2001 ANNUAL MEETING PROGRAM

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
2000 – 2001

President James L. Cox, Washington, DC
Vice-President Timothy J. Gardner, Philadelphia, PA
Secretary Tirone E. David, Toronto, ON, Canada
Treasurer Richard A. Jonas, Boston, MA
Editor Andrew S. Wechsler, Philadelphia, PA

Councilors Delos M. Cosgrove (2001), Cleveland, OH
O. Wayne Isom (2003), New York, NY
G. Alexander Patterson (2002), St. Louis, MO
Carolyn E. Reed (2004), Charleston, SC
Edward D. Verrier (2001), Seattle, WA
Historian John A. Waldhausen, Hershey, PA

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Association Representative, The American Board of Thoracic Surgery
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Douglas J. Mathisen (2001), Boston, MA
Lawrence H. Cohn (2004), Boston, MA

Board of Governors, American College of Surgeons
Alex G. Little (2002), Las Vegas, NV
Sidney Levitsky (2003)A(f, Boston, MA

THE AMERICAN ASSOCIATION FORTHORACIC SURGERY 2001 ANNUAL MEETING COMMITTEES

LOCAL ARRANGEMENTS

Walter P. Dembitsky, Chair
Richard G. Fosburg
Stuart W. Jamieson
Giacomo A. DeLaria

SPOUSE HOSPITALITY COMMITTEE

Becky Dembitsky, Chair
Cathy Fosburg
Ellen Jamieson

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY 2001 ANNUAL MEETING

PROGRAM COMMITTEE

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Douglas E. Wood (2002)	Seattle, Washington
Andrew S. Wechsler (2001)	Philadelphia, Pennsylvania

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Valerie W. Rusch (2001), Chair	New York, New York
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Richard A. Jonas (2001)	Boston, Massachusetts .
John A. Elefteriades (2003)	New Haven, Connecticut
Edward D. Verrier (2002)	Seattle, Washington
Mark R. deLeval (2004)	London, England

GRAHAM EDUCATION & RESEARCH FOUNDATION

Tirone E. David, Chair (2001)	Toronto, ON, Canada
Richard A. Jonas (2001)	Boston, Massachusetts
William T. Maloney (2001)	Manchester, Massachusetts
Valerie W. Rusch (2001)	New York, New York

YOUNG MEMBERS ADVISORY COMMITTEE

Robbin G. Cohen, Chair (2001)	Los Angeles, California
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R. Scott Mitchell (2002)	Woodside, California
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Clifford H. VanMeter (2003)	New Orleans, Louisiana
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AATS / STS NEW TECHNOLOGY ASSESSMENT COMMITTEE

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Lawrence H. Cohn	Brookline, Massachusetts
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Larry R. Kaiser	Philadelphia, Pennsylvania
Rodney J. Landreneau	Pittsburgh, Pennsylvania
Antoon E. M. R. Lerut	Leuven, Belgium
Michael J. Mack	Dallas, Texas
Lawrence R. McBride	St. Louis, Missouri
John D. Puskas	Atlanta, Georgia
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Hartzel V. Schaff	Rochester, Minnesota
Valavanur A. Subramanian	New York, New York
Jan L. Svennevig	Oslo, Norway
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Daniel J. Ullyot	Burlingame, California
Marco Zenati	Pittsburgh, Pennsylvania

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Martin F. McKneally, Chair (2001)	Toronto, ON, Canada
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John J. Collins, Jr. (2001)	Boston, Massachusetts
Marvin Pomerantz (2001)	Denver, Colorado
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John A.Waldhausen(2001)	Hershey, Pennsylvania

CARDIOTHORACIC RESIDENTS COMMITTEE

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Ralph J. Damiano, Jr. (2001)	St. Louis, Missouri
Davis C. Drinkwater (2001)	Nashville, Tennessee
Timothy J. Gardner (2001)	Philadelphia, Pennsylvania
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Steven J. Mentzer (2001)	Boston, Massachusetts
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Tirone E. David, Chair (2001)	Toronto, Ontario, Canada
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Randall B. Griepp, Chair (2001)	New York, New York
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Mortimer J. Buckley, Chair (2001)	Osterville, Massachusetts
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Delos M. Cosgrove (2005)	Cleveland, Ohio
Floyd D. Loop (2003)	Cleveland, Ohio
David B. Skinner (2002)	New York, New York

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

2000-2001

AMERICAN COLLEGE OF SURGEONS ADVISORY COUNCIL FOR CARDIOTHORACIC SURGERY

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

AMERICAN MEDICAL ASSOCIATION
HOUSE OF DELEGATES
L. Penfield Faber, Chicago, Illinois (2001)

AMERICAN MEDICAL ASSOCIATION
CPT-4 ADVISORY COMMITTEE
James M. Levett, Cedar Rapids, Iowa (2001)

ASSOCIATION OF AMERICAN MEDICAL COLLEGES
COUNCIL OF ACADEMIC SOCIETIES
Stanton P. Nolan, Charlottesville, Virginia (2001)
Richard J. Shemin, Boston, Massachusetts (Alt)

ASSOCIATION OF PHYSICIANS' ASSISTANTS
IN CARDIOVASCULAR SURGERY
Bruce W. Lytle, Cleveland, Ohio (2001)

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)

EXTRACORPOREAL PERFUSION
(AMSECT, ABCPT, ACPE AND CAHEA, CAAHEP)
Hendrick B. Earner, St. Louis, Missouri (2001)
Stanton P. Nolan, Charlottesville, Virginia (2001)

NATIONAL ASSOCIATION FOR BIOMEDICAL RESEARCH
William A. Baumgartner, Baltimore, Maryland (2001)

AMERICAN ASSOCIATION OF BLOOD BANKS
Robert L. Thurer, Boston, Massachusetts (2001)

AMERICAN MEDICAL ASSOCIATION
COMMITTEE ON EDUCATION AND RESEARCH
Tirone E. David, Toronto, ON, Canada (2001)

MEDICAL TECHNOLOGY LEADERSHIP FORUM
Bruce W. Lytle, Cleveland, Ohio (2001)

JOINT COUNCIL ON THORACIC SURGERY
EDUCATION (AATS/STS/TSDA/ABTS/RRC)

Fred A. Crawford, Jr. (Chair)	Charleston, South Carolina
William A. Baumgartner (ABTS)	Baltimore, Maryland
John W. Brown (RRC)	Indianapolis, Indiana
Jeffrey P. Gold (TSDA)	Bronx, New York
Nicholas T. Kouchoukos (STS)	St. Louis, Missouri
Douglas J. Mathisen (RRC)	Boston, Massachusetts
Gordon F. Murray (STS)	Morgantown, West Virginia
Gordon N. Olinger (TSDA)	Boston, Massachusetts
Peter C. Pairolero (ABTS)	Rochester, Minnesota
G. Alexander Patterson (AATS)	St. Louis, Missouri
Robert L. Replogle (ACS)	Chicago, Illinois
Eric A. Rose (AATS)	New York, New York
Benson R. Wilcox (ACS)	Chapel Hill, North Carolina

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Sidney Levitsky, Co-Chair	Boston, Massachusetts

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	Atlanta, Georgia
John L. Ochsner	New Orleans, Louisiana
Ivan M. Rebeyka	Edmonton, Alberta, Canada
Thomas W. Rice	Cleveland, Ohio
Jack A. Roth	Houston, Texas
Valerie W. Rusch	New York, New York
Hartzell V. Schaff	Rochester, Minnesota
Frank W. Sellke	Boston, Massachusetts
Willem Van Oeveren	Groningen, Netherlands
Edward D. Verrier	Seattle, Washington
William G. Williams	Toronto, ON, Canada

JOINT COMMITTEE ON PROFESSIONAL AFFAIRS (AATS/STS)

John E. Mayer, Chair	Boston, Massachusetts
John C. Baldwin	Hanover, New Hampshire
Alfred S. Casale	Passaic, New Jersey
Lawrence H. Cohn	Boston, Massachusetts
Delos M. Cosgrove	Cleveland, Ohio
Timothy J. Gardner	Philadelphia, Pennsylvania
Frederick L. Grover	Denver, Colorado
George C. Kaiser	Webster Groves, Missouri
David B. Skinner	New York, New York
Victor F. Trastek	Rochester, Minnesota
Paul N. Uhlig	Concord, New Hampshire
Harold C. Urschel Jr	Dallas, Texas

PAST PRESIDENTS OF THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Year	*Meeting Location*	*President*
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 *VS.* 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	John H. Gibbon, Jr.
	(Deceased 1/11/61)	Richard H. Sweet

Year	*Meeting Location*	*President*
1961-1962	St. Louis, MO	O. Theron Clagett
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, CA	John W. Strieder
1972-1973	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, C A	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	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

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

DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

SATURDAY, MAY 5, 2001 12:00 NOON - 6:00 P.M.
SAN DIEGO CONVENTION CENTER

Co-Chairmen: Irving L. Kron, M.D. and Edward D. Verrier, M.D.

OBJECTIVE

The 2001 Academic Surgeon's Symposium is designed to help develop the Academic Cardiothoracic Surgeon. This is an effort by the American Association for Thoracic Surgery to help provide a specific educational conference for potential and active academic cardiothoracic surgeons. The present symposium will focus on several areas including building a clinical program, developing new technology, getting published, administrative skills and mentorship. This Symposium is designed for Residents interested in a career in academic cardiothoracic surgery, junior Faculty in academic institutions, as well as senior Faculty including Division and Department Heads. It is intended that at the completion of this Symposium, participants should have better knowledge regarding developing and teaching academic skills.

ACCREDITATION

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing education activity for 4 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

PROGRAM

12:00 P.M. LUNCH

1:00 P.M. THE FUTURE OF ACADEMIC CARDIOTHORACIC SURGERY: CANADIAN PERSPECTIVE
Tirone E. David, M.D., Toronto General Hospital, Toronto, Ontario, Canada

1:30 P.M. THE FUTURE OF ACADEMIC CARDIOTHORACIC SURGERY: AMERICAN PERSPECTIVE

James L. Cox, M.D., President, American Association for Thoracic Surgery

2:00 P.M. TRANSFERRING INTELLECTUAL PROPERTY AND TECHNOLOGY
†Richard D. Weisel, M.D., Toronto General Hospital, Toronto, Ontario, Canada

2:30 P.M. THE USE OF EDUCATION TO DEVELOP AN ACADEMIC THORACIC CAREER
Jeffrey P. Gold, M.D., Montefiore Hospital and Medical Center, Bronx, New York

3:00 P.M. BREAK

3:15 P.M. HUMAN GENOMICS (PROTEONOMICS): POTENTIAL IMPACTS ON CTS RESEARCH AND FUNDING STRATEGIES

Alden H. Harken, M.D., University of Colorado, Denver, Colorado

3:45 P.M. NEGOTIATING A SUCCESSFUL ACADEMIC ENVIRONMENT

Larry R. Kaiser, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania

4:15 P.M. ACADEMIC USE OF INFORMATICS AND THE INTERNET
Peter S. Greene, M.D., Johns Hopkins Hospital, Baltimore, Maryland

4:45 P.M. FINANCING AND DEVELOPING NEW TECHNOLOGY
††W. Randolph Chitwood, Jr., M.D., E. Carolina University School of Medicine, Greenville, North Carolina

5:15 P.M. - 6:00 P.M. RECEPTION

†Presenter has a relationship with Institute Surgical and Computer Motion ††Presenter has a relationship with The Genzyme Corporation

GENERAL THORACIC SURGERY SYMPOSIUM

SPONSORED IN COOPERATION WITH THE GENERAL THORACIC SURGICAL CLUB

SUNDAY, MAY 6, 2001 7:50 A.M. - 5:10 P.M.
SAN DIEGO CONVENTION CENTER

OBJECTIVE:

The objective of this year's Postgraduate Course in General Thoracic Surgery is to expose the participants to four broad areas. A greater emphasis has recently been placed on more minimally invasive approaches to thoracic surgical problems. This course will focus on some of the more complex problems and demanding procedures of our specialty. There is a strong emphasis on demanding technical procedures involving tracheobronchial and vascular surgical procedures. There is a strong emphasis on the management of failed surgical management of esophageal disease. There is an emphasis on the evolving approach to pathologic processes impairing pulmonary function. Finally there is an emphasis on those thoracic surgical problems that often involve interaction with other surgical specialties. This program highlights the variety and demanding nature of our specialty.

ACCREDITATION

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing education activity for 7 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

PROGRAM

7:00 A.M. REGISTRATION & CONTINENTAL BREAKFAST

7:50 A.M. INTRODUCTION:

Douglas J. Mathisen, M.D., Chairman

SESSION I ESOPHAGEAL DISEASE

Moderator: Richard F. Heitmiller, M.D.

Reisterstown, Maryland

8:00 A.M. MANAGEMENT OF ESOPHAGEAL DIVERTICULA

Malcolm M. DeCamp, Jr., M.D., Cleveland Clinic Foundation, Cleveland, Ohio

8:15 A.M. MANAGEMENT OF FAILED THERAPY FOR ACHALASIA

Mark B. Orringer, M.D., University of Michigan Ann Arbor, Michigan

8:30 A.M. MANAGEMENT OF THE FAILED ANTIREFLUX PROCEDURE

Mark S. Allen, M.D., Mayo Clinic, Rochester, Minnesota

8:45 A.M. DISCUSSION

SESSION II MANAGEMENT OF SURGICAL INFECTIONS

Moderator: Thomas W. Rice, M.D.

Cleveland, Ohio

9:05 A.M. SURGICAL MANAGEMENT OF MYCOBACTERIAL DISEASE

Marvin Pomerantz, M.D., University of Colorado Health Science Center, Denver, Colorado

9:20 A.M. SURGICAL MANAGEMENT OF FUNGAL DISEASES

Daniel Miller, M.D., Mayo Medical Center, Rochester, Minnesota

9:35 A.M. SURGICAL MANAGEMENT OF DESCENDINGMEDIASTINITIS

Mark Iannettoni, M.D., University of Michigan Ann Arbor, Michigan

9:50 A.M. SURGICAL MANAGEMENT OF THORACIC ESOPHAGEAL PERFORATION

Richard Whyte, M.D., Stanford University Stanford, California

10:05 A.M. DISCUSSION

10:30 A.M. BREAK

SECTION III GENERAL THORACIC SURGEON AS A VASCULAR SURGEON

Moderator: James Luketich, M.D.

Pittsburgh, Pennsylvania

11:00 A.M. MANAGEMENT OF TUMORS INVOLVING THE SUPERIOR VENA CAVA

Philippe G. Dartevelle, M.D., Chir Ctr Marie Lannelongue Le Plessis Robinson, France

11:15 A.M. PULMONARY THROMBOENDARTERECTOMY

Michael S. Mulligan, M.D., University of Washington Seattle, Washington

11:30 A.M. VASCULAR COMPRESSIVE SYNDROMES

Cameron D. Wright, M.D., Massachusetts General Hospital, Boston, Massachusetts

11:45 A.M. DISCUSSION

12:00 P.M. LUNCH

SESSION IV GENERAL THORACIC SURGEON AS AN INTERDISCIPLINARY COLLABORATOR

Moderator: Valerie W. Rusch, M.D.

New York, New York

1:00 P.M. MUSCLE FLAPS IN THORACIC SURGERY

Peter C. Pairolero, M.D., Mayo Clinic Rochester, Minnesota

1:15 P.M. VERTEBRECTOMY AND RECONSTRUCTION

FOR TUMORS INVADING THE SPINE

Garret Walsh, M.D., M D Anderson Cancer Center Houston, Texas

1:30 P.M. DUMBBELL NEUROGENIC TUMORS

Dean Donahue, M.D., Massachusetts General Hospital Boston, Massachusetts

1:45 P.M. OMENTUM IN THE MANAGEMENT OF COMPLICATED THORACIC PROBLEMS

Joseph B. Shrager, M.D., University of Philadelphia School of Medicine, Philadelphia, Pennsylvania

2:00 P.M. DISCUSSION

SESSION V TRACHEOBRONCHIAL SURGERY

Moderator: Douglas J. Mathisen, M.D.

Boston, Massachusetts

2:20 P.M. CONGENITAL TRACHEAL STENOSIS

Hermes Grillo, M.D., Massachusetts General Hospital

Boston, Massachusetts

2:35 P.M. COMBINED BRONCHOPLASTY AND ANGIOPLASTY

Joachim Schirren, M.D., Dr. Horst-Schmidt Kliniken, Wiesbaden, Germany

2:50 P.M. CARINAL RESECTION FOR BRONCHOGENIC CANCER

Douglas J. Mathisen, M.D., Massachusetts General Hospital, Boston, Massachusetts

3:05 P.M. MANAGEMENT OF COMPLICATIONS OF BRONCHOLITHIASIS

L. Penfield Faber, M.D., Rush-Presby-St. Luke's Medical Center, Chicago, Illinois

3:20 P.M. DISCUSSION

SESSION VI SURGICAL INTERVENTION FOR RESPIRATORY DYSFUNCTION

Moderator: G. Alexander Patterson, M.D.

St. Louis, Missouri

3:45 P.M. ADULT ECMO

Robert H. Bartlett, M.D., University of Michigan Medical Center, Ann Arbor, Michigan

4:00 P.M. CURRENT STATUS OF LVRS

Douglas E. Wood, M.D., University of Washington Seattle, Washington

4:15 P.M. MANAGEMENT OF COMPLICATIONS OF LVRS

Bryan Meyers, M.D., Washington University Barnes Hospital, St. Louis, Missouri

4:30 P.M. MANAGEMENT OF GIANT BULLAE AND ALTERNATIVE SURGICAL THERAPIES FOR EMPHYSEMA

Peter Goldstraw, M.D., Royal Brompton Hospital London, England

4:45 P.M. DISCUSSION

5:10 P.M. ADJOURN - WELCOMING RECEPTION

EXHIBIT HALL

ADULT CARDIAC SURGERY SYMPOSIUM

SUNDAY, MAY 6, 2001 7:55 A.M. - 5:00 P.M.

SAN DIEGO CONVENTION CENTER

OBJECTIVE:

The Adult Cardiac Surgery Symposium for 2001 will build upon the foundation established last year by Chairman Bruce Lytle, where aortic valve replacement and coronary artery surgery were covered exhaustively. This year the symposium will focus in depth on four different broad topics. Video segments depicting surgical technique and illustrative echo imaging will be part of certain presentations.

1.) Mitral valve surgery and what surgeons need to know about interpretation of intraoperative transesophageal echocardiography. Divergent surgical approaches will be covered, in additional to the question of whether or not the choice of type of prosthesis or bioprostheses selected has any meaningful impact on late patient outcome.

2.) An update on biostatistics for cardiovascular surgeons will emphasize fresh statistical tools for the assessment of cardiac surgical clinical outcomes and innovative, more meaningful ways to compare surgical clinical outcomes and innovative, more meaningful ways to compare surgical treatment methods in terms of non-fatal postoperative complications.

3.) Non-transplant, new surgical methods to treat advanced left ventricular (LV) dysfunction and congestive heart failure will be addressed, including the rationale behind these concepts and early clinical results. This topic will also include the continuing dilemma posed by patients with ischemic mitral regurgitation (IMR), who also invariably present with some degree of LV failure. The surgical alternatives of repair (ring annuloplasty) or replacement will be discussed in the perpective of individual patient decision-making. Novel, creative applications of echocardiography to quantitate the hemodynamic burden of the valvular regurgitant lesion, *e.g.,* MR, and to characterize the mechanistic basis of IMR in patients will be comprehensively covered.

Finally, continuing dilemmas in the field of thoracic aortic surgery will be presented, including ways to minimize the devastating complications of stroke and paraplegia. Valve-sparing aortic root replacement is gaining popularity, but the choice of which technique is superior continues to evolve over time. Whether to reimplant the aortic valve inside a Dacron tube graft ("T. David-I procedure"), to remodel the aortic root ("Yacoub procedure"), or some hybrid combination coupled with aortic annuloplasty will be discussed, including specific patient criteria which favor one technique over the other.

At the end of the day, you should have a better grasp of the rationale and details of surgical technique used today for patients with mitral regurgitation arid those with CHF due to LV dilatation and systolic dysfunction from various causes. You will have a comprehensive working knowledge of what to expect from echocardiography, including appreciation of the pitfalls inherent in the intra-operative use of TEE. You will also be exposed to new statistical tools which will help you decide more intelligently when reading the medical and surgical literature if one treatment method or surgical technique is better than, equivalent to, or inferior to another approach. And, you will be aware of the continuing challenges and ‘state-of-the-art' in treating patients with complicated, thoracic aortic aneurysms and aortic dissections.

ACCREDITATION

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The

American Association for Thoracic Surgery designates this continuing education activity for 7 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

PROGRAM

7:00 A.M. REGISTRATION AND CONTINENTAL BREAKFAST

7:55 A.M. INTRODUCTION:

D. Craig Miller, M.D., Chairman

SESSION I MITRAL VALVE SURGERY AND ECHOCARDIOGRAPHY

8:00 A.M. VALVULAR SURGERY - WHAT SURGEONS NEED KNOW ABOUT INTRA-OPERATIVE TEE

Maurice E. Sarano, M.D., Mayo Clinic, Rochester, Minnesota

8:30 A.M. MVR - DOES CHOICE OF VALVE PROSTHESIS OR BIOPROSTHESIS INFLUENCE LONG-TERM OUTCOME?

Bruce W. Lytle, M.D., The Cleveland Clinic Foundation Cleveland, Ohio

9:00 A.M. IS THERE STILL A ROLE FOR 'MAXIMALLY INVASIVE' MITRAL VALVE SURGERY?

Francis C. Wells, M.D., Papworth Hospital, Cambridge, England

9:30 A.M. MINIMALLY INVASIVE' MITRAL SURGERY - PASSING FAD OR HERE TO STAY?

