr
Greensboro

Van Trigt, Peter, III
Greenville

Chitwood, W. Randolph, Jr
Elbeery, Joseph R

High Point
Mills, Stephen A

Winston-Salem
Cordell, A Robert
Hammon, John W, Jr
Hudspeth, Allen S
Kon, Neal D
Meredith, Jesse H

OHIO

Chargin Falls
Ankeney, Jay L
Cross, Frederick S

Cincinnati
Albers, John E
Callard, George M
Flege, John B, Jr
Helmsworth, James A
Hiratzka, Loren F
Ivey, Tom D
Wilson, James M
Wright, Creighton B

Fishers Island
Baue, Arthur E

Floral Park
Crasnopol Philip

Honeoye Falls
Craver, William L

Larchmont
Steichen, Felicien M

Lido Beach
Hines George L

Millerton
Green George E

New York
Adams, David H
Altorki, Nasser K
Anagnostopoulos, C E
Bains, Manjit S
Boyd, Arthur D
Brodman, Richard F
Cahan, William G
Clauss, Roy H
Colvin, Stephen B
Culliford, Alfred T
Friedlander, Ralph
Calloway, Aubrey C, Jr
Griep, Randall B
Grossi Eugene A

Plattsburgh
Potter, Robert T
Rochester
DeWeese, James
Hicks, George L
Schwartz, Seymour I
Stewart, Scott

Roslyn
Thomson, Norman B, Jr
Wislof, George

Saranac Lake
Decker, Alfred M, Jr

Slingerlands
Kausel, Harvey W

Staten Island
Adams, Peter X

Stony Brook
Bilfinger, Thomas B
Soroff, Harry S

Syracuse
Brandt, Berkeley, III
Kohman, Leslie J
Meyer, John A

Valhalla
Moggio, Richard A
Reed, George E

Voorhees Ville

Isom, O. Wayne
King, Thomas C
Kirschner, Paul A
Krieger, Karl H
Lamerti, John J
Lansman, Steven L
Litwak, Robert S
Martini, Nael
McCord, Colin W
Mosca, Ralph S
Oz, Mehmet C
Quaegebeur, Jan M
Redo, S. Frank
Rose, Eric A
Rusch, Valerie W
Skinner, David B
Smith, Craig R
Spencer, Frank C
Spotnitz, Henry M
Subramanian, Valavanur A
Swanson, Scott J
Tice, David A
Tyras, Denis H
Waters, Paul F
Wichern, Walter, Jr
Wolff, William I

Foster, Eric D
NORTH CAROLINA
Asheville
Kroncke, George M
Takaro, Timothy
Atlantic Beach
Kerth, William J
Chapel Hill
Bowman, Frederick, Jr
Egan, Thomas M
Keagy, Blair A
Starek, Peter J
Wilcox, Benson R
Charlotte
Robicsek, Francis
Selle, Jay G
Durham
Anderson, Robert W
Davis, Duane R, Jr
Glower, Donald D
Harpole, David H, Jr
Jones, Robert
Lowe, James E
Oldham, H. Newland, Jr
Sabiston, David C, Jr
Smith, Peter K
Wolfe, Walter G

Cleveland
Blacksotone, Eugene H
Cosgrove, Delos M
Kirby, Thomas J
Loop Floyd D
Lytle, Bruce W
McCarthy, Patrick M
Mee, Roger B. B
Pettersson, Gosta B
Rice, Thomas W
Svensson, Lars G
Van Heeckeren, Daniel W
Columbus
Davis, J Terrance
Kakos, Gerard S
Meckstroth, Charles
Michler, Robert E
Williams, Thomas E, J
Dayton
DeWall, Richard A
Grove City
Kilman James W
Willoughby
Groves, Laurence K

OKLAHOMA**Jenks**

LeBeck, Martin B

Oklahoma City

Elkins, Ronald C
Felton, Warren L, II
Fishcer, R Darryl
Greer, Allen E
Munnell, Edward R
Zuhdi, M Nazih

OREGON**Ashland**

Campbell, Daniel C, Jr

Days Creek

Miller, Arthur C

Portland

Cobanoglu, Adnan
Krause Albert H
Lemmer, John H. Jr
Okies, J. Edward
Poppe, J Karl
Starr, Albert
Ungerleider, Ross M

PENNSYLVANIA**Bryn Mawr**

Haupt, George J
Templeton, John Y, III

Camp Hill

Pennock, John L

Carlisle

DeMuth, William E, Jr

Cochranville

Brockman, Stanley K

Darby

McKeown, John J, Jr

Hershey

Campbell, David B
Myers, John L
Pae, Walter, E, Jr
Pierce, William S

Bowles, L Thompson
Diehl, James T
Edie, Richard N
Edmunds, L. Henry, Jr
Gardner, Timothy J
Goldberg, Melvyn
Guerraty, Albert J
Hargrove, W Clark, III
Jacobs, Marshall L
Kaiser, Larry R
MacVaugh, Horace
Mannion, John D
Spray, Thomas L
Wechsler, Andrew S
Whitman, Glenn J. R

Pittsburgh

Bahnsen, Henry T
Griffith, Bartley P
Hadresty, Robert L
Hattler, Brack G, Jr
Keenan, Robert J
Konmos, Robert L
Landreneau, Rodney J
Luketich, James D
Magovern, George J
Magovern, George J, Jr
Magovern, James A
Ponitus, Robert G
Rams, James J
Siewers, Ralph D

Rosemont

Sink, James D

Rydel

Frobese, Alfred S

Sewickley

Clark, Richard E

Wayne

Lemmon, William M

West Chester

DiSesa, Verdi J

SOUTH CAROLINA**Charlestown**

Bradhman, R Randolph
Crawford, Fred A, Jr
Kratz, John M
Reed, Carolyn E
Rubin, Joseph W
Sade, Robert M
Swenson, Orvar

Columbia

Almond, Carl H

Greenwood

Bolton, J.W. Randolph

Hilton Head Island

Humphrey, Edward W

Isle of Palms

Mullen, Donald C

Landrum

Stayman, Joseph W

TENNESSEE**Johnson City**

Pennington, D. Glenn

Jonesborough

Bryant, Lester R

Knoxville

Blake, Hu Al
Brott, Walter H
Domm, Sheldon E

Memphis

Mainwaring, Richard D
McBurney, Robert P
Pate, James W
Robbins, S Gwin, Sr
Rosenweig, Jacob
Shochat, Stephen J
Watson, Donald C
Weiman, Darryl S

Nashville

Alford, William, Jr
Bender, Harvey W, Jr

Waldhausen, John A
Johnstown
Kolff, Jacob
Lancaster
Bonchek, Lawrence I
Rosemond, George P
Philadelphia
Acker, Michael A
Addonizio, V. Paul
Bavaria, Joseph E

Wilkes-Barre
Cimochowski, George E
Wynnewood
Wallace, Herbert W
Yardley
Sommer, George N, Jr
RHODE ISLAND

Providence
Hopkins, Richard A
Moulton, Anthony L
Singh, Aurn K

Drinkwater, Davis C
Gobbel, Walter G, Jr
Merrill, Walter H
Phillips, Steven J
Pierson, Richard N, III
Randolph, Judson, G
Rankin, J. Scott
Sawyers, John L
Stoney, William S
Thomas, Clarence, Jr

TEXAS

Austin
Tyson, Kenneth R T
Bedford
McPhail, Jasper L
Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Jessen, Michael E
Lambert, Cary J
Mack, Michael J
Platt, Melvin R
Ring, W Steves
Seybold, William D
Urschel, Harold C, Jr
Dilley
Hood, Richard H, Jr
Gaveston
Conti, Vincent R
Zwischenberger, Joseph B
Houston
Beall, Arthur C, Jr
Burdette, Walter J
Cooley, Denton A
Coselli, Joseph S
DeBakey, Michael E
Espada, J. Rafael
Frazier, O. Howard
Hallman, Grady L
Henly, Walter S
Lawrie, Gerald M
Mattox, Kenneth L
Ott, David A
Overstreet, John W
Putnam, Joe B, Jr
Roth, Jack A
Safi, Hazim J
Walker, William E
Wukasch, Don C
Kemp
Davis, Milton V
Lubbock
Bricker, Donald L
Feola, Mario
Good, R Maurice
San Antonio
Calhoon, John H
Cohen, David J
Dooley, Byron N
Treasure, Robert L
Temple
Brindley G. Valter, Jr

UTAH

Salt Lake City
Dolty, Donald B
Hawkins, John A
Jones, Kent W
Karwande, Shreekanth V
Liddle, Harold V
McGough, Edwin C
Mortensen, J D
Nelson, Russell M
VERMONT