†Delos M. Cosgrove, M.D., The Cleveland Clinic Foundation Cleveland, Ohio

10:00 A.M. PANEL DISCUSSION, Q & A

10:30 A.M. BREAK

SESSION II BIOSTATISTICS FOR CARDIOVASCULAR SURGEONS

11:00 A.M. ANALYSIS OF CLINICAL OUTCOMES: HELPFUL NEW STATISTICAL APPROACHES YOU NEED TO UNDERSTAND BETTER

Eugene H. Blackstone, M.D., The Cleveland Clinic Foundation, Cleveland, Ohio

11:20 A.M. MORE MEANINGFUL INTERPRETATION OF NON-FATAL EVENTS AFTER CARDIAC SURGERY - ACTUAL VERSUS ACTUARIAL REPORTING

Gary L. Grunkemeier, M.D., Providence Health System Portland, Oregon

11:40 A.M. PANEL DISCUSSION, Q & A

12:00 P.M. LUNCHEON

SESSION III SURGICAL TREATMENT OF LV DYSFUNCTION/CHF AND ISCHEMIC MITRAL REGURGITATION

1:00 P.M. ECHO QUANTITATION OF MR ("ERO") AND INSIGHTS INTO THE MECHANISMS OF IMR

Maurice E. Sarano, M.D., Mayo Clinic, Rochester, Minnesota

1:30 P.M. ISCHEMIC MITRAL REGURGITATION - A SPECTRUM OF DISEASES. WHICH OPERATION FOR WHICH PATIENT?

†Patrick Perier, M.D., Institut Arnault Tzanck Saint Laurent du Var Cedex, France

2:00 P.M. RATIONALE BEHIND NEW NON-TRANSPLANT SURGICAL APPROACHES TO ADVANCED LV DYSFUNCTION AND CHF

Gerald D. Buckberg, M.D., U.C.L.A. Medical Center

Los Angeles, California

2:30 P.M. PANEL DISCUSSION, Q & A

3:00 P.M. BREAK

SESSION IV PERSISTING DILEMMAS IN THORACIC AORTIC SURGERY

3:30 P.M. PROXIMAL AORTIC SURGERY - HOW BEST TO PROTECT THE BRAIN

Randall B. Griepp, M.D., Mount Sinai Medical Center New York, New York

3:50 P.M. DESCENDING THORACIC AND THORACOABDOMINAL AORTIC SURGERY - MINIMIZING STROKE AND PARAPLEGIA

Lars G. Svensson, M.D., Lahey Clinic Medical Center Burlington, Massachusetts

4:10 P.M. CHANGING CHOICE OF TECHNIQUES FOR VALVE-SPARING AORTIC ROOT REPLACEMENT

Tirone E. David, Toronto General Hospital Toronto, Ontario, Canada

4:30 P.M. PANEL DISCUSSION, Q & A

5:00 P.M. ADJOURN - WELCOMING RECEPTION

EXHIBIT HALL

†*Presenter has a relationship with Edwards Life Science*

CONGENITAL HEART DISEASE SYMPOSIUM

SUNDAY, MAY 6, 2001 8:00 A.M. - 5:00 P.M.

SAN DIEGO CONVENTION CENTER

OBJECTIVE

The 2001 AATS Congenital Heart Disease Symposium will be divided into three sessions, each one addressing different aspects of complex congenital heart surgery. The first part of the symposium is a video session, which will address different techniques for achieving operative repairs involving VSD closure, AV canal repair, and Ebstein's anomaly. In addition two important video presentations will showcase the Ross operation and options for repair of double outlet right ventricle with uncommitted VSD. The presenters will discuss their preferences and operative results.

The second session will be devoted to the complications associated with the Fontan operation. These lecture and discussion sessions will include techniques to treat complications associated with protein losing enteropathy, arrhythmias, heterotaxy syndrome, and assessment of anticoagulation strategies. The presenters will focus on their experience and review of the literature.

The third session will discuss new techniques and strategies for the initial and subsequent treatment of patients with truncus arteriosus. Neonatal truncus arteriosus repair including methods to treat truncal valve insufficiency and interrupted aortic arch will be emphasized. Long-term follow-up will also be discussed which will center on conduit replacement options.

At the completion of this symposium the participants should have attained an enhanced appreciation of how different methods can be used to successfully treat the same congenital defect. In addition, participants will be exposed to the current strategies to treat the complications of the Fontan operation as well as the current status of Truncus Arteriosus reepair.

ACCREDITATION

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PROGRAM

7:00 A.M. REGISTRATION AND CONTINENTAL BREAKFAST

8:00 A.M. INTRODUCTION AND WELCOME

Constantine Mavroudis, M.D., Chairman

SESSION I OPERATIVE VARIATIONS FOR SELECTIVE CONGENITAL HEART DEFECTS (VIDEO PRESENTATIONS)

Moderator: Constantine Mavroudis, M.D

8:05 A.M. VSD CLOSURE: RUNNING SUTURE TECHNIQUE

Edward L. Bove, M.D., University of Michigan Hospital

Ann Arbor, Michigan

8:25 A.M. VSD CLOSURE: INTERRUPTED SUTURE TECHNIQUE

Constantine Mavroudis, M.D., Children's Memorial Hospital Chicago, Illinois

8:45 A.M. DISCUSSION

8:55 A.M. DOUBLE OUTLET RIGHT VENTRICLE WITH UNCOMMITTED VSD

Francois Lacour-Gayet, M.D., Marie Lannelongue Hospital Paris, France

9:15 A.M. THE ROSS OPERATION

Ronald Elkins, M.D., University of Oklahoma Health Science Center, Oklahoma City, Oklahoma

9:35 A.M. DISCUSSION

9:45 A.M. BREAK

10:05 A.M. AV CANAL REPAIR: SINGLE PATCH TECHNIQUE

John E. Mayer, Jr., M.D., Children's Hospital

Boston, Massachusetts

10:25 A.M. AV CANAL REPAIR: TWO PATCH TECHNIQUE

Carl L. Backer, M.D., Children's Memorial Hospital Chicago, Illinois

10:45 A.M. AV CANAL REPAIR: WITHOUT VSD PATCH

Graham Nunn, M.D., Sydney Children's Hospital Sydney, Australia

11:05 A.M. AV CANAL REPAIR: WITH DORV OR TGA

Christo I. Tchervenkov, M.D., The Montreal Childrens Hospital, Montreal, Quebec, Canada

11:25 A.M. DISCUSSION

12:00 P.M. LUNCHEON

1:00 P.M. EBSTEIN'S ANOMALY OF THE TRICUSPID VALVE REPAIR

Gordon K. Danielson, M.D., Mayo Clinic Rochester, Minnesota

1:20 P.M. EBSTEIN'S ANOMALY OF THE TRICUSPID VALVE REPAIR

Alain Carpentier, M.D., European Medical Center-George Pompidou, Paris, France

1:40 P.M. DISCUSSION

SESSION II FONTAN COMPLICATIONS

Moderator: Pedro J. del Nido, M.D.

2:00 P.M. STRATEGIES TO TREAT PROTEIN LOSING ENTEROPATHY

Thomas L. Spray, M.D., Children's Hospital of Pennsylvania Philadelphia, Pennsylvania

2:20 P.M. DISCUSSION

2:25 P.M. MANAGEMENT OF AORTOPULMONARY COLLATERAL ARTERIES IN FONTAN PATIENTS

Scott M. Bradley, M.D., Medical University of South Charleston, South Carolina

2:45 P.M. DISCUSSION

2:50 P.M. COMPLICATIONS ASSOCIATED WITH HETEROTAXY SYNDROME

Marshal Jacobs, M.D., St Christopher's Hospital for Children Philadelphia, Pennsylvania

3:10 P.M. DISCUSSION

3:15 P.M. FANTICOAGULATION STRATEGIES AFTER THE FONTAN OPERATION

Paul Monagle, M.D., Melbourne, Australia

3:25 P.M. DISCUSSION

3:30 P.M. BREAK

SESSION III TRUNCUS ARTERIOSUS

Moderator: John Lamberti, M.D.

4:00 P.M. NEONATAL TRUNCUS ARTERIOSUS REPAIR

Frank L. Hanley, M.D., University of California - San Francisco, San Francisco, California

4:20 P.M. DISCUSSION

4:30 P.M. CONDUIT REPLACEMENT STRATEGIES FOR TRUNCUS ARTERIOSUS

John W. Brown, M.D., Indiana University

Indianapolis, Indiana

4:50 P.M. DISCUSSION

5:00 P.M. WELCOMING RECEPTION: EXHIBIT HALL

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
81st ANNUAL MEETING
San Diego Convention Center
San Diego, California, May 6-9, 2001

MONDAY, MAY 7, 2001

8:00 a.m. BUSINESS SESSION (Limited to Members)

8:15 a.m. SCIENTIFIC SESSION

Room 6A-C, San Diego Convention Center

Moderators: James L. Cox; Tirone E. David

1. Surgical Anterior Ventricular Endocardia! Restoration in the Dilated Remodeled Ventricle Following Anterior Myocardial Infarction

Constantine L. Athanasuleas*, Alfred W.H. Stanley*, Gerald D. Buckberg, Vincent Dor, Marissa Di Donato*, Eugene H. Blackstone, Restore Group; Birmingham, AL; Los Angeles, CA; Monte Carlo, Monaco; Florence, Italy; Cleveland, OH
Discussant: Henry M. Spotnitz

OBJECTIVE: Anterior myocardial infarction (AMI) changes ventricular shape and volume. Dyskinesia develops without reperfusion, whereas akinesia follows thrombolysis or PTCA. Dysfunction of remaining remote muscle causes dilatation and congestive heart failure (CHF). The SAVER (surgical anterior ventricular endocardial restoration) procedure excludes non-contracting anterior and septal segments, including akinetic scar that is not normally approached surgically. An international team evaluated safety and efficacy of SAVER in CHF patients.

METHODS: Cardiologists and surgeons in 11 centers on 4 continents (USA, Europe, Asia, South America) comprised the RESTORE GROUP, and performed SAVER in 586 patients (average 4.6 years post AMI) from January 1998, to August 2000. Concomitant procedures included CABG in 91%, mitral valve repair in 23%, and mitral replacement in 3%. Morphology included akinesia in 68% and dyskinesia in 32%.

RESULTS: Overall mortality was 6.7% (39 patients), and unaffected by the addition of mitral valve repair. Mechanical support was needed in only 9%, including IABP (7.8%), LVAD (0.5%), or ECMO (0.7%). Postoperative ejection fraction (EF) increased from 29.5 ± 11 to 40.0 ± 12, left ventricular end systolic volume index (LVESVI) decreased from 98 ± 95 to 64 ± 40 ml/m^2 ($p<$ .005). There were 20 post-discharge deaths or 10% total mortality. Risk factors for death included older age, and lower postoperative EF.

CONCLUSIONS: SAVER is a safe and effective operation in the treatment of CHF patients with a remodeled dilated ventricle following AMI. It excludes septal and anterior segments, which are either dyskinetic or akinetic (most common). Operative mortality was low with few patients requiring intraoperative mechanical support. Systolic function improved postoperatively. Intermediate follow-up (31 month) results are encouraging.

**By Invitation*

2. Total Cavopulmonary Conversion and Maze Procedure for Patients with Failed Fontan

Constantine Mavroudis, Barbara]. Deal*, Carl L. Backer, Christopher L.

Johnsrude*, Scott H. Buck*; Chicago, IL

Discussant: Scott Bradley

OBJECTIVE: Hemodynamic abnormalities and refractory atrial arrhythmias in late post-op Fontan patients (pts) result in significant morbidity and mortality. We review our experience with conversion to total cavopulmonary artery connections (TCPC) and arrhythmia surgery.

METHODS: From 1994 through 2000, 38 pts underwent Fontan conversion and arrhythmia surgery. Significant hemodynamic lesions such as aortic aneurysm (n=1), A-V valve insufficiency (n=8), and pulmonary stenosis (n=8) were repaired concomitantly. 29 pts were in NYHA class III or IV. Mean age at original Fontan was 7.52 + 6.5 yrs and mean age at Fontan conversion was 18.7 + 9.0 yrs. Arrhythmia surgery has evolved from isthmus cryoablation in 8 pts to right-sided Maze in 20 pts for atrial reentry tachycardia. Maze-Cox III was used for 10 patients with atrial fibrillation (AF). Atrial (N=32) and dual chamber (n=4) pacemakers were placed.

RESULTS: There has been no early or late mortality. Three pts required cardiac transplantation at 1 week, 7 months, and 3 yrs post-op. Chest tubes were removed on post-op day 9.5 ± 6.5. Hospital stay was 11±5 days. Mean follow up is 34 months. Three pts from the early isthmus cryoablation period had arrhythmia recurrence (8%) requiring one medication. All pts are currently NYHA Class I or II.

CONCLUSIONS: Fontan conversion to TCPC with concomitant arrhythmia surgery is excellent therapy for pts with failed Fontan. Fontan conversion is safe, improves NYHA class, and has a low incidence of recurrent arrhythmias.

Dwight Harken Research
Scholar Presentation
Bruce Rosengard, Philadelphia, Pennsylvania

**By Invitation*

3. Gain and Subsequent Loss of FEV1 after Lung Volume Reduction Surgery in Severe Emphysema with Different Morphology

Walter Weder*, Carmina L. Georgescu*, Erich W. Russi*, Konrad E. Bloch*; Zurich, Switzerland

Discussant: Joel D, Cooper

OBJECTIVE: Benefit from surgical lung volume reduction (LVR) is greatest in markedly heterogeneous emphysema and less pronounced in the homogeneous type. We investigated whether subsequent loss of function depends on morphology as well.

METHODS: 81 patients (46 men, mean ±SE age 63 ± 10y) with >1 y follow-up after bilateral thoracoscopic LVR were studied at 3,6 months, and then half yearly. They were grouped according to a CT-based morphological emphysema grading system (Ann Thorac Surg 1997;64:313).

RESULTS: Mean ±SE follow-up was 30 ± 2months (12 to 66) and similar among groups. The time course of FEV1 is summarized in the table. Loss refers to the mean decline in FEV1 over time after initial maximal gain (ml/y), and to the mean decline in % of maximal gain (%/y).

CONCLUSIONS: Markedly heterogeneous emphysema was associated with larger initial improvement but also with more rapid subsequent loss in FEV1 than homogeneous or intermediately heterogeneous emphysema. However, during a comparable observation time, the relative yearly decline in FEV1 was independent of emphysema heterogeneity.

Time Course of FEV1 After LVR

Emphysema Heterogeneity	n	FEV1 pre LVR (L)	3 m post (L)	Loss (ml/y)	Loss (%/y)
homogeneous	21	.71 ± .04	.99 ± .06*	123 ± 3	12 ± 2
intermediate	20	.67 ± .03	.98 ± .06*	128 ± 3	12 ± 3
heterogeneous	40	.84 ± .04 §	1.4 ± .08* +	168 ± 2 #	12 ± 1

P<0.05: § vs. intermediate; * vs. preop.; + vs. homog.; vs. homog. & intermed.

48th Evarts A. Graham Memorial Traveling

Fellowship Presentation

Albertus Scheule, Tuebingen, Germany

**By Invitation*

4. Appropriate Timing of Surgical Intervention after Transmural Acute Myocardial Infarction

Daniel C. Lee*, [1]Mehmet C. Oz, Alan D. Weinberg*, Windsor Ting*; New York, NY

Discussant: Robert A. Guyton

OBJECTIVE(s): Recommended timing of coronary revascularization (CABG) after transmural acute myocardial infarction (AMI) ranges from immediate surgery to 4 weeks post MI. Such wide variation created dilemma in the management of this patient cohort. The objective of this study is to delineate optimal timing of CABG after transmural AMI in a large and contemporary patient population.

METHODS: A retrospective multicenter analysis of 22,984 patients who underwent CABG after transmural myocardial infarction between 1993 and 1996 by 179 surgeons at 32 hospitals in New York State.

RESULTS: Overall hospital mortality for all patients who underwent CABG with history of transmural myocardial infarction was 3.1%. The table below revealed that hospital mortality decreased with increasing time interval between CABG and transmural AMI. Multivariate analyses of 37 risk factors confirmed that CABG within 3 days of transmural AMI is an independent risk factor for mortality.

CONCLUSIONS: After transmural AMI, a waiting period before CABG should be considered if feasible, since surgery within 3 days is an independent risk factor for mortality. If surgery cannot

be delayed, surgeons must be ready to utilize aggressive cardiac support such as left ventricular assist devices in this ailing population.

Time between CABG	Mortality	Multivariate Analysis**	
and Transmural AMI		Odds Ratio	p-value
<6 Hours (n=406)	12.1%*	1.8	0.005
6-23 Hours (n=251)	13.6%*	2.7	<0.001
1-3Days (n=718)	6.8%*	1.5	0.024
4-7 Days (n=2343)	3.5%	1.1	0.658
8-14 Days (n=299l)	2.4%	0.8	0.135

*p<0.05 when compared to >7 days. **Portion pertaining to timing shown here.

9:45 a.m. INTERMISSION - VISIT EXHIBITS

[1]*1994-96 AATS Research Scholar*

**By Invitation*

10:30 a.m. SCIENTIFIC SESSION

Room 6A-C, San Diego Convention Center

Moderators: Timothy J. Gardner; Tirone E. David

5. Hypoplastic Left Heart Syndrome is Not a Risk Factor for Death after the Fontan Operation

Thomas L. Spray, J. William Gaynor*, Nancy D. Bridges*, Mitchell I. Cohen*, William T. Mahle*, William M. Decampli*, James M. Steven*, Susan C. Nicolson*; Philadelphia, PA

OBJECTIVE: To evaluate factors contributing to a decrease in morbidity and mortality following the Fontan procedure between 1/1/92-12/31/99.

METHODS: Outcomes evaluated were survival and duration of pleural effusions and hospitalization. Potential predictors of survival were evaluated using logistic regression and Cox regression used for duration of effusions and hospitalization. Predictors evaluated included anatomic diagnosis, presence of common AV valve, staging, year of Fontan, type of Fontan, fenestration, duration of cardiopulmonary bypass and deep hypothermic circulatory arrest (DHCA), and use of modified ultrafiltration (MUF).

RESULTS: The Fontan procedure was performed in 332 pts: Group I (1992-1995, n=198) and Group II (1996-1999, n=134). Hypoplastic left heart syndrome (HLHS) was present in 176 pts (53%) and Stage I reconstruction performed in 210 (63%). Interim superior cavo-pulmonary shunt was performed in 318 pts (96%). Median age was 22 mos (range 11-380) and median weight 10.9 kg (range 5.8-120). Lateral tunnel Fontan was performed in 281 pts and Extracardiac Fontan in 51. A fenestration was created in 298 pts (90%). Mortality for Group I was 10.6% and 0.8% for Group II. Median duration of pleural drainage for Group I was 5 days (range 1-258) with 55 pts > 14 days (28%) and for Group II was 1 day (range 1-29) with 7 pts > 14 days (5%). Median duration of hospitalization was 14 days (range 1-278) for Group I and 6 days (range 2-56) for Group II. MUF

was introduced in 1995 and was collinear with year of Fontan, surgeon, and type of fenestration. However, in a multivariable analysis of the entire cohort, the model which best predicted death included only non-use of MUF (OR 15.75, 95% CL 1.96-127, p=0.01), longer DHCA (OR 1.04/min > 17,95% CL 1.01-1.07, p=0.006), and presence of a common AV valve (OR 6.79,95% CL 2.12-21.75, p=0.001). The model which best predicted absence of prolonged pleural effusions contained only use of MUF (OR 2.85,95% CL 2.08-3.89, p<0.001).

CONCLUSIONS: In a contemporary series of Fontan operations composed largely of patients with HLHS or variants, operative factors, rather than anatomic diagnosis, determined mortality and morbidity.

**By Invitation*

6. Mitral Valve Repair with Carpentier's Techniques. The Third Decade.

Alain Deloche*, Alain Carpentier, Sylvain Chauvaud, Eric Braunberger*, Alain Berrebi*, Jean-Pierre Marino*, Jean-Noel Fabiani*, John Ym Relland*; Paris, France

Discussant: Magdi Yacoub

OBJECTIVE: Carpentier valve repair techniques are recognized to provide a high degree of reliability. However the very long term stability remained to be evaluated. This study analyses the early series of patients (pts) with 15 to 30 years follow-up.

METHODS: From 1970 to 84, 434 pts underwent reconstructive surgery for isolated severe mitral valve insufficiency. There were 272 (63%) rheumatic disease (group A: GA), 146 (34%) degenerative (group B: GB) and 16 endocarditis (3%) (group C: GC). Mean age was 28±12 years in GA, 57±9 in GB and 48±16 in GC. Atrial fibrillation (AF) was present in 54% in GA, 39% in GB and 44% in GC. Predominant valve dysfunction in GA was leaflet prolapse in 47% and restricted leaflet motion in 74% whereas in GB leaflet prolapse was present in 99.3%. A remodelling annuloplasty ring was used in 95% of the cases, additional procedures were necessary in 92% of the cases.

RESULTS: The hospital mortality was 3% in GA, 1.4% in GB, 6.2% in GC. Follow-up (FU) was complete in 96% with 6619 pts/yr. Mean FU was 17 years with a maximum of 29 years. Actuarial survival at 15, 20, 25 yrs were 97,92 and 75% in GA, 55,44 and 27% in GB. Freedom from reoperation at 15, 20 and 25 yrs was 70, 52 and 40% in GA, 93,93 and 93% in GB. The actuarial reoperation rate was 2.2%/pt/yr in GA, 0.5%/pt/yr in GB, 0.4%/ pt/yr in GC. At reoperation, the valve was re-repaired in 6% in GA and in 36% in GB. At the time of this study 42% were in AF in GA, 27% in GB. Despite the fact that only 70% of the patients in AF received anticoagulants, the cumulative rate of thromboembolic and hemorrhage events was 0.2% in GA, 0.3% in GB.