Burlington

Leavitt, Bruce J
Hartland
Marrin, Charles A. S

Richford

Grondin, Claude M

VIRGINIA**Altavista**

Pierucci, Louis, Jr

Annandale

Alk, Bechara F
Burton, Nelson A
Lefrak, Edward A

Arlington

Kelpser, Roy G

Aylett

Gwathmey, Owen

Charlottesville

Crosby, Ivan Keith
Dammann, John F
Daniel, Thomas M
Kron, Irving L
Minor, George R
Muller, William H, Jr
Nolan, Stanton P
Tribble, Curtis G
Wellons, Harry A Jr

Fredericksburg

Armitage, John M

McLean

Gomes, Mario N
Mills, Mitchell
Wallace, Robert B

Norfolk

Baker, Lenox D

Reston

Boyd, Thomas F

Richmond

Bosher, Lewis H, Jr
Brooks, James W
Lower, Richard R

WASHINGTON**Belfair**

Jones, Thomas W

Bellingham

Varco, Richard L

Issaquah

Gentsch, Thomas O

Kirkland

Mills, Waldo O

Mercer Island

Li, Wei-I

Poulsbo

Malette, William G

Seattle

Aldea, Gabriel S
Allen, Margaret D
Anderson, Richard P
Lupinetti, F. Mark
Manhas, Dev R
Mansfield, Peter B
Merendino, K. Alvin
Miller, Donald W, Jr
Rittenhouse, Edward
Sauvage, Lester R
Thomas, George I
Verrier, Edward D
Wood, Douglas E

Spokane

Berg, Ralph, Jr

WEST VIRGINIA**Charleston**

Walker, James H

Huntington

Ferraris, Victor A

Morgantown

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

Parkersburg

Tarnay, Thomas J

WISCONSIN**Altoona**

McEnany, M Terry

Brookfield

Johnson, W. Dudley

Madison

Chopra, Paramjeet S
Cochran, Richard P
Young, William P

Marshfield**Milwaukee**

WYOMING

Myers, William O
Mequon
Narodick, Benjamin

Almassi, G. Hossein
Haasler, George B
Litwin, S Bert
Olinger, Gordon N
Tector, Alfred J

Shell
Scott, Meredith L
Tenton Village
Kaunitz, Victor H

West Bend
Gardner, Robert J

AMERICAN ASSOCIATION FOR THORACIC SURGERY

2001 - 2002

GEOGRAPHICAL ROSTER

OTHER COUNTRIES

ARGENTINA

Buenos Aires
Kreutzer, Guillermo O

AUSTRALIA

QUEENSLAND

Brisbane
O'Brien, Mark F

SOUTH AUSTRALIA

Beaumont
Sutherland, H D'Arcy M.S.

AUSTRIA

Leonding
Bruecke, Peter E

Salzburg
Unger, Felix H

Vienna
Wolner, Erst

BELGIUM

Bertem
Sergeant, Paul T

Leuven
Flameng Willem J
Lerut, Antoon E. M. R.

Toronto

Baird, Ronald J
Bigelow, Wilfred G
Christakis, George T
Coles, John G
David, Tirone E
Feindel, Christopher M
Fremes, Stephen E
Ginsberg, Robert J
Keshavjee, Shaf
McKneally, Martin F
Mickleborough, Lynda L
Scully, Hugh E
Trimble, Alan S
Trusler, George A
Weisel, Richard D
Williams, William G