CONCLUSIONS: The long term stability of mitral valve repair is strongly influenced by the etiology of the valvular disease and not by the complexity of the repair. Twenty years after valve repair, valve replacement had been necessary in 48% of rheumatic pts and only 6.2% of degenerative pts. This remarkable stability is thought to be due to the complete remodelling annuloplasty ring and to the primary repair of all the lesions. Remarkable also is the very low incidence of valve related mortality and thrombo-hemorrhagic complications which makes this type of valve repair preferable to valve replacement even in rheumatic disease.

**By Invitation*

11:15 a.m. PRESIDENTIAL ADDRESS - Changing Boundaries

James L. Cox, Washington, D.C.
Introduced By: Timothy J. Gardner, Philadelphia, PA

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

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

Room 6A-C, San Diego Convention Center

Moderators: D. Craig Miller; Verdi J. Di Sesa

†7. Beating Heart Techniques Improved Outcomes in Coronary Artery Bypass Crafting

Michael J. Mack, Mitchell J. Magee*, James R. Edgerton*, Todd M. Dewey*, Syma L. Prince*, Tea Acuff*; Dallas, TX

Discussant: Antonio M. Calaflore

OBJECTIVE: Improved outcomes in select patients undergoing coronary artery bypass grafting (CABG) by elimination of cardiopulmonary bypass (CPB) has been demonstrated. Benefit in all patients undergoing CABG has yet to be proven.

METHODS: We reviewed our experience with beating heart surgery from January 1995 through June 2000.11,405 patients underwent isolated CABG including 1,588 (13.9%) performed without CPB. Groups were compared by uni variate analysis for preop risk factors and postop complications and predicted risk was determined by the STS risk algorithm

RESULTS: The mortality in all patients was 3.22%. There was a significant difference in the observed mortality between the off pump group 1.89% (mean predicted mortality 3.11%) compared to the on pump group 3.65% (mean predicted 2.81%, p=.0003). Additionally, decreased morbidity in the off pump group was evidenced by reduced need for blood products (28.45% vs. 54.65%, p=.0001), prolonged ventilation (5.83% vs. 10.93%, p=.0001), less reoperation for bleeding (2.41% vs. 3.65%, p=.0237) and shorter hospital stay (5.98 vs. 7.32 days, p=.0001).

CONCLUSIONS: Beating heart surgery can be assimilated into a surgical practice. Gradual integration leads to improved outcomes in the CABG population.

	1995	**1996**	**1997**	**1998**	**1999**	**2000 (6mos)**
Total	1635	2084	2110	2208	2137	1231
Off Pump AB	21	111	148	289	631	388
% Off Pump	1.3	5.3	7.5	15	29.5	31.5
On Pump	3.04/NA	3.70/2.78	3.72/2.82	3.70/2.85	3.59/2.70	4.52/3.06
Mortality Obs/Predicted		8	2	5	0	
Off Pump	0/NA	1.80/2.60	1.99/2.79	2.77/3.36	1.74/3.19	1.29/2.63
Moratlity Obs/Predicted		0	9	6	9	

*By Invitation

†Authors have a relationship with Medtronic & Guidant

8. A Prospective Randomised Comparison of CarboMedics and St Jude Medical Mechanical Heart Valve Protheses: an Interim Report.

Kelvin H.H. Lim*, Massimo Caputo*, Raimondo Ascione*, Janet M. Wild*, Robert West*, Gianni D. Angelini*, Alan J. Bryan*; Bristol, United Kingdom
Discussant: Gary W. Akins

OBJECTIVE: A prospective randomised comparison of the clinical performance of CarboMedics (CM) and St Jude Medical (SJM) heart valve prostheses over a projected 10 year period. An interim report is presented at mid term follow-up.

METHODS: Between 1992 and 1996, 485 patients undergoing mechanical heart valve replacement were prospectively randomised to receive either CM (234) or SJM (251) for aortic (288), mitral (160) or double (37) valve replacements. Annual follow-up was undertaken prospectively, using patient questionnaire, general practitioner contact and hospital notes.

RESULTS: There was no difference in the two groups with respect to pre-operative age, sex, NYHA status, coronary artery disease, previous surgery, incidence of arrhythmias and history of thromboembolic events. There was no difference in cardiopulmonary bypass and ischemic times, myocardial protection and valve suture technique. Mean follow-up was 49.8122.1 months for CM and 47.5±19.5 months for SJM, giving 1965 patient-years in total. Complete follow up was 97% and 96% for the CM and SJM groups respectively. Thirty-day mortality and 5-year actuarial survival (and composite linearised rates) were 5.6% and 82.5±2.6% (4.22%) in the CM group, and 4.4% and 80.7±2.7% (4.63%) in the SJM group (p=NS). Freedom from valve-related mortality at 5 years was 86.3±2.3% (3.19%) and 85.4±2.3% (3.42%) for the CM and SJM groups respectively. Freedom at 5 years from major/fatal thromboembolism, haemorrhage, and other non-structural valve dysfunction were, respectively, 91.1±2.1% (1.96%), 87.3±2.5% (3.6%), 95.2±1.5% in the CM group and 92.1±1.8% (2.01%), 82.6±2.8% (4.33%), 93.5±1.6% in the SJM group, with no overall inter-group differences. Anticoagulation was monitored throughout and no significant differences in mean INR values were detected between the CM and SJM groups.

CONCLUSIONS: This prospective randomised study shows no significant differences in the early and midterm clinical outcome in patients who received CM or SJM mechanical valve prostheses. Choices with respect to valve type can be based on consideration other than patient outcome.

*By Invitation

9. Prospective Randomized Study of Bioglue Tissue Adhesive During Repair of Acute Type A. Aortic Dissection

Joseph E. Bavaria, Alberto Pochettino*, Derek R. Brinster*, Robert C. Gorman*, Thomas Wozniak*, Timothy J. Gardner, John Fehrenbacher*; Philadelphia, PA; Indianapolis, IN
Discussant: Jean A Bachet

OBJECTIVE: This study presents the results of two institutions of a prospective, randomized trial comparing outcomes after surgical repair of acute Type A Dissection using Bioglue, a serum albumin and glutaraldehyde tissue adhesive, versus a standard control cohort.

METHODS: Over a 17 month period from 8/25/98 to February, 2000, 35 (17 bioglue and 18 control) patients at two institutions were randomly assigned Bioglue with standard repair versus standard repair alone. All patients underwent repair using an open arch technique, antegrade graft perfusion, and resuspension of the aortic valve. Cerebral protection strategies were equally applied to both the Bioglue and control groups. 66% of patients were male, 16% of patients had concomitant CABG and 11% had redo sternotomies.

RESULTS: In hospital mortality was 17%. There were three (8.6%) late deaths during the 2 year follow up. There was no statistical significance in perioperative or late deaths between groups. Study results are presented in the following table:

CONCLUSIONS: This study shows that the use of serum albumin/ glu-taraldehyde tissue adhesive (Bioglue) as an adjunct to the repair of acute Type A aortic dissections has resulted in significantly shorter periods of circulatory arrest, CPB, proximal aortic reconstruction, and total operative time. Utilizing Bioglue to obliterate the false lumen and seal the adventitia and intima at the aortic arch and root may obviate the need for felt during dissection repair thereby allowing more rapid proximal aortic reconstruction.

	Bioglue (minutes)	Control (minutes)	P value
Time for Aortic Repair	211	295	0.026
Circulatory Arrest	32	46	0.018
Cardiopulmonary bypass	170	197	0.067
Total OR time	298	346	0.099

**By Invitation*

10. Pacopexy-New Restoration Procedure for Non-ischemic Dilated Cardiomyopathy

Hisayoshi Suma, Friedhelm Beyersdorf, Sergio De Oliveira*, Constantine L. Athanasuleas*, Francisco Torrent-Guasp*, Restore Group, Gerald D. Buckberg; Kanagawa, Japan; Freiburg, Germany; Sao Paulo, Brazil; Birmingham, AL; Denia, Spain; Los Angeles, CA
Discussant: Patrick M. McCarthy

OBJECTIVE: Congestive heart failure follows changing normal LV elliptical shape to spherical configuration. An international team (Asia, Europe, South America, USA) evaluated a novel geometric LV configuration procedure (Pacopexy) where apical vortex in failing heart is restored by rebuilding more natural LV muscle loop configuration.

METHODS: Nineteen patients with non-ischemic idiopathic dilated car-diomyopathy (DCM), E.F. 21 ± 4%, LVESVI 168 ± 30 ml/m^2 (normal 25 ± 10 ml/m^2) and CHF, NYHA III/IV underwent LV restoration without muscular resection. Pacopexy procedure created an intraventricular curtain to bisect the spherical chamber to restore the elliptical shape. An intraventricular patch (hemishield or pericardium) was placed between anterior papillary muscle and the LV septum just beneath aortic valve. Conical ventriculectomy closure covered the patch with excluded muscle.

RESULTS: Perioperative course included no LVAD, two IABP, and only one of two deaths (11%) from cardiac causes. Initial ejection fraction increased from 21 to 34%, LVESVI fell from 169 to 94 ml/m^2, and NYHA class improved from 3.6 to 1.8. Follow-up was ~14 months. The underlying myocyte cause of IDCM (including sarcoidosis, muscular dystrophy, viral disease) persisted, contributing to recurrent dilatation and 5 late deaths (26%): two from ventricular tachyarrhythmia. Three patients are on transplant list (16%). Survival was highest (100%) with NYHA < II in 7/19 patients if LVESVI < 150 ml/m^2, implying benefit from early intervention.

CONCLUSIONS: The procedure restores LV elliptical vortex from a CHF sphere and initial response to Pacopexy is safe and effective. However, underlying IDCM may progress with recurrent dilatation to prevent success in small subset with *intrinsically* abnormal DCM myocytes. Pacopexy may become more effective in larger worldwide subset with *more normal* myocytes by H/E stain; valvular disease with aortic or mitral insufficiency. Despite ensuring valve competence, postop 10-year mortality is 60% with E.F. < 40%, without LV restoration.

**By Invitation*

11. Mortality Data in the journals May Create Unrealistic Expectations

Ani C. Anyanwu*, Tom Treasure*; London, United Kingdom

Discussant: T. Bruce Ferguson, Jr.

OBJECTIVE: To test the hypothesis that published series are biased towards reporting lower mortality than is achieved in practice in most centers.

METHODS: Papers from single centres quoting inter alia mortality for coronary artery bypass (CABG), aortic (AYR) and mitral (MVR) valve replacement in the three major international cardiothoracic journals were identified for three years from 1997 through 1999. Mortality data were compared to that in National Registries in the United States and United Kingdom. Only papers with 50 or more subjects from single centers were considered.

RESULTS: 125 papers were identified (CABG 82, AYR 30, MVR 13). Papers were predominantly case series (75), randomized trials (22), retrospective comparative (20) and prospective studies (8). Reporting institutions were North American (53), European (57) and other (15). Median survival reported in these studies compared with the registries is shown in Table. 21 papers (18 CABG, 2 AYR and 1 MVR) reported 0% mortality. CABG studies with sample size <100 reported lower mortality (median 0%) compared with those > 100 (1.8%). Studies below the 25th centile of mortality distribution often had wide confidence intervals around the mortality figure which were rarely supplied. Exploration with graphical plots suggests bias towards reporting/publication of studies with below average mortality.

CONCLUSIONS: Published data particularly for CABG tend to under-represent the true risk of death. Outcomes and therefore the magnitude of effects reported in research studies are not necessarily representative of those experienced in routine practice and may distort expectations of patients and physicians. Caution should be taken in extrapolating results from studies with less than 100 patients to other surgical populations

% Mortality:	**LITERATURE (median IQR)**	**STS REGISTRY**	**UK REGISTRY**
CABG	**1.3** (0.3-2.6)	**2.9**	**2.6**
AVR	**3.4** (2.5-5.3)	**4.0**	**4.5**

MVR	**4.4** (2.7-7.0)	**6.0**	**6.3**

3:20 p.m INTERMISSION - VISIT EXHIBITS

**By Invitation*

4:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION A-2 -ADULT CARDIAC SURGERY

Room 6A-C, San Diego Convention Center

Moderators: D. Craig Miller; Verdi J. Di Sesa

12. Mitral Valve Repair with Advanced Myxomatous Degeneration

Catarina Bitkover*, Tirone E. David, Susan Armstrong*, Joan Ivanov*, Harry Rakowski*, Maria Erikkson*; Toronto, ON, Canada

Discussant: Alain F. Carpentier

OBJECTIVE: We recently found that massive annular dilation in patients with advanced myxomatous degeneration is often associated with posterior displacement of the mitral annulus combined with thinning and scarring of the first 5 to 10 mm of proximal ventricular wall. We believe standard annuloplasty does not address this problem. Correction requires detachment of the entire posterior leaflet from the annulus and plication of the thinned ventricular wall and displaced annulus with multiple horizontal mattressed sutures, reinforced by an annuloplasty ring. Prolapse of the leaflets is corrected by established techniques. This study examines the long-term results of mitral valve (MV) repair in patients with advanced myxomatous degeneration (voluminous and prolapsing leaflets with mitral annulus diameter >45 mm) and the potential role of annular displacement on outcomes.

METHODS: From 1982 to 1999,113 patients with advanced myxomatous degeneration underwent MV repair. The mean age was 53±13 years and 71% were male. Twenty-five patients (22%) had Marfan syndrome and 12 (10%) had heavily calcified annulus. Prolapse of the posterior leaflet was present in 92% of patients and the anterior leaflet in 70%. The mean follow-up was 5.3±3.4 years and was complete. All patients had echocardiography. Time-related variables were evaluated by Kaplan-Meier analysis.

RESULTS: Of 113 patients, standard annuloplasty was performed in 81 and repair of annular displacement with annuloplasty in 32. All patients who required MV replacement or had moderate MV regurgitation had standard annuloplasty. There was no operative death. The 10-year survival was 94±3%. Four patients required MV replacement during the first 2 years of follow-up. Freedom from MV replacement was 92±3% at 10 years. Freedom from moderate or severe mitral regurgitation was 89±5% at 10 years.

CONCLUSIONS: The long-term results of patients who underwent MV repair for advanced myxomatous degeneration of the MV may be improved by correcting the posterior displacement of the mitral annulus,which is often present.

**By Invitation*

13. Curative Treatment of Atrial Fibrillation - Acute and Mid-Term Results of Intraoperative Radiofrequency Ablation of Atrial Fibrillation in 150 Patients

Friedrich W. Mohr, Nikolaus Doll*, Volkmar Falk*, Thomas Walther*, Gerd Hindricks*, Hans Kottkamp*, Ruediger Autschbach*; Leipzig, Germany

Discussant: Mehmet C. Oz

OBJECTIVE: The Maze procedure still represents the golden standard for curative treatment of atrial fibrillation and is superior to all catheter based interventions. However, intraoperative radiofrequency ablation of atrial fibrillation (IRAAF) by the induction of contiguous lesion lines avoiding multiple atriotomies may provide an alternative treatment strategy.

METHODS: We performed IRAAF restricted to the left atrium in 150 patients (pts.) with chronic atrial fibrillation. Linear and contiguous lesions were induced between the mitral anulus and the pulmonary veins to eliminate left atrial anchor" re-entry. In 42 pts. AF was the primary indication for surgery while in 108 patients intraoperative ablation was performed in conjunction with other surgical procedures (mitral valve repair or replacement in 92 pts). The procedure was performed using a less invasive surgical approach (right minithoracotomy with transthoracic aortic clamping)in 80 pts. and in 70 pts. through a median sternotomy.

RESULTS: In pts. with AF as the primary indication time for intraoperative ablation was 16±4 minutes, and clamp time was 42±18 minutes, respectively. Six months following surgery, 94% of these pts. were in stable sinus rhythm. In pts. with mitral valve disease and AF, stable sinus rhythm at 6 months following surgery was achieved in 84%. However, in pts. with giant left atria and a long history of AF (_10 yrs.), the outcome was less favourable since 42% of these pts. developed atypical atrial flutter during follow-up. One severe complication related to IRAAF was observed. In one patient perforation of the oesophagus occured presumably due to thermal damage.

CONCLUSIONS: Intraoperative radiofrequency ablation of AF can be performed with a success rate comparable to the Maze procedure. IRAAF is advantageous as the times for intervention are short and a less invasive access is possible. However, in pts. with giant left atria, additional reduction of left atrial mass may be necessary to further improve the results.

**By Invitation*

14. Routine IMA Grafting in the Elderly: An Area for Process Improvement?

T. Bruce Ferguson, Jr., Laura P. Coombs*, Eric D. Peterson*, National Database Committee; Durham, NC; Chicago, IL *Discussant: Timothy J.. Gardner*

OBJECTIVE: IMA grafting may be the single most important technique in interventional cardiovascular therapy. As CABG patients (pts) age, however, the benefits of IMA grafting become less clear since surgical risk increases and life expectancy post-CABG is less. This study examined the use, complication (Cx) risks, and operative (30-day) mortality (OM) associated with IMA grafting in pts ‰¥ 75 yrs.

METHODS: Between 1996 and 1999, 540,148 pts in the STS National Database underwent primary, non-emergent/salvage CABG; of these, 106,075 were ‰¥75 yrs of age. The influence of IMA use on OM and four major Cx was examined by 1) logistic regression (adjusting for 28

baseline risk factors) and 2) a treatment propensity score (PS) analysis (matches pts with similar baseline likelihood for IMA graft).

RESULTS: 90% of pts age 20-55 but only 70% age 75-84 received an IMA graft. IMA use was strongly associated with decreased OM [baseline risk adjusted OR=.82; 95% CI (.775, .875)] IMA use was also associated with lower or equal risk of four major Cx. As seen in the TABLE, all pt subgroups with similar PS for IMA grafting had a lower mortality if they received an IMA graft (average difference in mortality rate: 1.14, $p<0.001$).

CONCLUSIONS: In elderly pts, IMA use in primary, non-emergent CABG conveys an acute survival benefit, without an increase in perioperative Cx risk. This benefit, similar to that seen in younger pts, persists after adjusting for pt and provider selection factors. IMA grafting (as a process measure) appears to be underutilized in elderly CABG pts, and is a potential area for quality improvement.

Prop Score	% IMA used	OM: IMA=No	OM: IMA=Yes	Survival ^†
0.01-.51	31.5	6.79	6.41	0.38
0.51-.71	54.8	5.93	4.75	1.18
0.7-.82	71.9	5.51	4.18	1.33
0.82-.91	84.6	4.94	3.82	1.12
0.91-1.0	91.9	4.55	2.85	1.70

**By Invitation*

15. Ischemic Mitral Regurgitation; Intraventricular Papillary Muscle Imbrication Without Mitral Ring

Lorenzo Menicanti*, Allesandro Frigiola*, Gerald D. Buckberg, Carlo Santambrogio*, Marco Ranucci*, Danilo Santo*, Marisa Di Donate, Restore Group; Milan, Italy; Los Angeles, CA
Discussant: Steven F. Bolling

OBJECTIVE: In dilated hearts, intraventricular muscle widening between papillary muscle heads compounds cardiac dilatation to cause restrictive mitral regurgitation. The anterior ventriculotomy for surgical akinetic ventricular restoration allows access to internally reduce widening. Our approach shows intraventricular muscle imbrication during LV volume reduction can relieve mitral regurgitation without mitral ring replacement.

METHODS: In 34 patients with prior anterior infarction and congestive heart failure (NYHA IV/70%, 111/30%) mitral regurgitation was severe in 28 patients and moderate in six patients. LV volume was restored by direct suture (79%) and patch (21%) during cardioplegic aortic clamping (St. Thomas solution). Each patient underwent anterior restoration by either patch or direct suture. The regurgitation approach was through the intraventricular incision and included muscle imbrication between papillary muscle heads, and suture reduction of posterior annulus between the trigones; a plastic ring was not needed.

RESULTS: Restoration raised ejection fraction 14% (EF 25% to 39%),* reduced LV end systolic volume index 42% (94 to 54 ml/m^2),* LV end dias-tolic volume index 33% (137 to 91 ml/m^2),* and increased septal thickening from 5% to 35%.* Transesophageal echocardiographic evaluation of intraventricular mitral repair showed absent or trace MR in 92%, moderate in 5%, and significant

in one patient requiring immediate mitral valve replacement. Global mortality was 14% (5/34), with 0% in EF >25% and 23% in EF <25%. Duration of mean follow up was 29 months (1-86 months) with 12% late mortality; three cardiac deaths, one from neoplasm.

CONCLUSIONS: An innovative transventricular procedure is described during LV volume reduction. Intraventricular muscle imbrication between papillary muscle heads relieves mitral regurgitation without mitral ring requirement. *p < .05 vs pre op

**By Invitation*

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

Room 1A/B, San Diego Convention Center

Moderators: David J. Sugarbaker; Richard I. Whyte

16. Preoperative Assessment of Cardiac Ischemia in the Non-Cardiac Thoracic Surgical Patient: The Value of Thallium Scintigraphy

WITHDRAWN

17. Impact of Comorbidity on Survival after Surgical Resection in Patients with Stage I. Non-Small Cell Lung Cancer

§RichardJ. Battafarano*, Jay F. Piccirillo*, Bryan F. Meyers*, TraceyJ. Guthrie*, Joel D. Cooper, G. Alexander Patterson; St. Louis, MO

*Discussant: John R. Roberts**

OBJECTIVE: As the mean age increases in patients with non-small cell lung cancer (NSCLC), the proportion of patients with serious comorbidity considered for surgical resection is also increasing. Patients diagnosed with NSCLC have been shown to have a higher burden of comorbidity than do patients with tumors of other sites such as breast, prostate, colon, and head and neck. The goal of this research was to determine the impact of comorbidity on survival after surgical resections in patients with stage I NSCLC.

METHODS: A database analysis of our hospital's tumor registry was performed on 484 patients who underwent surgical resection for pathologic stage I NSCLC between September 1994 and December 1999. Comorbidity severity was prospectively determined using the Kaplan-Feinstein Comorbidity Index. Survival data was collected on each patient from the date of surgery with a mean duration of follow-up of 30.1 months. Bivari-ate statistics and Cox proportional hazards model analyses were used.

RESULTS: The distribution of overall comorbidty severity was None 145 (30%), Mild 144 (30%), Moderate 138 (29%), and Severe 52 (11%). The mean age was 67 years and 254 (51%) patients were male. Kaplan-Meier estimated survival at 3 years was None 82.0%, Mild 51.8%, Moderate 51.5%, and Severe 53.6%. Duration of survival was related to age ($p<0.0045$), gender ($p<0.04$), and comorbidity ($p<0.0001$). After adjusting for age and gender, the relative risk (95% CI) of mortality as a function of comorbidity was Mild 2.35 (1.39-3.96), Moderate 3.12 (1.88-5.18), and Severe 3.04 (1.64-5.61). The overall 30-day mortality rate was 2.5%(12/484). There was a non-

significant trend toward higher 30-day mortality with greater comorbidity: None 0.6% (1/146), Mild 3.4% (5/148), Moderate 2.2% (3/ 138), and Severe 5.8% (3/52) (p=0.185).

CONCLUSIONS: These results demonstrate that comorbidity has a significant impact on survival after surgical resections in patients with stage I NSCLC. These data may help to explain the lower than expected survival results in patients after surgical resection for stage I NSCLC.

[§]2001-03 AATS Research Scholar

**By Invitation*

18. An Economic Evaluation of Lung Transplantation

Ani C. Anyanwu*, Alistair McGuire*, Chris A. Rogers*, Andrew J. Murday*;
London, United Kingdom
*Discussant: Bryan F. Meyers**

OBJECTIVE: To examine the cost-effectiveness of the main forms of lung transplantation, heart-lung (HLT), bilateral (BUT) and single lung (SLT), compared to medical treatment for end-stage lung disease, in a European setting.

METHODS: Waiting list patients were used to represent medical treatment. Using parametric techniques, 4 year survival data derived from a national cohort of waiting list and transplanted patients were extrapolated to 15 years. Survival was adjusted using utility scores obtained from a cross-section of waiting list and transplant patients to allow computation of quality-adjusted life years (QALYs). Resource consumption and costs were derived from local and national sources, published data, and cost data from two centers. All costs and benefits were discounted at an annual rate of 6%.

RESULTS: Over a 15 year period, lung transplantation yielded a mean (discounted) survival gain over medical treatment of 2.0,2.4 and 2.5 years for SLT, BLT and HLT respectively. With quality of life incorporated, benefits were 2.1, 3.3 and 3.6 QALYs respectively. Over the same period, the mean cost of medical treatment was estimated at $68,000 compared to $161,000, $165,000 and $164,000 per transplanted patient for SLT, BLT and HLT respectively. Of the total transplant cost, 40% was accrued before or at time of transplant, 20% in the first 2 post transplant years and 40% in subsequent years, the cost of follow-up decreasing from $9,000 per transplanted patient in year 2 to $3,000 in year 15 (because of fewer survivors). The cost per QALY gained (over medical treatment) was $44,000 for SLT, $30,000 for BLT and $27,000 for HLT. On sensitivity analysis, the principal determinants of cost-effectiveness were quality of life and maintenance costs after transplantation.

CONCLUSIONS: Lung transplantation results in gains in survival and quality of life, but its cost-effectiveness is limited by high costs and substantial mortality and morbidity. Improved survival and quality of life after transplantation and lower immunosupression costs would improve the cost-effectiveness of lung transplantation. Transplantation of two lungs appears to be a more cost-effective option compared to single lung transplantation.

**By Invitation*

19. Molecular Assessment of Pathologic Staging in Patients with Resected Stage I. Non-Small Cell Lung Cancer- Preliminary Results of a Prospective Study

Stephen Yang, Steven Ahrendt*, David Sidransky*, Li Wu*, Carmen Roig*, Russell Pamela*, William Westra*, Jin Jen*, Malcolm Brock*, Richard Heitmiller; Baltimore, MD, Rochester, NY
*Discussant: Thomas A. D'Amico**

OBJECTIVE: Routine histologic examination of resected lymph nodes (LN) in stage I patients with non-small cell lung cancer (NSCLC) may underestimate the incidence of advanced disease. The presence of occult metastases could explain the high incidence of recurrence following curative resection. The purpose of this study was to determine the prognostic significance of p53 and K-ras mutations in histologically negative LN from patients with Stage I NSCLC using common molecular techniques.

METHOD: Since July 1995, tissue samples of primary tumor and LN were collected prospectively from all patients undergoing primary resection for NSCLC. Samples of each were submitted for pathologic (Path) and molecular (Mole) examination. Primary tumors were analyzed for p53 and/or K-ras (adenocarcinoma only) mutations by direct sequence analysis. If alterations were found, the corresponding LN were examined for these same p53 (oligonucleotide hybridization) and K-ras (allele-specific ligation) mutations.

RESULTS: p53 and K-ras mutations were found in 52% and 44% of 84 tumor samples, respectively. To date, 290 LN have been analyzed from 51 patients. In 10 patients (20%), the same p53 and/or K-ras mutations were found in both tumor and LN, suggesting occult metastasis in the LN. Based on nodal location, 8 patients were upstaged by 1 stage, and 2 by 2 stages. Disease-free survival at 3 years was significantly higher ($p<0.001$) for patients with Path and Mole negative nodes (93%)compared to those with Path negative/Mole positive and Path positive/Mole positive LN (50% and 44%, respectively).

CONCLUSION: With LN specimens that were negative by histology, 20% of patients had p53 and/or K-ras mutations and thus clinically upstaged. The molecular detection of occult metastasis in these LN had a negative impact on survival. These data suggest that molecular analysis allows a more accurate assessment of staging compared to histology, and could determine which patients would require adjuvant therapy. Larger studies are needed to determine the role of molecular staging and clinical outcomes.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:50 p.m. SIMULTANEOUS SCIENTIFIC SESSION B-2 -GENERAL THORACIC SURGERY

Room 1A/B, San Diego Convention Center

Moderators: David J. Sugarbaker; Richard I. Whyte

20. Depth of Tumor Invasion in Esophageal Carcinoma: Importance of Staging and Downstaging

Thomas W. Rice, Eugene H. Blackstone, Malcolm M. Decamp*, Sudish C. Murthy*, Gregory Zuccaro*, John J. Vargo*, Lisa A. Rybicki*, David J. Adelstein*; Cleveland, OH

Discussant: Joshua Sonett

OBJECTIVE: To evaluate the accuracy of clinical T-staging and the efficacy and role of downstaging T by induction therapy.

METHODS: The accuracy of clinical T-staging by endoscopic ultrasound (BUS) was assessed in 193 patients undergoing surgery alone for esoph-ageal carcinoma. The efficacy of downstaging T was assessed in 100 patients receiving induction chemoradiotherapy and surgery (IT).

RESULTS: Comparison of clinical (cT) and pathologic (pT) T-stage (table) showed that clinical staging was 81 %-86% accurate, 83%-99% specific, and 79%-96% negatively predictive. However, it lacked sensitivity for tumors limited to the esophageal wall (pT2 or less) and was a poor predictor of pT2. Following IT, survival was poor for patients with >cT2 carcinomas that were not downstaged (n=33, 5-year survival 13±6%), understaged or progressed (n=8, 5-year survival 12±12%) or cT2 or less (n=18, 5-year survival 11±9%). Following IT, survival was better for patients downstaged from >cT2 to pT2 or less (n=41, 5-year survival 46±8%) (P =.015); however, it did not equal that of patients with pT2 or less treated with surgery alone (n=86, 5-year survival 66+6%) (P =.006).

CONCLUSIONS: 1) If EUS identifies tumors invading beyond the esophageal wall (>cT2), IT should be given, because EUS is accurate and patients who downstage to pT2 or less will benefit. 2) If EUS does not identify tumors invading beyond the esophageal wall, surgery alone should be used, because EUS lacks sensitivity and predictive power for tumors confined to the esophageal wall (pT2 or less) and IT may adversely affect survival in these patients.

	cT				
pT	**<t2< span=""></t2<>**	**T2**	**>T2**	**Sensitivity**	**PPV**
<t2< span=""></t2<>	39	21	11	55%	98%
T2	0	9	6	60%	23%
>T2	1	9	97	91%	85%

**By Invitation*

21. Phase II Trial of Preoperative Combined Modality Therapy for Localized Esophageal Carcinoma: Cisplatinum-Taxol Followed by Radiation Therapy and Concurrent Cisplatinum-Taxol.

Manjit S. Bains, Alexander Stojadinovic*, David Ilson*, Bruce Minsky*, Alan Turnbull*, Robert Korst*, Robert Ginsberg, David Kelsen*; New York, NY
Discussant: Richard F. Heitmiller

OBJECTIVE: Our aim was to evaluate treatment response to a novel combined modality treatment regimen for locally advanced esophageal carcinoma.

METHODS: Localized EC was confirmed with endoscopic ultrasound, computed tomography, and positron emission tomography prior to induction cisplatinum-taxol (C-75mg/m2;T-175mg/m2,

2 cycles) weeks 1 & 4, combined C(30mg/m2/week)-T(60mg/m2/week-96 hour infusion) with concurrent radiation(multi-field, external beam, 1.8 Gy/d, total 50.4 Gy) weeks 7-12 and esophagectomy week 16 after re-staging confirmed resectability.
RESULTS: Forty-two patients have been enrolled (37 male), with adenocarcinoma (n=25) and squamous cell cancer(n=17), stage T1-2N0-1 (n=7), T3N0-1 (n=34). Of 38 with pre-treatment dysphagia, symptoms resolved/improved in 35(92%). Nineteen (45%) were hospitalized (median, 6 days) for Grade III/IV chemoradiation-related toxicity. Only 2 patients required enteral feeding tube support during therapy. Major (eg anasto-motic leak, respiratory failure) post-operative morbidity occurred in 12/ 36(33%) patients. Thirty-six patients have completed treatment and are evaluable for response. There were two perioperative deaths. Of 34 R0 resections, 12(35%) had complete (pCR) pathologic, 19(56%) were down staged, 3(6%) had no response, and 1(3%) had disease progression. At a median follow-up of 17 months, 15/36(42%) are alive disease-free, 11/36(31%) alive with disease, and 8/36(22%) died of disease. Median progression-free survival is 18 months. One and 2-year disease-specific survivals are 82% and 62%.

CONCLUSIONS: This multimodality regimen of combined chemotherapy with concurrent radiation followed by surgery for locally advanced esophageal carcinoma produces significant treatment response, dysphagia relief with induction chemotherapy, and is associated with encouraging survival.

**By Invitation*

22. Marginal Donor Lungs: A. Re-Assessment

Andrew F. Pierre*, Yasuo Sekine*, Michael A. Hutcheon*, §Thomas K. Waddell*, Shaf H. Keshavjee; Toronto, ON, Canada *Discussant: Michael S. Mulligan**

OBJECTIVE: Lung transplantation is limited by the shortage of suitable donors. To overcome this problem many programs have begun to use marginal donors after reports suggesting equivalent outcomes with no additional risk. As our utilization of marginal donor lungs increased we felt it appropriate to evaluate outcomes with these organs compared to standard donor lungs.
METHODS: We performed a retrospective review at our institution of 128 consecutive lung or heart-lung transplants from January 1, 1997 to June 30, 2000. The primary end-point was 30 day mortality. Donors were considered standard if they met previously accepted criteria for lung procurement. Donors were considered marginal if any one of the following criteria were met: age>55 years, smoking>20 pack-years, CXR infiltrate, PO2<300mmHg, or purulent secretions on bronchoscopy.

RESULTS: We had a total of 123 donors, 63 were marginal for a marginal donor rate of 51%. Forty-eight donors failed 1 criteria, 10 failed 2 criteria, and 5 failed 3 criteria. Forty-one donors failed the CXR criteria, 26 failed smoking, 9 failed age, 8 failed bronchoscopy, and 0 failed PO2. One hundred twenty eight transplants were performed. The 30 day mortality for the standard donor group was 4/65 or 6.2% vs. 11 /63 or 17.5%, *p=0.047 for the marginal donor group.

CONCLUSIONS: While many marginal donors will result in acceptable post operative function, caution needs to be exercised in the utilization of marginal donor lungs since there seems to be an increased early mortality rate in that group of recipients.

§2000-02 AATS Research Scholar

**By Invitation*

23. Bronchial Carcinoid Tumours: Surgical Management and Long-Term Outcome.

Pierluigi Filosso*, Ottavio Rena*, Giovanni Donati*, Caterina Casadio*, Giuliano Maggi*; Turin, Italy
Discussant: Mark S. Allen

OBJECTIVE: We sought to determine the variables influencing long-term survival of patients treated for bronchial carcinoids.

METHODS: A retrospective, mono-istitutional review of patients submitted to surgical treatment since 1977 is carried out.

RESULTS: Over 22 years, 126 patients with final histological diagnosis of bronchial carcinoid tumours were assessed for surgery. There were 72 men (57%) and 54 women (43%) with mean age at presentation of 47±16(range 11-77) years. Symptoms were present in 65 (53%) patients. Operations included lobectomy or bilobectomy in 88 (with 4 bronchoplastic procedures), pneumonectomy in 15, segmentectomy in 3, wedge-resection in 16 and bronchial sleeve resection in 3 patients; one patient underwent explorative thoracotomy. One patient died in the perioperative period (0.7%). 82 patients (65%) had typical and 44 (35%) had atypical carcinoid tumuors. Postoperative staging was complete for 117 out of 126 patients (9 patients did'n undergo lymphadenectomy): stages were 1,94; II, 6; III, 15 and IV, 2. Typical subtype was stage I in 74 and more advanced (II-IV) in 5, whereas atypical one was stage I in 20 and more advanced in 18 ($p<0.05$). Mean follow up was 99±73 months (range 1-276) during which 19 (15%) patients died (12 from recurrent disease). Recurrent tumour developed in 4/ 82 (4.8%) patients with typical and 8/44 (18.2%) with atypical subtypes. Overall survival at 15-years was 74%; 15-years histological type, nodal status and stage related survival are significant($p<0.05$).

CONCLUSIONS: Bronchial carcinoid tumours have best biological behaviour and prognosis than other lung cancers. Surgical treatment requires radical excision and lymphonodes sampling. Survival and long-term out come are significantly related to the histological type, nodal status and pathology stage.

**By Invitation*

24. Results of En Bloc Resection for Bronchogenic Carcinoma with Chest Wall Invasion

Harold M. Burkhart*, Mark S. Allen, Francis C. Nichols*, Claude Deschamps, Daniel L. Miller*, Victor F. Trastek, Peter C. Pairolero; Rochester, MN; Scottsdale, AZ
*Discussant: Eric Vallieres**

OBJECTIVE: Lung cancer invading the chest wall has recently been re-staged. To determine the affect of this change on survival in our clinical experience, we reviewed patients treated with en bloc resection for bronchogenic carcinoma with chest wall invasion.

METHODS: From February 1985 to November 1999, 100 en bloc resections were performed on 99 patients (67 men, 33 women). Median age was 66 years (range 38-93). Pancoast tumors were excluded. Factors that affected survival were analyzed using univariate and multivariate analysis.

RESULTS: Initial symptoms included chest wall pain in 43 patients, cough in 18, and other in 18. Twenty-one patients were asymptomatic. Ninety-six patients were current or former smokers (median 50 pack-years, range 8-150). Operations included 77 lobectomies, 14 pneumonectomies, 6 bilobectomies, 2 wedges, and 1 segmentectomy. The number of ribs resected ranged from 1 to 5 (median 3). Sixty-six patients required chest wall reconstruction (prostheses in 63, autologous tissue in 2, and bovine patch in 1). Operative morbidity and mortality were 54% and 6%, respectively. The postsurgical stage was T3N0M0 in 64 patients, T3N1M0 in 16, T3N2M0 in 15, and other in 5. Squamous cell carcinoma was present in 59 patients, adenocarcinoma in 26, large cell carcinoma in 12, and other in 3. Follow-up was complete in 97 patients. Median follow-up in the 94 operative survivors was 19 months (range, 1 month to 15 years). Overall five-year actuarial survival was 36%; stage IIB (T3N0M0) patients had significantly better survival than stage IIIA (T3N1MO, T3N2M0) patients (43% vs 25%, p=0.0076). Women had a better 5-year survival than men (51% vs 27%, p=0.0059). The best 5-year survival was observed in women with stage IIB (T3N0M0) disease (61%). All other factors including age, tumor size, histopathology, FEV1, extent of operation, and radiation therapy did not significantly affect survival.

CONCLUSIONS: Survival of patients with bronchogenic carcinoma with chest wall invasion is stage and gender dependent. The best survival is observed in women who have no nodal involvement.

**By Invitation*

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

Room 6D/E, San Diego Convention Center

Moderators: Thomas L. Spray; Michael D. Black

25. Impediments to Anatomic Repair in Patients with Discordant Atrioventricular Connections and the Obstructed Pulmonary Pathway

Hideki Uemura*, Toshikatsu Yagihara*, Soichiro Kitamura, Youichi Kawahira*, Yoshiro Yoshikawa*; Osaka, Japan
Discussant: Constantine Mavroudis

OBJECTIVE: To determine whether anatomic repair is always ideal in patients with discordant atrioventricular connections and pulmonary stenosis or atresia.

METHODS: Since 1988, 36 patients with this malformation have undergone biventricular repair. Of these, anatomic repair was employed in 26 by intraatrial and intraventricular rerouting combined with RV outflow tract reconstruction, and functional repair in the other 10.

RESULTS: Anatomic repair was not considered feasible in those undergoing functional repair, because of VSD distantly oriented from the aortic orifice (in 5) or restrictedly small for intraventricular rerouting (in 2), small EDV of the morphologically LV (in 5), and morphologically mitral regur-gitation (in 3). Pulmonary atresia was less frequent in this group (p=0.01). Survival rate was 89, 84, and 67% at 1, 5, and 10 years after anatomic repair, respectively, while 90, 79, and 63% after functional repair. Risk factors of early death after anatomic repair (in 3) included long aortic cross-clamping (p=0.01) or cardiopulmonary bypass (p=0.05), abnormal PA arbolization (p=0.009), abundant systemic-to-pulmonary collaterals (p=0.001), and perimembranous inlet VSD (p=0.001). In the longer terms after anatomic repair, LV performance was impaired in 6, related to enlargement of VSD (p=0.01), 10 years or older of age at operation (p=0.01), and abundant

systemic-to-pulmonary collaterals (p=0.004). Atrial arrhythmia was commoner in the group of patients having their ventricular function impaired than in those with excelent cardiac performance (p=0.002). Two with LV dysfunction died suddenly because of atrial arrhythmia 30 and 106 months after anatomic repair. Freedom from rhythm disturbance was 92, 82, and 61 % at 1, 5, and 10 years after anatomic repair, respectively, while 80, 67, and 67 % after functional repair.

CONCLUSIONS: Functional results are not always ideal after anatomic repair in patients with this particular feature of cardiac malformation. Taking morphologic, operative, and functional impediments into account, indication for anatomic repair should be appropriately determined.

**By Invitation*

26. Early Surgical Intervention Improves Outcome in Patient of DORV with Heterotaxy and/or Complete AV Canal Defect.

Koh Takeuchi*, Francis X. McGowan*, John E. Mayer, David Zurakowski*, Emile A. Bacha, Richard A. Jonas, Pedro J. Del Nido*; Boston, MA, Chicago, IL

Discussant: John J. Lamberti

OBJECTIVE: Double outlet right ventricle (DORV) encompasses a broad spectrum of anomalies. Heterotaxy syndrome, which is often associated with total anomalous pulmonary venous connection(TAPVC), and complete AV canal defect (CAVC) has been considered a risk factor for surgical repair of DORV.

METHODS: From January 1992 to May 1999, 96 patients who had DORV with heterotaxy and/or CAVC were retrospectively reviewed (Median age=31 days at initial surgery).17 patients were neonates and symptomatic.

RESULTS: 66/96 patients had heterotaxy(30/66 with TAPVC). 83/96 had CAVC(63/83 with ‰¥ mild AV valve regurgitation(AWR). 10 patients received 2 ventricle repair, 83 patients were considered for 1 ventricle repair (procedures to date: bidirectional Glenn (BDG):21, Fontan:52, primary palliation (PAB,BT shunt, or Norwood I):9 and 3 other patients required surgery for AWR or TAPVC alone. One patient had heart transplantation after BDG. There were 16 deaths including 10 early (<30 days POD). Median follow-up was 16 months (1m-7.4years). Actuarial Kaplan-Meier survival (95% CD was 89%(83-96%) at 1 month; 84%(76-91%) at 1 year; 81%(73-89%) at 5 years. Symptomatic neonatal presentation (p<0.001) and AVVR (p=0.04) were risk factors. However, symptomatic neonates who then underwent palliation had 9 times lower risk than those medically managed. The instantaneous monthly risk of death was 9 times greater for non-palliated symptomatic neonates compared to those receiving palliation (hazard ratio=9.2, 95% confidence limits=2.1-40.0, Cox proportional-harzards model p<0.0001). Other variables including heterotaxy, arch hypoplasia, pulmonary stenosis/atresia, and TAPVC(+/-obstruction) were not predictive of survival by multivariable analysis.

CONCLUSIONS: AV valve regurgitation and neonatal presentation, but not heterotaxy or TAPVC are independent risk factors for mortality in DORV with heterotaxy and/or CAVC patients. Early surgical intervention for symptomatic neonates can significantly reduce mortality and improve outcome following staged operation.

**By Invitation*

27. Hemi-diaphragmatic Paralysis Increases Postoperative Morbidity Following Modified Fontan Procedure.

Zahid Amin*, Doff B. McElhinney*, Jennifer Strawn*, John D. Kugler*, Vadiyala M. Reddy, Kim F. Duncan*, Edwin Petrossian*, Frank L. Hanley; Omaha, NE; Philadelphia, PA; San Francisco, CA
Discussant: Marshall L Jacobs

OBJECTIVE: After a modified Fontan procedure, forward pulmonary blood flow is augmented in inspiration due to negative intrathoracic pressure. Total pulmonary blood flow is 63% higher during inspiratory cycle than during expiratory cycle. With hemi-diaphragmatic paralysis, inspiratory augmentation of pulmonary flow is lost. The objective of this study was to compare early postoperative morbidity following modified Fontan operation in patients (pts) with and without hemi-diaphragmatic paralysis.