Westbrook
Lynn, R Beverley

QUEBEC

Montreal
Blundell, Peter E
Carrier, Michel
Chartrand, Claude C. C

BRAZIL

Rio de Janeiro
Meier, Milton A

SÃ£o Paulo

Jatene, Adib D
Oliveria, Sergio A
SÃ£o JosÃ© do Rio Preto
Braile, Domingo M

CANADA

ALBERTA

Calgary
Bharadwaj, Baikunth
Miller, George E

Edmonton
Gelfand, Elliot T
Koshal, Arvind

Rebeyka, Ivan M
Ross, David B

Sterns, Laurence P

BRITISH COLUMBIA

Vancouver
Ashmore, Phillip G
Jamieson, W. R. Eric
Tyres, G. Frank O

Victoria

Field Paul
Sensstrom, John D

Stark, Jaroslav F
Taylor, Kenneth M
Yacoub, Magdi

Oxford

Westaby, Stephen

Somerset

Abbey-Smith, R

Worcestershire

Landymore, Roderick W

FINLAND

Helsinki
Harjula, Ari L. J

Kauniainen
Mattila, Severi P

FRANCE

Bordeaux
Fontan, Francis M

Bordeau-Pessac
Daudet, Eugene M

Creteil
Loisance, Daniel

Le Plessis Robinson
Binet, Jean-Paul
Dartevelle, Philippe G

MANITOBA

Winnipeg
Barwinsky, Jaroslaw
Cohen, Morley

NOVA SCOTIA

Halifax
Murphy, David A

ONTARIO

Collingwood
Heimbecker, Raymond

Hamilton
Urschel, John D

London
McKenzie, F Neil
Menkis, Alan H
Novick, Richard J

Mansfield
Pearson, F. Griffith

North York
Goldman, Bernard S

Oakville
Allen, Peter

Ottawa
Hendry, Paul J
Keon, Wilbert J

Berlin
Alexi-Meskishvili, Vladimir
Hetzer, Roland

Freiburg
Beyersdorf, Friedhelm
Hasse, Joachim T. W

Hamburg
Lacour-Gayet, Francois

Hannover
Haverich, Axel

Leipzig
Mohr, Freidrich W

Loiching
Sebening, Fritz

Munich
Borst, Hans G

Neuss
Bricks, Wolfgang H

GREECE

Athens
Palatianos, George M
Sarris, George E

GUATEMALA

Guatemala

**AMERICAN ASSOCIATION
FOR THORACIC SURGERY
CHARTER MEMBERS**

E. Wyllis Andrews	Arthur A. Law
John Auer	William Lerche
Edward R. Baldwin	Howard Lilienthal
Walter M. Boothby	William H. Luckett
William Branower	Morris Manges
Harlow Brooks	Walton Martin
Lawrason Brown	Rudolph Matas
Kenneth Bulkley	E.S. McSweeney
Alexis Carrel	Samuel J. Metzler
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	Walter I. Rathbun
Herman Fischer	Martin Rehling
Albert H. Carvin	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

**AMERICAN ASSOCIATION
FOR THORACIC SURGERY
THE BY-LAWS**

ARTICLE 1. NAME

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

ARTICLE II. PURPOSE

The purposes of the Association shall be:

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

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

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

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

ARTICLE III. MEMBERSHIP

Section 1. There shall be three classes of members: Honorary, Senior, and Active. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws. Honorary members shall have the privilege of voting but shall not be eligible to hold office.

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

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

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

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

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

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

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

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

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

ARTICLE IV. BOARD OF DIRECTORS ("COUNCIL")

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

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

- a. It may not alter the initiation fees or annual dues, or levy any general assessments against the membership, except that it may, in individual cases, waive annual dues or assessments.
- b. It may not change the Articles of incorporation or By-Laws.
- c. It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article 111, Section 8.

Section 3. At the conclusion of the annual meeting, the retiring President shall automatically become a Councilor for a one-year term 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 President, a Vice-President, a Secretary, and a Treasurer. All officers must be Active or Senior Members of the Association. Said officers shall be ex-officio members of the Council of the Association.

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

Section 3. The President, Vice-President, Secretary and Treasurer shall be elected at the annual meeting of the Association and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for a one-year term of office and neither 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 maybe reelected for not more that four additional terms.

Section 4. The President of the Association shall perform all duties customarily pertaining to the office of President. He shall preside at all meeting 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 President. The Council shall advance the Vice-President to the Presidency and appoint an interim Vice-President to the Presidency and appoint an interim Vice-President.

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

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve a Treasurer of the Association.

Section 8. The Editor of the Association is not an officer of the Association. He shall be appointed by the Council at its annual meeting; provided, however, that such appointment shall not become effective until approved by the Association at the annual meeting of the Association. The Editor shall be appointed for a five-year term and may not be appointed to more than two successive terms; provided, however, that an Editor completing two years or less of the unexpired term of a previous Editor may be appointed for two successive five-year terms. The Editor shall serve as the Editor of the Official Journal and shall be ex officio the Chairman of the Editorial Board and a member of the Council of the Association without vote.

Section 9. Vacancies occurring among the officers named in Section I or a vacancy in the position of Editor shall be temporarily filled by the Council, subject to approval of the Association at the next meeting of the Association.

ARTICLE VI. COMMITTEES

Section 1. The Council is empowered to appoint a Membership Committee, A Program Committee, a Necrology Committee and such other committees as may in its opinion be necessary or desirable. All such committees shall render their reports at an executive session of the Association, except that no ad hoc committee need report unless so directed by the Council.