METHODS: A case-control analysis was performed comparing 9 pts with documented hemi-diaphragmatic paralysis against 28 pts without hemi-diaphragmatic paralysis who were matched for diagnosis, age, and fenes-tration. Preoperative variables analyzed included age, weight, pulmonary artery pressure (PAP), atrioventricular (AV) and semilunar (SL) valve regurgitaqtion, arrhythmias and ventricular hypertrophy The following early postoperative outcomes were assessed: duration of ventilator support, duration of hospital stay, incidence of ascites, prolonged effusions (chest tube >14 days), and readmission.

RESULTS: Preoperatively, there were no significant differences between the 2 groups in age (8.0+/-6.4 vs 4.1+/-3.9 years, p=0.12), weight (19.7+/ -8.1 vs 15.2+/-9.4 kg, p=0.21), PAP (12.8+/-4.8 vs 11.4+/-1.7 mm Hg p=0.43), AV valve regurgitation (0% vs 32% p=0.06), SL valve regurgitation (0% vs 11% p=0.42), arrthythmias (0% vs 3.6% p=0.76) or ventricular hypertrophy (11% vs 11% p=0.65). However, the duration of hospital stay (25.1+/-17.7 vs 11.1+/-6.3 days, p=0.01), incidence of ascites (77% vs 3.6%, p<0.001), prolonged pleural effusions (66% vs 14%, p=0.01), and readmission (44% vs 7%, p=0.02) were significantly greater in pts with hemi-diaphragmatic paralysis than those without.

CONCLUSIONS: Hemi-diaphragmatic paralysis following modified Fontan operation is associated with an increase in early morbidity. Utmost care should be taken intraoperatively to avoid injuring the phrenic nerve. All patients with prolonged effusions should be evaluated for hemi-diaphragmatic paralysis by ultrasonography or fluoroscopy.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION C-2 - CONGENITAL HEART DISEASE

Room 6D/E, San Diego Convention Center

Moderators: Thomas L. Spray; Michael D. Black

28. Ross Procedure is the Procedure of Choice for Congenital Aortic Valve Disease

Zohair Al Halees, Frans Pieters*, Fatima Qadoura*, Male Shahid*, Fadel Al Fadley*; Riyadh, Saudi Arabia
Discussant: Ronald C. Elkins

OBJECTIVE: The Ross Procedure has emerged as an attractive option for aortic valve replacement particularly in children and young adults. Our objective is to review our experience with the Ross Procedure in patients with congenital aortic valve disease and look for evidence of growth of the autograft.

METHODS: From Jan 90 to July 2000, 260 patients (pts) underwent the Ross Procedure. There were 136 pts below 18 years of age. Fifty-three children (38%) had congenital aortic valve disease. This constitutes the study group. Age ranged from 3 months to 18 years, mean 12 years ±5 (median 9 years -10 pts were infants). Pure aortic stenosis was present in 18 pts, stenosis and regurgitation in 32, and pure aortic regurgitation in 3. Twenty nine pts had previous procedures (8 balloon dilatations, 12 surgical valvotomies and 9 other procedures). Aortic valve was bicuspid in 29 pts.

RESULTS: In all pts immediate results demonstrated all levels of obstruction were relieved. The gradient across the left ventricular outflow tract(LVOT) was completely abolished with not more than trivial aortic regurgitation. Hospital mortality was 3/53 (5.6%) (overall Ross mortality 4/260 (1.5%). Mean follow up was 4 years (max 10 years). One pt died late of non cardiac cause. Actuarial survival at 10 years was 94±2 and freedom from all events was 93+5. Only one pt needed autograft replacement for endocarditis. Intervention related to right ventricle to pulmonary artery conduit was required in 3 pts, balloon dilatation with "moderate" success in 2 and one reoperation. At last follow up all pts but one were in NYHA Class I or II with normal or near normal autograft function. Serial measurements of the LVOT and aortic root showed that as pts grow the size of the LVOT increases. When indexed to body surface area this increase correlated with pts expected somatic growth.

CONCLUSIONS: The Ross Procedure for congenital aortic valve disease in children and young adults offers excellent hemodynamics with the added advantage of real potential for growth. It should be considered the treatment of choice in this age group.

**By Invitation*

29. Repair of Recurrent Left Atrio-Ventricular Valve Regurgitation: Construction of a Coaptation Surface.

Christian P. Brizard*, Sunil K. Kaushal*, Tom R. Karl, Andrew D. Cochrane*; Melbourne, Australia; Philadelphia, PA
Discussant: Pedro J. del Nido

OBJECTIVE: Evaluation of a new technique of repair of recurrent severe left atrio-ventricular valve regurgitation (R) after correction of atrio-ven-tricular septal defect (AVSD). Conventional repairs do not address directly the mechanism of the regurgitation and often have poor results. We have used a technique in which a coaptation surface was constructed and suspended to the papillary muscles. A patch was sutured at the edge of the closed cleft, facing the left lateral leaflet when the latter was well developed. Alternatively, a patch was sutured on each side of the open cleft when the left lateral leaflet was absent or hypoplastic. We have mostly used treated autologous pericardium and less frequently a partial mitral valve homograft (PMVH) when the anatomy was severely distorted.

METHODS: Retrospective study of 17 consecutive patients operated on between March 1998 and October 2000.

RESULTS: Pre-operative R was greater than moderate n=7 or severe n=10. The median age was 95 months (17 to 274). Median number of previous procedures was 2 (1 to 3). Ten patients had

complete AVSD and 7 were partial. The median delay between this operation and the initial correction was 89 months (7-247). In five patients we used PMVH and autologous pericardium in 12. In 5 patients, there was one or more associated intra-cardiac procedure. There was one hospital death. A 22-year-old man died of refractory hyperpyrexia. Two patients required an intra-operative revision of the repair. At median 6 months follow-up (0 to 29), 16 patients are NYHA I. Echo results are stable in 14 patients with trivial/ mild R (n=12) and moderate R (n=2), or show degradation of the result in two (1 mild to moderate R and 1 severe R leading to valve replacement in one PMVH recipient). There is no diastolic gradient greater than 1.7 m/s.

CONCLUSIONS: This series demonstrates the feasibility of the repair with good initial result. It is possible to limit the indication for PMVH. At midterm follow-up, there is stability of the result in the majority of patients, whether a PMVH was used or not. Longer follow-up should support repair versus replacement strategy in this specific group of patients.

**By Invitation*

30. Homograft Conduit Failure in Infants is Not Due to Somatic Outgrowth

Winfield J. Wells, Hector Arroyo*, Ross M. Bremner*, John Wood*, Vaughn A. Starnes; Los Angeles, CA

Discussant: John W. Brown

OBJECTIVE: It has been assumed that the need for homograft replacement is due to somatic outgrowth, but this has not been adequately studied. Our objective was to identify reasons for homograft conduit failure.

METHODS: The records and imaging studies of 40 pts undergoing RVOT homograft conduit replacement from 1995 to 2000 were retrospectively reviewed.

RESULTS: The majority of pts carried a diagnosis of Tetralogy of Fallot (n=19) and Truncus Arteriosus (n=13). The median age at the initial surgery was 7 months (0.25-108). The initial homograft sizes ranged from 9-21mm, and 28 conduits were of pulmonary origin. When comparing size of the initial homograft to pts expected pulmonary valve diameter(Z=0), oversizing was note to be Z=+3.6 (+1-+13). Median interval to conduit failure was 5.16 years (0.5-11.3). At homograft replacement only 12 pts had an existing conduit that was one standard deviation below the homograft conduit size needed (Z

CONCLUSIONS: Somatic outgrowth is seldom a primary reason for RVOT homograft conduit replacement. The most common etiology for failure is conduit obstruction with thickening and shrinkage at the annular area. Conduit stenosis was responsible for failure in 53% of pts, technical issues were responsible for 39%, and only 8% failed as a result of somatic outgrowth. Placement of a smaller homograft (Z=0) at the initial operation may decrease the incidence of conduit kinking, sternal compression, and posterior shelf impingement.

**By Invitation*

31. Neuroprotective Effects of Pretreatment with Steroids in a Neonatal Piglet Model of Cardiopulmonary Bypass with Deep Hypothermic Circulatory Arrest.

Hashim Abdul-Khaliq*, Stephan Schubert*, Gisella Stoltenburg-Didinger*, Dirk Triotzsch*, Anke Wehsack*, Wolfgang Boettcher*, Eckard Gutsch*, Michael Huebler*, Vlademir Alexi Meskishvili*, Roland Hetzer, Peter E. Lange*; Berlin, Germany
Discussant: Ross M. Ungerkider

OBJECTIVE: We evaluated the mode of neuronal cell injury in neonatal piglets and the possible neuroprotective effects of pretreatment with high dose steroids.

METHODS: Neonatal piglets (age < 10 days, 2.1 +/- 0.5 kg BW) were included in this study. Ten animals were pretreated with high dose methylprednisolone (MP) (30mg/kg/BW) and underwent DHCA for 120 min. MP was administered either systemically 24 and five hours preoperatively (n=5), and intrathecally five hours preoperatively (n=5). Ten animals without pharmacological intervention served as control group. After median sternotomy the animals were connected to CPB by cannulation of the aorta and right atrium. Full flow CPB (200ml/kg/min) was initiated for homogeneous systemic cooling. DHCA for 120 min. was induced at a temperature of 14 °C. After rewarmed reperfusion the animals were weaned from CPB and monitored for 6-8 hours. Then the animals were sacrificed and the brain was immediately removed and fixated for further histological studies. Neuronal cells were counted in sector CA1-CA4 and dentate gyrus of hippocampus formation, basal ganglia, cortex and cerebellum in respect to apoptosis and necrosis.

RESULTS: The main preliminary findings in this brain ischemic model were the quantitative evaluation of necrotic and apoptotic neuronal cell injury according to the mode of steroid application. Significant reduction of necrotic neuronal cell changes in sector CA1-CA4 of hippocampus was found only in the group with intrathecal steroid application. For the dentate gyrus the mode of neuronal cell injury changed from necrosis to apoptosis for both groups, however with less apoptotic cell changes in the intrathecally treated group.

CONCLUSIONS: Due to the non-passage of steroids through the blood brain barrier, intrathecal rather then systemic application of steroids seems to have protective effect after DHCA. The pronounced apoptotic neuronal cell injury, of the steroid pre-treated animals, raises concern with regard to the routine systemic application of steroids during paediatric cardiac surgery.

**By Invitation*

32. Surgically Treated Bacterial Endocarditis in Children: Valve Replacement or Repair?

Andreas G. Sakopoulos*, David M. Overman*, Glen S. Van Arsdell*, Goran Delgren*, Bagwan Koirala*, John G. Coles, William G. Williams; Toronto, Ontario, Canada
Discussant: David R. Clarke

OBJECTIVE: To evaluate the results of valve replacement or repair in the treatment of endocarditis in children.

METHODS: This is a retrospective review of 23 consecutive children who underwent surgery for bacterial endocarditis from January 1978 to January 2000. Median age was 8 years and ranged from 3 months to 18 years. Staphylococcus species were responsible for 2/3 of cases. Surgery was

performed on an emergent basis in 83% of patients (19/23) for either mechanical or persistant infectious indications.

RESULTS: The repairs performed were classified as: simple (vegetation excision) in 5 (22%); intermediate (vegetation excision with part of leaflet) in 7 (30%); complex (debridement, leaflet or annular reconstruction) in 8 (35%). Valve replacement following wide resection was necessary in only 3 patients (13%). Fifteen patients (65%) had concomitant congenital cardiac anomaly corrections. Repair was performed in 20 patients. In 3 no attempt at repair was made because of extensive disease. One of the patients initially repaired required valve replacement 4 days later, for severe valvular insufficiency. Valvular repair was therefore successful in 95% (19/20) of patients in which it was attempted. Early mortality was 13% (3/23), with all deaths occuring in the valve replacement patients (3/4). No patients developed recurrent endocarditis. Intermediate term follow-up was available in 85% of patients (17/20) and ranged from 3 months to 22 years, with a mean of 8 years. Of the surviving patients, two required delayed cardiac reintervention: one had valvular re-repair 10 years following the original operation and the other underwent valve replacement 1 year following tricuspid valvectomy. The other surviving patients were all asymptomatic, on no medications, and in NYHA class I. Latest echocardiograms evaluating valvular regurgitation revealed: none or trivial in 4, mild in 9, and moderate in 2 patients.

CONCLUSIONS: Valve repair was achieved in most patients surgically treated for bacterial endocarditis (87%). At intermediate term follow-up 89% of survivors are free of cardiac reinterventions, 100% are free of endocarditis, and 100% are symptom free.

**By Invitation*

33. Aortic Arch Reconstruction with Pulmonary Autograft Patch Aortoplasty

Emre Belli*, Marie Lannelongue*, Regine Roussin*, Francois Lacour Gayet*, Jacqueline Bruniaux*, Claude Planche, "Alain Serraf; Le Plessis Robinson, France

Discussant: J. William Gaynor*

OBJECTIVE(s): The optimal technique for aortic arch reconstruction through median sternotomy is still under debate. We introduced the technique of pulmonary autograft patch aortoplasty as a reliable alternative.

METHODS: The outcome of 50 hospital survivors who underwent neonatal one-stage repair of interrupted aortic arch (n=29) or coarctation associated with aortic arch hypoplasia (n=21) and ventricular septal defect since 1992 was analysed. The patients were reviewed in 3 groups according to the aortic arch reconstruction technique: I-direct anastomosis (n=25), II-homograft or pericard patch aortoplasty (n=8), III-pulmonary autograft patch aortoplasty (n=17). The pulmonary autograft patch consisted in the anterior wall of the main pulmonary artery, between the supra-commis-sural level and the divided ductus arteriosus. The created defect was replaced by fresh autologus pericardium.

RESULTS: All patients were discharged without significant residual gradient at the level of aortic arch. In a median delay of 8 (2-36) months, 8 patients (16%) developped recurrent arch obstruction. Seven of them underwent balloon angioplasty (n=4) or surgery (n=3). One patient who had direct anastomosis required reoperation for bronchial compression. At a median follow-up of 39 months, freedom from recurrent arch obstruction was 88% in group I,, 38% in group II and 100% in group III (p=0.0001).

CONCLUSIONS: The aortic arch repair by means of pulmonary autograft patch aortoplasty results in superior midd-term outcome and constitutes a reliable alternative to the direct anastomosis technique. It diminishes the anastomotic tension, so that the risk of restenosis and tracheobron-chial compression. We observed a significantly higher rate of recurrence after patch aortoplasty with other materials.

ˆž1993-94 AATS Graham Fellow

**By Invitation*

TUESDAY MORNING, MAY 8, 2001

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

Room 6A-C, San Diego Convention Center *Moderators: Irving L. Kron; Richard D. Weisel*

L1. Septal-Lateral Annular Cinching Abolishes Acute Ischemic Mitral Regurgitation in Sheep

Tomasz A. Timek*, David TM Lai*, David Liang*, Frederick Tibayan*, George T. Daughters*, Paul Dagum*, Neil B. Ingels*, D. Craig Miller; Stanford, CA; Palo Alto, CA

OBJECTIVE: Ring annuloplasty prevents acute ischemic MR (IMR) in sheep but it also abolishes normal mitral annular dynamics. We investigated a novel surgical approach of simple septal-lateral annular cinching (SLAC) with sutures to treat acute IMR.

METHODS: Nine adult sheep underwent implantation of multiple ra-diopaque markers on the LV and around the mitral annulus (MA). A septal-lateral (S-L) transannular suture was anchored to the mid-anterior MA and externalized on a tourniquet through the mid-posterior MA and LV wall. Open-chest animals were studied immediately post-op. Acute IMR was induced by proximal LCx snare occlusion and three progressive steps of SLAC (each 2-3 mm suture tightening for 5 sec) were performed using the externalized transannular suture. Biplane videofluroscopy and TEE were performed continuously before and during LCx ischemia and SLAC. MA area (MAA) and S-L diameter were calculated from the annular markers at end-diastole (ED). MAA contraction (MAA_{cont}) was expressed as percent decrease from max to min MAA. MR was graded +0 to +4.

RESULTS: Acute ischemia decreased LV dP/dt (23851352 vs 1265±300 mmHg/s)and LVPmax (110±9 vs 83±12 mmHg), while EDV (146+25 vs 177±30 ml) and MR (+0.5±0.4 vs +2.0±0.7)increased (all p£.01). Hemody-namic variables did not change further during progressive SLAC. Table-*p<.05 vs ischemia.

CONCLUSIONS: Isolated 22±10% reduction in MA S-L dimension abolished acute IMR in normal sheep while only modestly affecting MAA contraction dynamics. SLAC may represent a quick method for the surgical treatment of IMR, either as an adjunctive technique or alone.

	Ischemia	SLAC-1	SLAC-2	SLAC-3	ANOVA
MR	+2.0±0.7	+1.8±0.7	+1.0±0.4*	+0.6±0.5*	.0005
S-L_{ED} (mm)	27.7±2.7	26.0±3.4	24.5±3.0	21.7±3.9	.0005

MAA_{ED} (cm^2)	8.56±0.97	8.23±1.10	7.98±1.03	7.37±1.18	.0005
MAA_{cont} (%)	8.5±1.0	8.0±1.0	7.4±0.9*	.6.9±1.9*	.005

**By Invitation*

L2. Contrasting Nature of Lung Growth Following Transplantation and Lobectomy

Aditya K. Kaza*, Victor E. Laubach*, Jeffrey T. Cope*, James J. Gangemi*, Stewart M. Long*, Steven M. Fiser*, Scott D. Ross*, David C. Cassada*, John A. Kern*, Curtis G. Tribble, Irving L. Kron; Charlottesville, VA

OBJECTIVE: We hypothesized that post-transplant lung growth and post-lobectomy compensatory lung growth (CLG) are two distinct processes.

METHODS: Mature MHC defined miniswine underwent left upper lobec-tomy, and the growth of the left lower lobe (LLL) was studied at 2 weeks (2wk Lob) and 3 months (3mo Lob). LLL from another set of mature pigs were transplanted into immature animals after left pneumonectomy; the growth of the transplanted lobe was studied at 2 weeks (2wk Tx) and 3 months (3mo Tx). LLL from unoperated mature animals were used as controls (NORM). LLL weight at time of harvest was recorded and the lobes fixed intrabronchially. H&E-stained sections were used for morphometric analysis to determine alveolar surface density (ASD) and relative volume of respiratory region (Vvr, % of total lung volume). Immunostaining for 5-bromodeoxy-uridine was used to determine cellular proliferative index (CPI). Epidermal growth factor receptor expression (EGFR) was detected using Western blot.

RESULTS: Post-lobectomy CLG reaches a peak at 2 weeks with a concomitant peak in CPI. The transplanted lobe, however, exhibits a gradual growth response up until 3 months, even at which time it is not as prolific as CLG. There was no difference in ASD between the various groups. The Vvr was noted to increase only in the transplanted lobe at 3 months. The results are indicated in the table (mean ± sem). EGFR expression was upregulated in the lobectomy group at 2 weeks and transplant group at 3 months when compared with controls.

CONCLUSIONS: Post-lobectomy CLG appears to occur by a different mechanism when compared with transplant lung growth. CLG is a much rapid and restorative process when compared with transplant lung growth. The growth peaks in both processes correlates with EGFR upregulation.

	NORM (n=5)	2wk Tx (n=5)	2wk Lob (n=5)	3mo Tx (n=5)	3mo Lob (n=5)	ANOVA
LLL Weight	93±5	115±7	197±10*	138±7*	181±20*	*p<0.05 vs NORM
CPI	3.9±0.3	4.6±0.2	7.2±0.6*	7.6±0.8*	5.3±0.3	*p<0.001 vs all
Vvr	82±2.4	88±0.8	86±1.0	96±1.3*	86±1.2	*p<0.001 vs all

**By Invitation*

L3. Molecular Staging Of Lung Cancer: Quantitation of Micrometastatic Tumor Burden by Quantitative Polymerase Chain Reaction

Jonathan D'Cunha*, Angela L. Corfits*, Robert A. Kratzke*, Michael A. Maddaus; Minneapolis, MN

OBJECTIVE: Stage I NSCLC survival is 60-70% due to undetected systemic occult micrometastases (OM). OM detection by conventional PCR only provides yes/no answers about OM presence. Quantitative PCR (QPCR) permits reproducible quantitation of target molecules. This study evaluates QPCR's ability to quantitate OM burden in lymph nodes (LN).

METHODS: Standard PCR and QPCR for CEA mRNA were performed on 85 lymph nodes (18 stage I patients, LN negative by histology). QPCR determines CEA mRNA quantity by detecting fluorescence increase at a threshold cycle (C_T). Lower C_T values correspond to higher CEA mRNA concentrations. Standard curves were created with dilutions of CEA positive MCF-7 tumor cells.

RESULTS: Detection rates of OM and upstaging of patients were similar for conventional PCR and QPCR: 20/85 (24%) vs. 16/85 (19%) and 12/18 (67%) vs. 9/18 (50%) respectively. QPCR positive LN (C_T<45) were compared to a standard curve allowing estimation of number of micrometastatic cells per LN (Table 1).

CONCLUSIONS: 1) Conventional and QPCR detect LN OM at similar rates. 2) Both upstage [a]50% of stage I patients. 3) QPCR estimates the number of micrometastatic cells per LN, allowing greater precision in assessing recurrence risk.