Section 2. The Membership Committee shall consist of seven Active or Senior Members. The Council may appoint not more than one of its own members to serve on this Committee. The Duties of the Membership Committee are to investigate all candidates for membership in the Association and to report its findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than five of the members may be reappointed to succeed themselves. This Committee is also charged with maintaining a record of membership attendance and participation in the scientific programs and reporting to the affected members and to the Council any deviations from the requirement of Article VIII, Section 4, of these By-Laws.

Section 3. The Program Committee shall consist of at least 13 members: the President, the Vice President, the Secretary and the Editor and at least 9 members-at-large, three each representing the areas of adult cardiac, pediatric cardiac and general thoracic surgery. Three of these members-at-large shall be appointed each year by the President for a three-year term. Additional Committee members shall be appointed for one or two-year terms. 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 Nomination 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 eight members: two cardiac surgeons, two general thoracic surgeons, two transplant surgeons, and two pediatric heart surgeons, two to be appointed each year for four year terms with the senior two members of the Committee serving as Co-Chairs. The duties of the Committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and Fellows, past, present and future.

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

Section 9. The Ethics Committee shall consist of five members appointed by the Council. The Ethics Committee shall advise the Council concerning alleged breaches of ethics. Complaints regarding alleged breaches of ethics shall be received in writing by the Ethics Committee and shall be investigated by it. In addition, the Ethics Committee may investigate on its own initiative.

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

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

Section 12. The Committee on Publications shall consist of the Secretary, the Treasurer, and the Executive Director. The Committee shall oversee the business relationships between the Association and the publisher of its journal and maintain liaison among the publisher, the editor, and the Council.

Section 13. The Editorial Advisory Committee shall consist of five members appointed by the council including the Secretary, who shall serve as Chairman. One member shall be appointed each year for a four year term. The committee shall have advisory oversight for all

official scientific publications of the Association and make recommendations to the Editor and the Council.

ARTICLE VII. FINANCES

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

Section 2. Members shall contribute to the financial maintenance of the Association through initiation fees, annual dues, and special assessments. The amount of the annual dues and the initiation fees shall be determined by these By-Laws. If, at the end of any fiscal year, there is a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Active members to contribute the necessary amount so that no deficit is carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment 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 expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact.

ARTICLE VIII. MEETINGS

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

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

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

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

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

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

1. Appointment of necessary committees.
2. 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 of the last fiscal year.
3. Audit Report.

4. Report of the Necrology Committee.
5. Report of the Program Committee.
6. Action on amendments to the Article 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 Nominating Committee.
13. Election of officers.

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

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

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

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

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

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

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

ARTICLE IX. INDEMNIFICATION AND DIRECTORS AND OFFICERS

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

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

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

ARTICLE X. PAPERS

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

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

ARTICLE XL INITIATION FEES, DUES AND ASSESSMENTS

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

Section 2. Annual dues for Active Members shall be \$200.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Senior Members are exempt from dues.

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

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

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

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

ARTICLE XII. PARLIAMENTARY PROCEDURE

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

ARTICLE XIII. AMENDMENTS

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

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

As amended, May, 2001

As amended, May, 2001

DISCLOSURE INDEX

As a sponsor accredited by the Accreditation Council for Continuing Medical Education (ACCME), the American Association for Thoracic Surgery must ensure balance, independence, objectivity, and scientific rigor in all its individually sponsored or jointly sponsored educational activities. All faculty participating in a sponsored activity are expected to disclose to the activity audience any significant financial interest or other relationship (1) with the manufacturer(s) of any commercial product(s) and/or provider(s) of commercial services discussed in an educational presentation and (2) with any commercial supporters of the activity. (Significant financial interest or other relationship can include such things *as* grants or research support, employee, consultant, major stockholder, member of speakers bureau, etc.). The intent of this disclosure is not to prevent a presenter with a significant financial or other relationship from making a presentation, but rather to provide listeners with information on which they can make their own judgments. It remains for the audience to determine whether the speaker's interests or relationships may influence the presentation. In addition, presenters must make a meaningful disclosure to the audience of their discussions of unlabeled or unapproved drugs or devices. *Please note that one or more of the listed authors of the papers below may have a relationship as noted.*

Program #: Author Names;

Relationship Code - *Device, Product or Drug, Company Name*

F03: Abujiang Pataer*, Sunil Chada*, Kelly K. Hunt*, Jack A. Roth, Stephen G. Swisher*;

A - *Latrogen Corporation*

F08: Meinoshin Okumura*, Mitsunori Ohta*, Yoshitaka Fujii*, Hikaru Matsuda;

A - *A Grant-in-Aid for Scientific Research from the Ministry of Education, Science, Sports and Culture*

F12: Mahmoud Malas*, Craig J. Baker*, Suzanne M. Quardt*, Mark L. Barr*, Winfield J. Wells;

A - *Cryolife, Inc.*

F13: Vicki Lynn Mahan*, Saroja Ilangovan*, Reuben Cuison*, Jyothi Patil*, Sarah Docktor*, Vincent Rizzo*, Michel N. Ilbawi;

E - *Department of Pediatric CTS Hope Children's Medical Fund Advocate Christ Hospital*

F21: James J. Pilla*, Daniel J. Brockman*, Aaron S. Blom*, Qing Yuan*, Michael A. Acker;

B - *Acorn*

F23: Thomas Walther*, Andreas Schubert*, Volkmar Falk*, Christian Binner*, Niko Doll*, Jan Gummert*, Friedrich W. Mohr;

B - *Prop*

F29: Harold L. Lazar, Yusheng Bao*, Yu Zhang*, Shelia Bernard*;

A - *Pfizer Corporation*

6: John D. Puskas, Willis H. Williams, Peggy G. Duke, James Staples, John Jeffrey Marshall, Susan A. McCall, Bonnie H. Sammons, Rebecca J. Petersen, Dianne E. Bailey, Elizabeth M. Mahoney, William S. Weintraub, Robert A. Guyton;

A, B, D - *Medtronic, Quest Medical;*

E - *Carlyle Fraser Heart Center Foundation*

38: Jai Raman*, Susumu Ishikawa*, John M. Power*;

E - *EPT-Boston Scientific Corporation*

- 40:** Beat H. Walpoth*, Dirk Sprunge*, Beat Kipfer*, Pascal Berdat*, Peter Neidhart*, Jiirgen Robe*, Otto M. Hess*, Thierry Carrel*;
A - *Switzerland Heart Foundation*
- 41:** Brian F. Buxton*, Jai Raman*, Alexander Rosalion*, George Matalanis*, David L. Hare*;
A - *Johnson and Johnson*
- 50:** Stephen Yang*, Richard Whyte, Fred Askin*, Sharon Thomsen*, Pheroze Tamboli*, Gerald Berry*, Carmen Roig*, Joe Putnam;
B - *RITA Medical, Inc.*
- L8:** William M. Yarbrough*, Rupak Mukherjee*, Theresa A. Brinsa*, Kathryn B. Dowdy*, Amelia A. Scott*, G. Patricia Escobar*, Cassandra Joffs*, David G. Lucas*, Fred A. Crawford, Jr., Francis G. Spinale*;
A - *Procter and Gamble*
- T1:** Randall K. Wolf, Edwin L. Alderman, Michael P. Caskey, Allen R. Raczkowski, Mercedes K. Dullum, Dwight C. Lundell, Arthur C. Hill, Nan Wang, Michael A. Daniel;
A - *Caulscent Surgical*
- T2:** David H. Adams, Farzan Filsoufi, Lishan Aklog*, R. Saeid Farivar, Curds A. Anderson, Raymond H. Chen, Samuel Lichtenstein, Ji Zhang;
A - *Ventrica, Inc.*
- T4:** Ronald C. Elkins, Steven Goldstein, Steven P. Walsh, Kirby S. Black;
A, B, C, E - *Cryolife, Inc.*
- T5:** Mehmet C. Oz, Wolfgang F. Konertz*, Franz X. Kleber*, Friedrich W. Mohr, Jan F. Gummert*, Jorg Ostermeyer*, Michael Lass*, Jai Raman*, Michael A. Acker, Nicholas Smedira*;
B - *Acorn*
- T6:** Abul Kashem*, Satoshi Furukawa*, David B. Melvin*, William P. Santamore*, Sarmina Hassan*, Deborah L. Crabbe*, Kenneth B. Margulies*, Bruce Goldman*;
A, B, D - *CardioClasp Inc.*
- T8:** Christoph Schmitz, Armin Welz;
A - *Embol-X*

Relationship Codes

- A** = Grants/Research Support
B = Consultant
C = Stock Shareholder (directly purchased)
D = Honorarium
E = Other Financial or Material Support