Table 1. QPCR Estimation of Micrometastic Tumor Cell Burden

C_T Value	Number of LN Stations Positive (N=16)	Estimated Number of Micrometastic Cells Per LN
26	1	73000
32	3	14000
33	5	11000
34	1	5800
36	1	5000
37	3	3500
39	2	2000

**By Invitation*

L4. Diastolic Counterpulsation: Circulatory Benefits in an Ischemic Heart Failure Model One Year Following Aortomyoplasty

Nasira Hedayati, John T. Sherwood*, Steve J. Schomisch*, Joseph L. Carino*, Brian L. Cmolik*; Cleveland, OH

OBJECTIVE: Aortomyoplasty (AMP) is a form of skeletal muscle cardiac assist that provides diastolic Counterpulsation. We hypothesized that AMP can generate hemodynamic augmentation comparable to the intraaortic balloon pump (IABP) in a model of chronic ischemic heart failure.

METHODS: Six mongrel dogs (25-30 kg) underwent AMP. Following chronic conditioning of the latissimus dorsi muscle, multiple coronary microembolizations to induce ischemic cardiac dysfunction were performed. Ejection fraction decreased from 62% to 35%. Studies were conducted one year after AMP (4 months after the final microembolization). Endcardial viability

ratio (EVR), mean diastolic aortic pressure (mDAP), and peak left ventricular pressure (pLVP) were measured during one hour of AMP Counterpulsation and one hour of IABP.

RESULTS: The EVR, mDAP, and pLVP obtained during acute AMP coun-terpulsation and IABP showed significant differences between augmented and nonaugmented beats (Table 1). AMP provided similar improvements compared to IABP.

CONCLUSIONS: One year following surgery, acute AMP counterpulsa-tion, in a model of chronic ischemic heart failure, provided hemodynamic augmentation comparable to IABP. By increasing mDAP and decreasing pLVP, AMP can improve myocardial performance. The data support the long-term viability and efficacy of AMP. AMP Counterpulsation has the ability to generate the same cardiac benefits that the IABP provides for patients with ischemic cardiac dysfunction, with the advantages of chronic application.

Table 1. AMP and IABP: Effects of EVR, mDAP, and pLVP.

	AMP/Start	AMP/End	IABP/Start	IABP/End
EVR	23.8±7.9*	19.9±7.7*	22.7±12.9*	21.0±11.7*
mDAP	15.9±3.9*	12.1±2.7*	17.4±9.5	16.8±8.9*
pLVP	-2.4±0.4*	-2.6±1.6*	-2.2±1.3	-2.0±0.9*

Mean % change, *p<0.05 vs non-augmented beat, start and end of 1 hour counterpulsation

**By Invitation*

L5. Endothelin-1 Mediates Perioperative Ischemia-Reperfusion Injury in Diabetes

Subodh Verma*, Aaron S. Dumont*, Paul M. Fedak*, Gideon Cohen*, Michael A. Borger*, Renke Li*, Richard D. Weisel, Andrew Maitland*, Todd J. Anderson*; Toronto, ON, Canada; Charlottesville, VA; Calgary, AB, Canada

OBJECTIVE: Diabetic patients are at increased risk of ischemic events & mount an inordinate response to perioperative ischemia-reperfusion (I/ R) with pronounced effects on neutrophil adhesion, endothelial function, myocyte contractility, oxidative stress & myocardial energetics. Diabetes (D) is an independent predictor of low cardiac output syndrome following CABG. The central hypothesis tested in these studies was that exaggerated production of ET_1 in D patients represents the common denominator underlying I/R induced myocardial dysfunction.

METHODS: Study 1 Arterial & coronary sinus ET_1 levels were assessed at baseline and at 1 & 10 mins following reperfusion in 9 D & 10 non-diabetic patients undergoing CABG. The difference between arterial & and coronary ET_1 Ievels served as an index of myocardial production. ET_1, was measured following acidic extraction using a commercial ELISA. Study 2 The effects of ET_1 & ET receptor blockade (using BQ-123) on vascular reactivity of atrial arterioles subjected to perioperative I/R was evaluated using videomicroscopy. Atrial microvessels (from appendages) were studied before & after removal of the aortic cross-clamp in a pressurized fashion. Reactivity to ET_1 & substance P was studied in the presence/absence of BQ-123. Study 3Effects of ET_1 on perioperative reactive oxygen species (ROS) were assessed using fluorescence activated cell sorting (FACS) in the presence of ET_1.

RESULTS: D patients elaborated more ET_1 at 1 & 10 mins following reperfusion ($p<0.01$). Atrial microvessels obtained before CPB & following 15 minutes of reperfusion revealed exaggerated responses to ET_1 in the D group ($p<0.01$). In addition endothelial responses to substance P were

attenuated in D arterioles & restored by BQ-123. Exposure to ET_1 Caused an increase in neutrophil ROS generation assessed using the FACS; an effect that was blocked by BQ-123 (p=0.003).

CONCLUSIONS: Perioperative production of ET,& vascular reactivity to exogenously administered ET_1 are augmented in D. ET_1 impairs endothelial function & enhances neutrophil ROS generation. These data underscore the importance of ET_1 as an important target of perioperative I/R injury in D.

**By Invitation*

L6. Intramyocardial Delivery of bFGF (Fibroblast Growth Factor) Improves Blood Flow in a Porcine Model of Hibernating Myocardium

Shankha S. Biswas*, Patrick W. Domkowski*, Luis H. Diodato*, John E. Scarborough*, Chad G. Hughes*, Carolyn Landolfo*, James E. Lowe*, Brian H. Annex*, Kevin P. Landolfo*, Victor J. Dzau*; Durham, NC

OBJECTIVE: Therpeutic angiogenesis is emerging as an alternative method of myocardial revascularization for certain end-stage coronary artery disease patients. In the current study we determined the effects of intramyocardial (IM) and intracoronary (IC) deliveries of bFGF on myocardial blood flow (MBF) and function in a porcine model of hibernating myocardium.

METHODS: Twenty four mini-swine with 90% left circumflex (LCx) stenosis and documented hibernating myocardium by positron emmision tomography (PET)& dobutamine stress echocardiography (DSE) were randomized to recieve either IM bFGF at 0.6mg/kg (mid-dose, 30 injections) or 6mg/kg (high-dose) or IM vehicle control. AN IC group received 6mg/ kg bFGF injected into the right coronary and Lex. PET and DSE were repeated at 1 & 3 months. DSE measures ischemia by changes in viable myocardium at peak stress determined by a regional wall motion score index, where 1= normal, 2=hypokinetic, 3=akineitc, and 4=dyskinetic. High dose stress response is shown.

RESULTS: in table.

CONCLUSIONS: In this model of hibernating myocardium, both doses of intramyocardial bFGF resulted in a significant improvement in regional MBF versus baseline. There were no changes in peak stress echocardiography in any group at either time point. These data suggest that intramyocardial delivery of bFGF, rather than intracoronary delivery may be optimal for augmenting myocardial blood flow in end-stage coronary artery disease.

	MID DOSE		**HIGH DOSE**		**CONTROL**		**IC (n=6)**	
	MBF	DSE	MBF	DSE	MBF	DSE	MBF	DSE
Baseline (n=6)	6.7±.4	1.8±.2	7.1±.2	2.0±.3	6.8±.3	2.2±.2	6.7±.4	2.0±.3
1-month (n=6)	7.9±.6	1.7±.2	8.1±.6	1.8±.2	7.4±.4	2.0±.2	7.1±.3	2.0±.2
3-month (n=6)	8.5±.5†	1.8±.2	8.2±.4†	1.9±.2	7.4±.7	2.1±.2	7.2±.4	2.2±.3

†P<0.05 vs. baseline, MVF expressed ml/gm/min, values ± SEM

**By Invitation*

L7. Cavopulmonary Anastomosis Induces the Expression of Stress-Related Genes: Implications for Pulmonary AVM Formation

Sunil P. Malhotra*, V. Mohan Reddy, You-Ping He*, Stephan Thelitz*, Frank L. Hanley, R. Kirk Riemer*; San Francisco, CA

OBJECTIVE: Cavopulmonary anastomosis (CPA) is often used for palliation of cyanotic heart disease. Unfortunately, clinically significant pulmonary arteriovenous malformations (PAVMs) occur in up to 25% of patients following surgery. CPA causes several modifications to the pulmonary circulation that may contribute to PAVM development. Our objective was to examine the role of one such alteration, reduced pulmonary blood flow (PBF), in the development of PAVMs by studying angiogenic and stress-related gene expression following pulmonary artery banding (PAB) and CPA.

METHODS: Lambs aged 35 to 45 days were placed into three groups: CPA (n=6), PAB (n=4), and sham controls (n=6). In our model, PAVMs are detectable by bubble-contrast echocardiography 8 weeks following CPA. To examine genes involved in PAVM development, tissue was harvested at 2 and 5 weeks after surgery. Expression of angiogenic and stress-related genes was determined by Western blotting. Quantitative assessment of expression was accomplished using scanning densitometry.

RESULTS: CPA and PAB both increased angiogenic gene expression, but only CPA induced the expression of endothelial stress-related genes. Vascular endothelial growth factor (VEGF) was upregulated 2.5 fold following both CPA ($p=0.002$) and PAB ($p=0.007$). However, CPA alone upregulated two markers of oxidative stress, hemoxygenase-1 and glucose transporter-1,2 ($p=0.004$) and 5 fold ($p=0.003$), respectively. PAB failed to induce expression of either protein. Expression of CD62, a marker of endothelial activation, was also unchanged following PAB, but increased 4 fold ($p=0.001$) following CPA.

CONCLUSIONS: Reduced PBF induces a pulmonary angiogenic response, but not an endothelial stress response. These results suggest that oxidative stress is more relevant to PAVM formation than angiogenic signaling, as PA banding does not result in PAVMs. The chronic oxidative stress of the pulmonary endothelium resulting from Cavopulmonary anastomosis may predispose the affected vasculature to arteriovenous shunting.

**By Invitation*

L8. Incorporation and Integration of Implanted Myogenic and Stem Cells into Native Myocardial Fibers: Anatomical Basis for Functional Improvements.

Edgar G. Chedrawy*, Jih-Shiuan Wang*, Dao M. Nguyen*, Dominique Shum-Tim*, Chu-Jeng Chiu; Montreal, PQ, Canada

OBJECTIVE: Myogenic and stem cells implanted into myocardium can differentiate into myocytes and functionally improve impaired ventricles. However, for implanted cells to actually contribute to the synchronous contractions of the heart, we need to demonstrate that they can be anatomically integrated with the existing native myocardial fibers.

METHODS: Isogenic Lewis rats were used as donors and recipients to stimulate clinical auto-transplantations. From donors, either skeletal myo-blasts or marrow stromal cells were isolated and culture-expanded. For cell labelling, either Lac-Z gene with adenovirus vector, or DAPI (4',6'-diamidino-2-phenylindole) was used. Labelled cells were then injected into the myocardium of recipients. At intervals, the specimens were obtained, sectioned and stained with H&E, X-Gal stain for beta-gal activities, and Connexin-43 stain to demonstrate gap junctions (intercalated discs).

RESULTS: At one week, the labelled cells were still undifferentiated, but at contact points between the implanted cells and the native myocytes, early expression of Connexin-43 can be detected. By 4 to 6 weeks, labelled fully differentiated myocytes can be seen to inter-connect among themselves and with native cardiomyocytes by means of intercalated discs. In sections parallel to the myofibers, full integration of new, labelled myocytes into the native myofiber cells can be observed. We postulate that such structural integration was enhanced by fiber stretching during cardiac contractions, sending signals for cellular re-orientation and incorporation.

CONCLUSIONS: We conclude that implanted precursor cells can be integrated into native myocardial structure to be functionally beneficial. Direct cell-to-cell contact seems to be an important signaling mechanism which has implications for cell implant strategies.

**By Invitation*

9:00 a.m. SCIENTIFIC SESSION -

Room 6A-C, San Diego Convention Center

Moderators: James L. Cox; Tirone E. David

Thoracic Surgery Foundation for Research and Education
David B. Skinner, President, New York, NY

34. Dilation of the Sinotubular junction Causes Aortic Insufficiency after Aortic Valve Replacement with the Toronto SPV Bioprosthesis

Tirone E. David, Maria Eriksson*, Joanne Bos*, Harry Rakowski*, Christopher M. Feindel, Joan Ivanov*; Toronto, ON, Canada

Discussant Neal D. Kon

OBJECTIVE: Dilation of the sinotubular junction (STJ) causes AI in patients with normal aortic cusps and in those who had aortic valve replacement (AVR) with stentless biological valves implanted in the sub-coronary position. This study was undertaken to determine the cause of late aortic insufficiency (AI) after AVR with the Toronto SPV (T-SPV). Since the diameter of the aortic annulus (AA) corresponds to the size of the T-SPV, we examined the echocardiographic relationship between the ratio STJ/AA and the development of AI.

METHODS: From 1991 to 1994, 174 patients underwent AVR with the T-SPV. To date, there have been a total of 19 deaths, none due to valve dysfunction. Patients have been followed from 7 to 9 years, mean of 7.4 years. All patients have had annual Doppler echocardiographic studies to assess valve function and to measure the diameters of the AA and STJ. The association of the ratio of STJ/AA with time and the degree of AI were evaluated by both, analysis of covariance and a mixed linear model.

RESULTS: Two patients had moderate AI since surgery probably due to technical error and were removed from analysis. Eighteen patients developed some degree of AI; moderate to severe AI began at one year (n=3, 18%) and increased thereafter: 2 years (18%), 3 years (22%), 4 years (28%), 5 years (44%), 6 years (65%), 7 years (60%), and 8 years (67%, p=0.001). Increasing AI was significantly associated with an increasing STJ/AA ratio: AI 0= 1.27±0.1, AI 1+ = 1.29±0.1, AI 2+=1.31±0.1, and AI 3+=1.44±0.1 (p=0.001 by one-way ANOVA). Analysis of covariance revealed that increasing AI (p=0.0001) and year of follow-up (p=0.017) were independently

associated with increased STJ/AA. The mixed linear model revealed an interaction between AI and year of follow-up (p=0.021) associated with increased STJ/AA.

CONCLUSIONS: Mild to severe AI appeared in 11% of patients during the first decade of follow-up. Dilation of the STJ was the main cause of late AI after AVR with T-SPV. We believe the use of a band of Dacron fabric around the aortic root at the level of the STJ can solve this problem.

**By Invitation*

35. Off-Pump Techniques Do Not Reduce Major Morbidity and Mortality of CABG

Joseph F. Sabik*, A. Marc Gillinov*, Eugene H. Blackstone, Cathy Vacha*, Penny Houghtalling*, Jose Navia*, Nicholas G. Smedira*, Patrick M. McCarthy, Bruce W. Lytle; Cleveland, OH

Discussant: Lawrence H Cohn

OBJECTIVE: To determine whether off-pump techniques reduce major morbidity and mortality associated with coronary artery bypass grafting.

METHODS: From 1997 to 2000, 528 consecutive patients (pts)had off-pump coronary artery bypass grafting (OPCAB). During the same period, 4184 pts had on-pump coronary artery bypass grafting (PCAB). Using propensity-based matching, 441 OPCAB pts and 502 PCAB pts were selected for comparison to assess the impact of off-pump techniques. OPCAB and PCAB pts were similar with respect to age(P=.4), stroke history (P=.7), peripheral vascular disease (P=.3), and chronic obstructive pulmonary disease (P=.2). However, PCAB pts had more 3 vessel coronary artery disease (P=.006), more left main disease (P=.001), and higher New York Heart Association functional class (P<.001).

RESULTS: Mean number of bypass grafts was 2.7 ± 1.1 in OPCAB pts vs 3.3 ± 0.9 in PCAB pts (P<.001). Morbidity and mortality are recorded in the table below.

CONCLUSIONS: Both OPCAB and PCAB produced excellent and comparable clinical results with low mortality and morbidity. Advantages associated with OPCAB may be limited to transient perioperative events such as postoperative encephalopathy.

Morbidity and Mortality of OPCAB and PCAB

	OPCAB_	PCAB_	P_
Mortality (%)	1.1	1.0	1
Stroke (%)	0.7	1.4	.4
Encephalopathy (%)	0.2	2.2	.007
MI (%)	0.9	1.2	.8
Reop for bleeding (%)	1.6	2	.6
Renal failure (%)	1.4	0.2	.06

The Helix and the Heart
Gerald D. Buckberg, Los Angeles, CA

Introduced by: James L. Cox, Washington, DC

10:15 a.m. INTERMISSION - VISIT EXHIBITS

11:00 a.m. SCIENTIFIC SESSION -

**By Invitation*

C. WALTON LILLEHEI RESIDENT
FORUM AWARD PRESENTATION

Room 6A-C, San Diego Convention Center

Moderators: James L. Cox.; Tirone E. David

36. Intraoperative Molecular Detection of Occult Micrometastases.

Siva Raja*, James D. Luketich, Sydney D. Finkelstein*, Lori A. Kelly*, Tony E. Godfrey*; Pittsburgh, PA

Discussant: Jack A. Roth

OBJECTIVE: Manypatients with histologically node negative tumors still suffer disease recurrence due to the presence of occult micrometastases (OM). We have recently shown that quantitative RT-PCR (QRT-PCR) can detect OM and predict recurrence. Current evidence indicates that these patients should benefit from neoadjuvant chemotherapy. To avoid separate staging and resection procedures however, OM status needs to be determined intraoperatively. The objective of this study was to develop and evaluate a rapid technique for QRT-PCR staging of lymph nodes that could be carried out intraoperatively.

METHODS: Our approach utilizes a new quantitative thermocycler (SmartCycler®) capable of fast cycling, combined with rapid RNA isolation, and a sensitive one tube RT-PCR assay. To validate the clinical relevance of this method, we studied carcinoembryonic antigen (CEA) expression in archived lymph nodes from 32N0 esophagus cancer patients with known outcome. Subsequently we studied 30 fresh-frozen, peri-esophageal lymph nodes from 17 patients (12 cancer patients and 5 non-cancer controls). Results were compared with frozen and fixed tissue histology.

RESULTS: In the retrospective analysis, rapid QRT-PCR predicted disease recurrence with a sensitivity and specificity of 88% and 75% respectively. Survival of patients who were QRT-PCR positive was lower than for those who were negative (40% vs 94%, p=0.002). On the fresh tissues, QRT-PCR was carried out in < 30 minutes starting with frozen tissue sections. All non-cancer control nodes had low or zero CEA expression. Rapid QRT-PCR found high levels of CEA expression in all histologically Nl nodes and in the nodes from two Nl patients who were N0 by intraopera-tive frozen section. All nodes from histologically N0 patients in this study had CEA expression levels similar to control nodes and to date these patients are disease free.

CONCLUSIONS: In this study, intraoperative QRT-PCR was consistently positive in all nodes that were histologically Nl by final pathology. Furthermore, rapid QRT-PCR was more accurate than frozen section examination. Thus, rapid QRT-PCR may be superior to intraoperative histology for determining patients nodal stage.

*By Invitation

37. Long-Term Effects of Angiogenic Therapy with bFGF Protein

Frank W. Sellke, Roger J. Laham*, Marc Ruel*, MarkJ. Post*, Anthony J. Ware*, Blazer R. Edelman*, Michael Simons*; Boston, MA; Bronx, NY

Discussant: Todd K. Rosengart

OBJECTIVE: The long-term effects of angiogenic therapy for ungraftable coronary artery disease are unknown. Three-year results of a randomized controlled trial of basic fibroblast growth factor (bFGF) therapy are reported.

METHODS: We conducted a randomized, double-blind, placebo-controlled study of bFGF (10 or 100 mg versus placebo) delivered via sustained-release heparin-alginate microcapsules implanted in ischemic and viable myocardial territories in patients undergoing CABG. Twenty-four patients were randomized to 10 mg of bFGF (n=8), 100 ug of bFGF (n=8), or placebo, in addition to undergoing concomitant CABG. Patients were followed 3 months and 3 years postoperatively with clinical assessment and stress nuclear perfusion imaging. Clinical follow-up was available for all patients (mean 32.2 ± 6.8 months, range 36 to 43 months).

RESULTS: There were two late deaths, one of pancreatic cancer and one of end-stage peripheral vascular disease (both in the 100 ug bFGF group; P=NS). Two patients in the control group underwent repeat cardiac catheterizations for recurrent coronary events. Mean Canadian Cardiovascular Society (CCS) angina class improved at 3 months and 3 years from baseline in all groups (2.14±1.35 and .86±.90 versus 2.86±.69 in the control group at 3 months, 3 years, and baseline, respectively; .88±.83 and .13+.35 versus 2.86±.69 in the 10 ug bFGF group; 43±79 and .29±.79 versus 3.14±.64 in the 100 µg bFGF group; all P<.05). Improvements in CCS angina class were more pronounced in patients who received either 10 µg or 100 µg of bFGF over controls (P<.05). At 3 years, stress nuclear perfusion scans revealed a persistent reversible defect in all patients in the control group, while 3 out of 8 patients who received 10 µg or 100 µg of bFGF had no recognizable perfusion defect.

CONCLUSIONS: Angiogenic therapy appears to exert a prolonged myocardial revascularization effect that may lead to sustained clinical benefit. Further studies are required to fully determine the long-term safety of this new therapeutic modality.

*By Invitation

11:45 a.m. ADDRESS BY HONORED SPEAKER

Bioengineering for the Exploration of Space: New Challenges

Professor Mory Gharib

Aeronautics and Bioengineering

California Institute of Technology, Pasadena, California

Introduced By: James L. Cox, Washington, D.C.

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

12:30 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

Room 7A, San Diego Convention Center

TUESDAY AFTERNOON, MAY 8, 2001

1:45 p.m SIMULTANEOUS SCIENTIFIC SESSION A-3 -ADULT CARDIAC SURGERY

Room 6A-C, San Diego Convention Center

Moderators: Delos M. Cosgrove; O. Wayne Isom

38. Impact of Target Stenosis and Location on Radial Artery Graft Patency

Hersh Maniar*, Hendrick Earner, Sunil Prasad*, Tarek Absi*, Pavlos Moustakidis*, Yi Zhang*, Marci Bailey*, Thoralf Sundt; Saint Louis, MO
*Discussant: Brian Buxton**

OBJECTIVE: The radial artery (RA) is widely used for coronary artery bypass (CAB); however, factors impacting RA patency have not been thoroughly examined. We investigated the influences of target vessel stenosis and location on RA graft patency.

METHODS: Between October 1993 and April 2000, 960 patients underwent CAB with an internal thoracic artery (ITA) / RA T-graft. We reviewed the angiograms for 76 patients presenting with signs and symptoms of ischemia, at an average of 22.3 ± 15.8 months postoperatively.

RESULTS: A total of 161 RA anastomoses were studied and overall RA graft patency was 77% (124/161). Among the patent grafts, target vessels had an average stenosis of 80.6% and of the occluded grafts, target vessels had a stenosis of 71.3%, $p<0.01$. The patency rate of bypass grafts in lesions with >75% stenosis was 83.3% while the patency rate in lesions with <75% stenosis was 67.2%, $p<0.05$. Overall patency for distal anastomoses to the left coronary artery and its branches was 85.9%; anastomoses to the branches of the right coronary artery (RCA) had a patency rate of 65.2%, $p<0.01$. Among occluded grafts, there was a greater proportion of anastomoses to the RCA and its branches (24/37) when compared to patent grafts (45/124). 64.9% vs 36.3% respectively, $p<0.01$. The patency of ITA anastomoses was 94% (102/110) and was not influenced by either target vessel stenosis or location.

CONCLUSIONS: Within this group of symptomatic patients, RA graft patency appears to be influenced by both target vessel location as well as target vessel stenosis. Although RA graft patency to the left coronary circulation remains encouraging, the use of RA conduits in a T-graft may be sub optimal in less than severely stenosed vessels or to branches of the right coronary artery.

**By Invitation*

39. Neurological and Pulmonary Protection During Surgical Treatment of Type A Acute Aortic Dissection

Maurizio Cotrufo, Attilio Renzulli*, Luca S. De Santo*, Cristiano Amarelli*;

Naples, Italy

Discussant: Joseph Bavaria

OBJECTIVE: Strategies of brain protection during repair of type A acute aortic dissection imply hypotermia. Brain injury, respiratory failure and coagulation disturbance represent major complications, whose mechanism are still under investigation. Aim of our prospective randomized study was to assess the efficacy of Kazui technique, selective hypotermic cerebral perfusion with mild systemic hypothermia.

METHODS: From January 1998 to September 2000,106 consecutive patients referred for acute type A aortic dissection, who were free from pre-operative neurological and respiratory dysfunction, were randomly assigned to three groups: group A included 38 patients operated on hypotermic circulatory arrest, group B 38 patients operated on retrograde cerebral perfusion and group C 30 patients operated on Kazui technique. Clinical outcomes and perioperative data were analysed. Cerebral injury was evaluated by S-100 serum release, while lung function was evaluated by intubation time, serum levels of IL-1, TNF alfa, 11-6, 11-8 and arterial/ alveolar oxygen tension ratios at nine intervals in the first 72 hours.

RESULTS: Study groups were homogeneous as to age, sex, interval between symptom onset and surgical operation, previous aortic surgery and concomitant surgical procedures. Cardiopulmonary bypass time and operation time, along with early post operative blood substitutes requirement were significantly higher in whole body hypotermia groups (A+B). Stroke rate and incidence of pulmonary dysfunction proved significantly lower ($p<0.05$) in Group C. Increases in postoperative serum S-100 and IL-1, TNF alfa, Il-6, Il-8 levels were significantly higher ($p=0.034$, $p= 0.025$ respectively). Incidence of prolonged ventilator support (>72 hours) and the mean arterial/alveolar oxygen tension ratios were lower in group C ($p=0.023$, $p=0.016$ respectively). Statistical difference was found in hospital mortality (12.8 % overall) among the three groups with better outcomes in Group C (8.4% vs 16% vs 20%).

CONCLUSIONS: Kazui technique proved safe and effective in protecting brain function and reducing the incidence of blood loss and pulmonary dysfunction.

**By Invitation*

40. Incomplete Regression of Hypertophy Following AVR is not Influenced by Valve Size, nor Patient-Prosthesis Mismatch.

Naoji Hanayama*, Hari R. Mallidi*, Vivek Rao*, Gideon Cohen*, Bernard S. Goldman, Stephen E. Fremes, Peter R. Mitoff*, Christopher D. Morgan*, Campbell D. Joyner*, George T. Christakis; Toronto, ON, Canada

Discussant: Tirone E. David

OBJECTIVE: Incomplete regression of left ventricular hypertrophy (abn-LVMI) following AVR for aortic stenosis (AS) may decrease long-term survival. In this prospective study, we identified the causes of abn-LVMI.

METHODS: Between 1990-00, 529 pts undergoing AVR for AS had clinical and hemodynamic data collected prospectively. Preoperative and annual postoperative transthoracic echoes were employed to assess left ventricular mass index (LVMI) and hemodynamics. Abn-LVMI was defined as the 25^{th} percentile or less of the lowest postoperative LVMI (>128mg/ m^2n=133). All other patients were included in the normal regression group (N-LVMI). Univariate and multivariable logistic regression analyses were used to determine the predictors of abn-LVMI.

RESULTS: Valve size (SIZE mm), mean postoperative gradients (MGRAD mmHg) and effective orifice area (EOA cm^2) and patient-prosthesis mismatch (PPM, indexed EOA<0.59cm^2/m^2) did not predict abn-LVMI. The extent of preoperative hypertrophy (pre-LVMI) was significantly predictive (*=P<0.0001). Survival (93.4+1.8% vs 94.8±2.3%, p=0.90) and freedom from NYHAIII-IV (75.0±3.7% vs 76.6±5.3%,p=0.60) were similar for both groups at 7 years. By logistic regression the most important positive predictor of abn-LVMI was the extent of preoperative LVMI, with an odds ratio of 37.5 (p<0.0001).

CONCLUSIONS: Valve hemodynamics was not an important predictor of incomplete regression of hypertrophy. The extent of preoperative hypertrophy was the most important predictor, suggesting earlier surgical intervention may reduce the incidence of hypertrophy postoperatively. Furthermore the importance of LV hypertrophy on long-term survival must be reassessed, in the absence of scientific evidence.

	SIZE	MGRAD	EOA	PPM %	Pre-LVMI
Abn-LVMI	24.5±2.3	13.6±6.8	1.7±0.57	8.6	169.2±42.11
N-LVMI	24.0±2.5	14.9±8.0	1.7±0.56	9.7	126.3±35.8

**By Invitation*

41. Impact of the Cox Maze Procedure on the Outcome in Patients with Atrial Fibrillation and Mitral Valve Disease

Ko Bando, Junjiro Kobayashi*, Yoshio Kosakai*, Yoshikado Sasako*, Hiroaki Konishi*, Yoshio Kosakai*, Yoshikado Sosako*, Hiroaki Konishi*, Kanshi Komatzu*, Toshihiko Sato*, Satoshi Nakatani*, Kazuo Niwaya*, Osamu Tagusari*, Toshikatsu Yagihara*, Soichiro Kitamura; Osaka, Takarazuka, Tochigi, and Sapporo, Japan; Geneva, Switzerland
Discussant: Renee S Hartz

OBJECTIVE: We sought to determine whether the Cox maze procedure provides adjunctive benefit to patients (pts) with atrial fibrillation (Af) undergoing mitral valve surgery.
METHODS: Between 5/92 and 8/00, we performed 256 Cox maze procedure plus mitral valve replacement (MVR/maze, n=144) or repair (MVP/maze, n=112). We compared these outcomes with those of 65 control pts with pre-op Af who had MVR alone during the same time interval. Pts in these 3 cohorts were similar for age, gender, pre-op NYHA III/IV class. Control pts did not undergo a maze procedure primarily due to 1)multiple previous heart operations, 2)longer duration of pre-op Af (>20yrs), 3)f-wave in V_1 leads <0.1 mV, or 4) emergent surgery.

RESULTS: See table. Multivariable analysis using Cox hazards model revealed that risks for late stroke were No Cox maze procedure (p=0.0001) and Af 3 months after surgery (p=0.002). Systemic anticoagulation did not prevent late strokes after MVR. A Cox maze procedure with MVR significantly reduced the stroke risk.

CONCLUSIONS: The addition of the Cox maze procedure to mitral valve repair/replacement is safe and effective in selected pts. Elimination of Af significantly decreased the incidence of late stroke and improved functional outcome.

	MVR	MVR/maze	MVP/maze	p Value
Mechanical valve	61/65	140/144	_____	*0.43
Anticoagulation	59/59	134/138	9/109	*<0.001

Op mortality	1.6%	1.4%	1.8%	*0.97
Late survival (3yrs)	93.1±3.3%	95.0±2.0%	98.2±1.3%	†0.063
Free from stroke (3yrs)	94.6±5.9%	98.3±1.2%	99.1±0.9%	†<0.0001
Free of Af (3yrs)	4.6±2.6%	83.3±3.2%	87.2±3.4%	†<0.0001
Post/pre VO_2 increase	11.6±1.9%	22.2±1.5%	24.6±2.1%	‡0.0003

*by chi square test, †by log rank test, ‡ by ANOVA

3:05 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A-4 -ADULT CARDIAC SURGERY

Room 6A-C, San Diego Convention Center

Moderators: Delos M. Cosgrove; O. Wayne Isom

42. Hemodynamic Stability Over 17 Years of the Carpentier-Edwards Aortic Pericardial Bioprosthesis

Michael K. Banbury*, Delos M. Cosgrove, James D. Thomas*, Eugene Blackstone, J. Edward Okies, Albert H. Krause, †Leon Q. Colburn*,†Robert M. Prater; Cleveland, OH; Portland, OR; Bronx, NY

Discussant: W.R. Eric Jamieson

OBJECTIVE: To determine the long-term stability of the hemodynamic performance for the Carpentier-Edwards aortic pericardial bioprosthesis using late and serial echocardiographic studies.

METHODS: 87 patients prospectively followed since implant underwent echocardiographic examinations as late as 17.4 years post implant. Serial echo examinations collected by a core group of sonographers were available on 40 patients. Summary hemodynamic data and longitudinal analyses of serial data were performed. Results are correlated with patient functional class.

RESULTS: Summary hemodynamic and functional class data are presented in the table. Serial echo examinations with a mean interval (latest-earliest) of 5.0 ± 3.0 years between echoes had a 6.5 ± 15.7 mmHg increase in mean gradient (p=0.045), mean increase in effective orifice area (EOA) of 0.11 ± 0.25 (p=0.18) and small, though significant increase in aortic re-gurgitation (AR) of 0.6 ± 0.9 (p=0.002).

CONCLUSIONS: The Carpentier-Edwards aortic pericardial bioprosthesis demonstrates acceptable long-term transvavular gradients and mild aortic regurgitation while New York Heart Association class remained low up to 17 years post implant. Serial echo exams demonstrate clinically trivial changes in valve function over time.

Valve size	N	Mean time to echo ± SD (years)	Mean gradient ± SD (mmHg)	AR±SD (0-4+)	Mean NYHA class
19 mm	10	12±4.2	33.1±31.4	1.5±1.4	1.3±0.5
21 mm	32	10.5±4.2	23.4±14.8	1.0±0.7	1.7±0.8

23 mm	26	11.2±3.8	17.3±8.8	1.0±1.0	1.5±0.7
25 mm	13	12.6±3.0	15.8±8.8	1.4±1.4	1.5±0.9
27 mm	3	12.6±1.6	N/A	N/A	1.7±0.6
29 mm	3	12.5±4.2	19.0±-	2.5±1.8	1.5±0.7
Overall	87	11.4±3.9	21.7±6.9	1.0±0.6	1.5±0.7

†Authors have a relationship with Edwards Lifesciences

**By Invitation*

43. Midterm Follow-up of Penetrating Ulcer and Intramural Hematoma of the Aorta

Raymond J. Lynch*, Patricia E. Cole*, Tittle L. Shawn*, Harsimran S. Singh*, John A. Rizzo*, Gary S. Kopf, John A. Elefteriades; New Haven, CT

Discussant: Thoralf M. Sundt

OBJECTIVE: Most studies on variant forms of aortic dissection - penetrating ulcer (PU) and intramural hematoma (IMH) - have focused on the initial presenting episode, with scant followup. This investigation provides midterm followup of PU and IMH to determine whether the aorta "heals" radiographically, goes on to dilate, or tends to rupture in later followup.

METHODS: 45 Pts with PU or IMH were treated at our institution (26 PU and 19 IMH). For IMH, 8 pts were male and 11 female, and age ranged from 54 to 88 (mean 74). For PU, 10 pts were male and 16 female, and age ranged from 54 to 87 (mean 72).

RESULTS: For IMH, 5 pts (26%) ruptured during the initial admission, 7 underwent surgery, and 16 survived to hospital discharge (84%). For PU, 10 pts (38%) ruptured during the initial admission, 16 underwent surgery and 22 survived to hospital discharge (84%). Follow-up ranges from 9 to 12.5 years (mean 3.4 years). No ischemic vascular complications occurred. On imaging followup, 23% of pts showed resolution, 23% worsened, 35% went on to typical dissection, and 18% were unchanged. 6 late deaths were known due to rupture. For IMH, aortic diameter increased from 5.3 to 5.9 cm over 21 months. For PU, aortic diameter increased from 4.8 to 5.1 cm over 14 months.

CONCLUSIONS: 1. IMH and PU rupture both early and late. 2. Radio-graphic worsening, improvement, or frank dissection may occur. 3. Aortic growth does occur (0.3 cm/yr). 4. Vascular ischemic complications do not occur.

**By Invitation*

44. Revascularization of the Lateral Wall: Radial Artery Versus Right Internal Mammary Artery. Long Term Angiographic and Clinical Results.

Antonio Maria Calafiore, Michele Di Mauro*, Valeric Mazzei*, Piero Pelini*, Giuseppe Vitolla*, Marco Contini*; Chieti, Italy

Discussant: Alfred J Tector

OBJECTIVE: To evaluate if radial artery (RA) has the same results of right internal mammary artery (RIMA) in lateral wall revascularization in the long term.

METHODS: From January 1992 to September 1996, 288 patients had myo-cardial revascularization using LIMA on LAD. The lateral wall was grafted with RA in 139 cases (group A) and with RIMA in 149 cases (group B). Groups were different only because of older age and higher incidence of urgent patients in group A. Y grafting was used in 86.4% of cases in group A and in 34.8% in group B ($p < 0.001$) Anastomoses per patient were similar in both groups (3.2±0.8 vs 3.2±0.9, p ns).

RESULTS: 30 day mortality was similar, 2.1% vs 2.0%, p ns. Late deaths were 13 in group A vs 9 in group B (p ns). Cause of death was cardiac related in 7 patients (group A) vs 7 (group B). Late redo or PTCA were performed in 2 patients in group A and in 1 in group B (p ns). 8 years survival was 88.512.7 in group A vs 92.6±2.1 in group B (p ns). Event free survival 87.0±2.0 vs 91.9±2.2 (p ns). Patency rate within 30 days was 99.0% (group A, 105/106 LIMA+RAanastomoses) vs 100% (group B, 52/52 BIMA anastomoses). After a mean of 35.4±27.6, patency rate was 95.3% (group A, 82/85 LIMA+RA anastomoses) and 100% (group B, 33/33 BIMA anastomoses), p ns.

CONCLUSIONS: In the long term lateral wall grafting with RA gives the same clinical and angiographic results than RIMA grafting.

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

Room 6A-C, San Diego Convention Center

7:00 p.m. ATTENDEE RECEPTION - NAUTICAL PORTS OF CALL

San Diego Convention Center Outdoor Terrace

(Separate Subscription)

**By Invitation*

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B-3 - GENERAL THORACIC SURGERY

Room 1A/B, San Diego Convention Center

Moderators: Douglas E. Wood; Michael A. Maddaus

45. Aggressive Therapy of Stage III Non-Small Cell Lung Cancer and Metachronous Brain Metastases

Nader Moazami*, Thomas W. Rice, Malcolm M. Decamp*, Sudish C. Murthy*, Eugene H. Blackstone, Lisa A. Rybicki*, John H. Suh*, Gene H. Barnett*, David J. Adelstein*; Cleveland, OH

*Discussant: Raphael Bueno**

OBJECTIVE: We have aggressively treated stage III non-small cell lung cancer (NSCLC) and metachronous brain matastases(MBM)whenever possible.The purposes of the study were to

evaluate results of this protocol,identify predictors of improved survival and characterize ideal profiles for aggressive therapy.

METHODS: Between 1989 and 2000, 93 stage III NSCLC patients had MBM, 53(57%)were stage IIIA and 40(43%) IIIB. Median age was 60 years (range 32-78). Aggressive NSCLC therapy was resection in 58(62%), 26(28%) with induction therapy and 22(24%) with post-operative adjuvant therapy. 35(38%) were palliated, 29 (31%) with radiation therapy and 6(6%) with supportive care. The interval between NSCLC treatment and MBM diagnosis was 9 months(range 1-59). MBM therapy was aggressive in 48(52%), 24(26%) with resection and 24(26%)with gamma knife therapy. 45(48%)were palliated, 44 (47%)with whole brain radiation therapy and 1 (l%)with supportive care.

RESULTS: Median, 1, and 2-year survivals were 5 months, 22% and 10%, respectively. Multivariable analysis identified younger age(P<.001), stage IIIA (P=.003), NSCLC resection (P=.02),and aggressive therapy of MBM, by either resection or gamma knife therapy CP<0.001) as predictors of improved survival. Patient profiles and survival are listed in table.

CONCLUSIONS: Younger patients with resected IIIA NSCLC should have either resection or gamma knife therapy of MBM.

Age (yrs)	Stage	Aggressive Lung/ MBM RX (mo.)	Median survival	1-year survival	2-year survival
40	IIIA	Yes	NR	94%	88%
60	IIIA	Yes	NR	83%	70%
40	IIIB	No	5	5%	0
60	IIIB	No	2	0	0

NR= not reached

**By Invitation*

46. Is Surgical Salvage Indicated for Recurrent Esophageal Tumors Following Definitive Chemoradiation?

Stephen G. Swisher*, Paula Wynn*, Melinda B. Mosheim*, Joe B. Putnam,

Ritsuko R. Komaki*, Jaffer A. Ajani*, W. Roy Smythe*, Ara A. Vaporciyan",

Jack A. Roth, Garrett L. Walsh*; Houston, TX

Discussant Douglas J. Mathisen

OBJECTIVE: Some patients (pts) and oncologists choose to treat localized esophageal cancer with definitive chemotherapy and radiation therapy rather than surgery. A subset of these pts will relapse locally without distant metastases and have no other "curative intent" treatment option except for esophagectomy. The results of salvage surgery in this group of pts have not previously been reported.

METHODS: We reviewed our experience with salvage surgery after failed definitive chemoradiation from 1987 to 2000 (n=13) and compared them with pts undergoing immediate surgical resection after planned preoperative chemoradiation (n=99).

RESULTS: All pts who relapsed following definitive chemoradiation (n=13) had pathologic documentation of recurrent esophageal tumor without evidence of metastatic disease. Average time to relapse was 18 mos (4 to 55 mos). 5 year actuarial survival following salvage surgery of relapsed esophageal tumors (n=13) was 25% with 2 pts still alive 5.6 and 12.3 yrs after resection.

CONCLUSIONS: Pts who undergo surgical salvage for recurrent esophageal tumors after definitive chemoradiation have increased morbidity, mortality and hospital resource utilization. Nevertheless, long-term survival can be achieved with surgical salvage and should therefore be considered in carefully selected pts.

Outcome	Definitive Chemorad (n=13)	Preop Chemorad (n=99)	P Value
Op Time (min)	541±172	429±141	0.01*
Blood Loss (ml)	1212±832	843±527	0.03*
Ventilator (days)	9.0±13.3	3.3±8.1	0.03*
ICU (days)	11.2±13.3	5.1±9.4	0.04*
Hosp Stay (days)	29.3±22.4	18.4±18.3	0.05*
Anast Leak	5 (38%)	7 (7%)	0.0005*
Op Mort	2 (15%)	6 (6%)	0.2

**By Invitation*

47. Primary Lung Carcinoma after Heart or Lung Transplantation-Epidemiology, Management and Outcome

Ani C. Anyanwu*, Edward R. Townsend*, Nicholas R. Banner*, Margaret Burke*, Ashghar Khaghani*, Magdi H. Yacoub; Middlesex, United Kingdom

Discussant: Thomas J. Kirby

OBJECTIVE: To examine the epidemiology, management and outcome of lung carcinoma occurring after heart or lung transplantation.

METHODS: Since 1980, we have performed over 2000 thoracic transplants at our institution. Annual review included a chest radiograph in all patients - those with suspicious symptoms or an abnormal radiograph were are investigated for possible lung carcinoma. We retrospectively reviewed all cases of primary lung carcinoma diagnosed between 1990 and 2000.

RESULTS: Seventeen cases were identified (15 orthotopic heart 1 hetero-topic heart, and 1 single lung (tumour in non-transplanted lung) transplant). Median time to diagnosis of lung carcinoma was 89 months after transplant (range of 46 to 138 months). The predominant presentation was as an incidental finding on chest radiography (13/17). All but one were aged above 50 years; 16/17 were male. All patients had smoked prior to transplant; 5 continuing to smoke after transplant. Histological types were squamous carcinoma (11), adenocarcinoma (3), small cell carcinoma (2), and anaplastic carcinoma (1). 11/17 patients had stage I or II disease at time of diagnosis. Of these, nine underwent surgery (2 patients unfit for surgery had radiotherapy). Other patients received palliative chemo/ra-diotherapy. Surgical procedures were lobectomy (5); wedge or segmental excision (3); thoracotomy, no resection (1). Median survival post diagnosis was 11 months for all

patients, and 24 months for those who had surgical resection. 6/8 patients who had surgical resection subsequently died (survival 2, 9, 21, 21, 21, 36 and 66 months); 2 remain alive after 8 and 48 months.

CONCLUSIONS: The epidemiology of lung cancer in these patients was similar to that in the general population. Immunosuppression appeared to be incidental as lung cancer did not occur in: younger recipients, non-smokers, or donor lungs. Poor respiratory or cardiac function sometimes prevented surgical treatment but, when possible, surgery appears to be the treatment of choice for those with stage I or II disease as it can result in medium term survival.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION B-4 - GENERAL THORACIC SURGERY

Room 1A/B, San Diego Convention Center

Moderators: Douglas E. Wood; Michael A. Maddaus

48. Donor Cause of Death: Does it Affect the Outcome of Lung Transplant?

ˆžAnna Maria Ciccone*, Bryan F. Meyers*, Tracey J. Guthrie*, Richard J. Battafarano*, Elbert P. Trulock*, Joel D. Cooper, G. Alexander Patterson; St. Louis, MO

*Discussant: John V. Conte**

OBJECTIVE: The inadequate supply of suitable donor lungs limits the application of lung transplantation. It has been suggested that cause of donor death influences post transplant allograft function. A retrospective analysis of our adult lung transplant experience was conducted to determine the influence of donor traumatic brain injury (TBI) versus non-traumatic brain injury (NTBI).

METHODS: A retrospective review of recipient medical charts and UNOS donor records was conducted on 500 consecutive transplants performed between 7/88 and 12/99. Recipient follow-up was complete with a minimum time interval of 10 months.

RESULTS: There were 295 and 205 donors in the TBI and NTBI groups respectively. Young minority male donors predominate the TBI group. With respect to recipient age, gender, diagnosis, type of transplant (single versus bilateral) or requirement for pre-transplant mechanical ventilatory assistance, there was no difference in allocation of TBI versus NTBI donors. Stratified by TBI versus NTBI recipient outcomes by univariant analysis revealed no significant difference in immediate or 24-hour PaO2/FiO2 ratio, ventilation time, hospital stay, incidence of early rejection by transbronchial biopsy, hospital mortality, or overall survival. However, overall incidence of bronchiolitis obilterans syndrome (BOS) was 51.2% in the TBI recipients as compared to 35.1% in the NTBI recipients ($p<0.001$). Kaplan-Meier estimation of freedom of BOS at 5 years was 34.5% and 50.8% for TBI and NTBI recipients respectively ($p=0.002$). Upon further analysis with proportional-hazards multivariate analysis, longer ischemic time ($p=0.004$), increased recipient age ($p=0.005$), as well as donor TBI ($p=0.03$) were found to be predictor variables for increased risk of BOS.

CONCLUSIONS: Cause of donor brain death does not influence early results of lung transplantation but donor TBI, as well as prolonged ischemic time and older age, may predispose recipients to subsequent development of BOS.
ˆž*2001-02 AATS Graham Fellow*

**By Invitation*

49. Influence of the Donor Lung on Outcomes Following Lung Transplantation: An Analysis of Paired Grafts from the Same Donor

Steven M. Fiser*, Irving L. Kron, Stewart M. Long*, Aditya K. Kaza*, John A. Kern*, Curtis G. Tribbie; Charlottesville, VA

Discussant: Shaf Keshavjee

OBJECTIVE: Some authors have suggested that inadequate preservation may account for many of the adverse outcomes that occur following lung transplantation. In order to study the influence of donor lung preservation on early and late outcomes following lung transplantation, we reviewed our paired recipients who received lung allografts from a single donor (2 recipients, 1 donor). Our hypothesis was that if one graft developed an adverse outcome, the other graft of each pair was at increased risk for developing that same outcome.

METHODS: Data on 70 lungs (35 paired sets) was reviewed for identification of adverse outcomes (Table I). For each paired set of lungs in which at least one lung had an adverse outcome, the incidence of that outcome in the other lung of each set was calculated (Study Group).

RESULTS: The incidence of postoperative ischemia-reperfusion (IR) injury, an initial arterial oxygenation (PO2) less than 150 on 100% oxygen, multiple acute rejection (AR) episodes within the first 6 months, bacterial/fungal pneumonia (PNA) within the first 6 months, cytomegalovirus (CMV) infection within the first 6 months, onset of bronchiolitis obliter-ans syndrome (BOS), one month mortality (1 M Mor), and one year mortality (1 Y Mor) were not significantly increased in the study group compared to the entire population (Table I).

CONCLUSIONS: Contrary to our hypothesis, adverse outcomes following lung transplantation, including ischemia-reperfusion injury, are not predicted by factors solely associated with preservation of donor lungs and are likely a result of complex interactions between the recipient and the transplanted graft.

Table 1: Incidence of Adverse Outcomes

	IR	PO2‰¤150	AR‰¥2	PNA	CMV	BOS	1 M Mor	1 Y Mor
All	16/70	8/70	8/70	17/70	8/70	25/70	8/70	16/70
Lungs	23%	11%	11%	24%	11%	36%	11%	28%
Study	3/13	1/7	1/7	4/13	1/7	4/21	0/8	2/14
Group	23%	14%	14%	31%	14%	19%	0%	14%
p value	0.99	0.96	0.96	0.67	0.97	0.20	0.59	0.72

**By Invitation*

50. Radical En Bloc Resection for Apical Lung Cancer Invading the Spine

Dominique H. Grunenwald*, Giulia Veronesi*, Philippe Girard*, Raffaele Caliandro*, Denis Debrosse*, Dominique Gossot*, Jean-Luc Le Guillou*, Lorenzo Spaggiari*, Thierry Le Chevalier*, Christian Mazel*; Paris, France; Milan, Italy; Villejuif, France

*Discussant: Garrett L. Walsh**

OBJECTIVE: Surgical resection of Pancoast's tumors invading vertebral bodies is a complex operation with high risk of postoperative morbidity but few data on long term survival and local failure are available. We reviewed our 7-years experience upon en bloc partial or total vertebrectomy and report outcome and survival rates.

METHODS: Nineteen patients with apical chest tumor involving the spine underwent en bloc resection. Eleven received induction treatment (chemotherapy: 5; chemoradiotherapy: 4; radiation: 2). Pneumonectomy was performed in 3 cases, lobectomy in 13, and wedge resection in 3. Hemivertebrectomy was done in 15 cases, total vertebrectomy in 4. Median number of resected vertebral bodies was 3 (1 to 4). Tumor stage was IIIB in 15 patients and IIIA in 4 (hemi vertebrectomy in case of T3 to get free margins). Nodal status was N0 in 11 patients, N1 in 3, N2 in 2, N3 (supraclavicular) in 2, and NX in one. Complete resection was achieved in 15 cases (79%).

RESULTS: There was no postoperative mortality. Morbidity was observed in 13 cases, including 5 complications (26%) related to spinal surgery. Median hospital stay was 33 days. Nine patients are alive after a mean follow-up of 19 months (1 to 63). One- and five-year predicted survival rates are 59% and 26%, respectively. Five local recurrences were observed.

CONCLUSIONS: Although en bloc resection of apical chest tumors with vertebrectomy is technically demanding and postoperative morbidity represents an important limit to this aggressive surgery, encouraging long term survival rate observed in this series suggests that it could be a valid option in selected patients with vertebral involvement.

**By Invitation*

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C-3 - CONGENITAL HEART DISEASE

Room 6D/E, San Diego Convention Center

Moderators: John J. Lamberti; Constantine Mavroudis

51. Early and Long-term Results of Surgical Treatment of Pulmonary Atresia with Ventricular Septal Defect.

John M. Cho*, Francisco J. Puga, Gordon K. Danielson, Joseph A. Dearani*,

Douglas D. Mair*, Donald J. Hagler*, Paul R. Julsrud*, Duane M. Ilstrup*;

Washington, DC; Rochester, MN

Discussant: William G. Williams

OBJECTIVE: To determine the results of surgical treatment of patients (pts) with pulmonary atresia (PA), ventricular septal defect (VSD) with or without aorto-pulmonary arterial collaterals (MAPCAs).

METHODS: The records of 490 pts operated from 1977-1999 were reviewed. Follow-up information was obtained by return visits, and/or letters to the patient and referring physician. Standard statistical methods were used. Pts were divided in two groups: A. Pts who have not yet undergone or have been rejected for complete repair. B. Pts who have undergone complete biventricular repair.

RESULTS: Group A (160 pts). 85 (53%) had palliative procedures; 45 (28%) had preliminary surgical stages (unifocalization (UNI), RVOT reconstruction and/or shunts) and are waiting for complete repair; 34 (21%) had all surgical stages but were rejected for complete repair. Early and late mortality were 16.3% (26) and 23.1% (31). Mean follow-up = 72.3 months (1-369 months). The sole risk factor for mortality was the presence of MAPCAs (p=0.0182). Group B (335 pts). Mean age at complete repair = 11.3 years SD 9.2. 103 pts (30%) had single stage repair, while 232 (69%)had staged reconstruction. Staged procedures were: shunts (195), UNI (126) and RVOT reconstruction (88). 24 pts (7%) underwent intra-operative reopening of the VSD because of high RV pressure. Mean post-repair PRV/LV presure = 0.65, SD 0.163. 15 pts died early. Risk factors were PRV/LV pressure ratio > 0.7, and reopening of VSD (p=<0.05). Mean follow-up = 10.4 years, SD > 7.8. 52 pts (16.1%) died late. Risk factors were female gender, nonconfluent or absent central pulmonary arteries, and reopening of the VSD (p=<0.05). Reoperations included conduit change (106), reclosure of VSD (29), aortic valve replacement (27), tricuspid annuloplasty (10). 84 pts needed interventional catheter procedures for residual lesions.

CONCLUSIONS: Surgical repair of pts with simple or complex forms of pulmonary atresia and ventricular septal defect can be achieved with low early mortality. Late mortality and need for reoperation, especially conduit replacement, continue to affect the long-term well-being of these pts.

**By Invitation*

52. Selective Use of Surgical Aortppulmonary Window in Pulmonary Atresia Patients with Poorly Developed True Pulmonary Arteries and Major Aortopulmonary Collaterals.

Mark D. Rodefeld*, V. Mohan Reddy, Lenardo D. Thompson*, Phil C.

Moore*, David F. Teitel*, Frank L. Hanley; San Francisco, CA

Discussant: Michael D. Black

OBJECTIVE: The morphology of the pulmonary circulation varies widely in patients with pulmonary atresia, ventricular septal defect, and major aortopulmonary collaterals (PA/VSD/MAPCAs). Although we continue to favor one-stage unifocalization and complete repair as the procedure of choice, a subgroup of patients who meet specific criteria have been managed with initial creation of surgical aortopulmonary (AP) window. The selection criteria, surgical procedure, and outcome of these patients are reviewed.

METHODS: Eighteen patients with PA/VSD/MAPCAs who were considered unsuitable candidates for one-stage repair underwent surgical AP window. The criteria for selection include: 1) presence of centrally confluent true pulmonary arteries (PAs) of less than 2.5 mm diameter, with

a relatively normal peripheral arborization pattern; 2) MAPCAs which are multiple, have significant hypoplasia or stenoses, and/or communicate with the true PA system. Mean age at operation was 3.6 months. Mean number of MAPCAs was 3.75 (range 2-6).

RESULTS: Seventeen patients underwent creation of AP window with native tissue-to-tissue anastomosis. In one case a synthetic aortic to PA shunt was required. There were no deaths. Morbidity was minor. Follow-up angiography (n=13) demonstrates good growth of true PAs in 12/13 cases (92%). Mean PA diameter increased from 2.0 mm to 4.5 mm. Subsequent operation has been performed in 13/18 patients (72%). One-stage complete unifocalization was achieved in 7/13 (54%). Six have had staged repair (46%); of these, 2 have attained completion repair. Mean time to second operation was 4.6 months.

CONCLUSIONS: The use of an initial surgical AP window in carefully selected patients can increase the size of the true PAs and encourage involution of small communicating MAPCAs, making these patients better candidates for eventual intracardiac repair. The procedure should be avoided in patients with high flow or isolated supply MAPCAs or true PAs larger than 2 to 3 mm in diameter, and is not applicable without a true PA central confluence.

**By Invitation*

53. Outcome after Reconstruction of Discontinuous Pulmonary Arteries

Christof Stamm*, Ingeborg Friehs*, David Zurakowski*, ˆžAlbertus M. Scheule, Lennart F. Duebener*, James E. Lock*, John E. Mayer, Pedro J. Del Nido, Richard A. Jonas; Boston, MA *Discussant: Vaughn A. Stames*

OBJECTIVE: Congenital or acquired discontinuity of branch pulmonary arteries (PA) is a complicating feature of several congenital heart defects. PA continuity is mandatory for eventual repair but treatment strategies remain to be defined.

METHODS: Between 1985 and 2000, PA continuity was established in 102 patients with discontinuous central PAs and normal peripheral arborization (median age 2.6 yrs, 5d-35yrs). Patients requiring complex unifocalization procedures were not included. Data were obtained retrospectively.

RESULTS: Diagnoses included TOF/PA (n=39), other forms of PA (n=21), TOF/PS (n=15), tricuspid atresia (n=12), truncus arteriosus (n=5), and others (n=10). Sixty-six patients had 2 ventricles and 36 had a single ventricle. Techniques to connect both PAs included direct PA-PA anastomoses (n=33), tubegraft interposition (n=47), or PA implantation in RV-PA conduits (n=22). At a mean follow-up of 5.4±3 yrs, 57 patients had a biventricular repair completed, 20 had undergone a Fontan operation, and 25 await final repair. In biventricular patients, survival was 85±8% at 5 yrs, and freedom from surgical or interventional PA plasty was 31±11%. At follow-up, mean branch PA Z-scores were -0.5±1.6 (RPA) and -1.4±1.3(LPA). Mean RV/LV pressure ratio was 0.61±0.26 and was >0.75 in 13/58,15/51 had a lung perfusion mismatch of >75/25%, and in 9/58 one branch PA was occluded. Twenty-two patients who underwent primary establishment of antegrade PA flow without prior shunt procedures (median age 0.25 yrs) had comparable survival and reintervention rate with a tendency toward higher PA Z-scores and lower RV/LV pressure ratio. In single ventricle patients 5-yr survival was 93+8% and freedom from PA plasty was 39±9%. 10/19 patients had a lung perfusion mismatch and in 4/31 one branch PA was occluded. Overall, a direct PA-PA anstomosis was associated with better survival (p=0.006). The presence of aortopulmonary collaterals was a risk factor for PA occlusion (p=0.03).

CONCLUSIONS: Good survival can be achieved in patients with PA discontinuity but requires frequent reinterventions. Direct PA-PA anastomoses and control of all collateral vessels may further improve outcome.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

ˆž2000-01 AATS Graham Fellow

**By Invitation*

3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION C-4 - CONGENITAL HEART DISEASE

Room 6D/E, San Diego Convention Center

Moderators: John J. Lamberti; Constantine Mavroudis

54. Arterial Switch Operation For Transposition of the Great Arteries with Single Coronary Artery

ˆžAlbertus M. Scheule, Elizabeth D. Blume*, David Zurakowski*, Christof Stamm*, Richard A. Jonas; Boston, MA

Discussant: Francois LaCour-Gayet

OBJECTIVE: To evaluate the impact of single coronary artery (CA) anatomy on survival and reintervention (RI) after the arterial switch operation (ASO).

METHODS: Retrospective review of 53 patients (pts) with single CA and transposition of the great arteries who underwent ASO at a single institution between January 1983 and July 2000 (Median age at surgery: 10 days). Statistical analyses performed included Kaplan-Meier product-limit method and Cox's multivariable regression.

RESULTS: Thirty-five pts had a single right CA, with the left main CA posterior to the pulmonary artery (PA) in 27. Eighteen pts had a single left CA (16 anterior to the neopulmonary artery). Six out of seven pts who died had a single right CA, all died before 1992. There were four early deaths, all due to coronary malperfusion, all with a single right CA. Seven pts had RI, four because of right ventricular outflow tract obstruction, one for heart transplant, one for mitral valve repair and one pacemaker. Mean follow-up was 6.6 ± 5 yrs. Survival rates (95% confidence interval) were 90% (83-97) at 6 mos, and 86% (78-94) at 1,5, and 10 yrs after ASO, with a higher risk for pts with a single right CA with the left main posterior to the PA compared to all other subtypes ($p=0.03$, log rank test). Patients with a single left CA anterior to the neopulmonary artery had lower freedom from RI than all other types: 70% at 1 yr, 60% at 5 yrs, 45% at 10 yrs, ($p=0.0002$, log-rank test). The Cox model confirmed that this pattern was predictive of a higher monthly risk of RI (hazard ratio = 12.2, $p = 0.01$). Age at surgery, gender, birth weight, presence of a VSD, prior PA banding, cross-clamp time, pump time, length of mechanical ventilation, delayed sternal closure, and position of the great arteries were not associated with survival or freedom from RI in the univariable or multivariable analyses (all $p>0.20$).

CONCLUSIONS: In the current era, ASO with a single CA can be performed safely irrespective of the single coronary anatomy. However, in pts with a single left CA anterior to the neopulmonary artery, the risk of RI is increased.

ˆž2000-01 AATS Graham Fellow

**By Invitation*

55. Biventricular Repair of Transposition of the Great Arteries and Unbalanced Ventricles

ˆžAlain Serraf, Doninique Plot*, Francois Lacour-Gayet, Anita Touchot*, Regine Roussin*, Joy Zoghbi*, Emre Belli*, Jacqueline Bruniaux*, Claude Planche; Le Plessis-Robinson, France

Discussant: Ivan M. Rebeyka

OBJECTIVE: The surgical approach in pts with TGA and unbalanced ventricules remained unanswered. Since the beginning of the ASO program, these pts underwent biventricular repair by ASO and repair of associated lesions if any through either a single or staged surgical procedures. The aim of this retrospective study was to analyze if this approach remains a valid option for these patients.

METHODS: Forty four patients with TGA and unbalanced ventricules received this surgical approach since 1984. Two groups were defined: Groupl: TGA with dominant right ventricle (n=28) and Group II; TGA with dominant left ventricle (n=16). In group I, the median age and weight at ASO were 8.5 days (Ranges: 5-10 days) and 3.1 kg (Ranges: 1.5-3.7). The median end diastolic LV volume, mass and long axis ratio (LAR) were 15 ml/m2 (Ranges: 12-17), 31.5 g/m2 (Ranges: 20-66) and 0.875 (R: 1-0.7). The mitral valve diameter was slightly hypoplastic with a median Z-value of -1.22 (R: -0.3-3.7). In group II, the median age and weight at ASO were 42 days (R: 8 days-15 years) and 3.5 kg (R: 2.8-35). Associated lesions in this group were Coarctation in 9, single (n=12) or multiple (n=4) VSD. The median LAR and tricuspid Z-value were: 0.6 (R: 0.3-0.8) and -0.9 (R: -0.5-3.3). In this group 10 pts had a single stage procedure with fenestrated VSD and/ or ASD patches, 6pts had staged approach.

RESULTS: In group I there was one early death from sepsis after weaning from post operative ECMO. Three pts developed severe pulmonary hypertension one of whom died one year later. All survivors demonstrated already at discharge from hospital equilibrated ventricular size with a median EDLW of 25ml/m2 (R: 21-30). In group II, there were 3 early and no late death. All early deaths occurred in pts without voluntary residuel intracardiac shunts. Median early post operative LAR and tricuspid z-value were: 0.8 (R: 0.7-1) and -0.2 (0.74-1.2).The actuarial survival rate at 5 years was 89 +/-0.5% (70% CL).

CONCLUSIONS: This study demonstrates that the ASO in pts with TGA and unbalanced ventricles remains a good surgical option.

ˆž1993-94 AATS Graham Fellow

**By Invitation*

56. Arterial Switch and Neurodevelopmental Outcome: Full Flow CPB with Limited Circulatory Arrest.

Tom R. Karl, Geoff Ford*, Elaine Kelly*, Roger B. B. Mee, Robert Weintraub,

Christian P. R. Brizard*, Andrew Cochrane*, Suzanne Hall*; Philadelphia, PA;

Melbourne, Australia; Cleveland, OH

Discussant: Richard A. Jonas

OBJECTIVE: Circulatory arrest is a risk factor for unfavorable neurodevelopmental events. Our objective was to investigate a patient cohort operated with full flow CPB (150 ml/kg/min., a-stat, a-blockade, median arrest = 6 minutes, temperature 22°C) as predominant support strategy for neonatal arterial switch operations (TGA, intact ventricular septum).

METHODS: 74 patients and "Best Friend" controls were assessed at 109 (48-166) months with: medical-neurologic evaluation, IQ testing, formal movement scores, and detailed parent-teacher behavioral/social reports. Fetal, neonatal and perioperative data were collated.

RESULTS: Incidence of perioperative seizures was 6.8% (4/5 preopera-tively), and for all perioperative neurologic abnormalities 20%. No perioperative-perinatal factor (including CPB parameters) significantly influenced these incidences (logistic regression). Patients had more neurologic exam findings than controls (p =.002). IQ (WPPSI and WISC 3) was higher in controls (101.9 vs 108.6, p=.0018), both groups having means above Wechsler norms. Fullscale IQ related to paternal education (b = 1.46, p =.008) and perioperative neurologic abnormalities (b = -8.69, p = .013). Patients had higher impairment scores (Movement Assessment Battery) than controls (p = .0004). Parents (Achenbach Child Behavior Checklist) gave higher (better) total social-behavioral competence scores to control (p = .05). Teachers (Teacher Report form) suggested behavioral competence scores to controls (p =.05). Teachers (Teacher Report Form) suggested that patients were more likely to have speech and expressive language problems (p =.02 and .05).

CONCLUSIONS: With the perioperative strategies employed, survivors have at least average intelligence, but cannot be considered neurodevelopmentally normal. The probability of significant impairment is extremely low. Perioperative neurologic problems had predictive value for late IQ. Continued application of the full flow CPB strategy is justified.

**By Invitation*

57. Surgical Management of the Scimitar Syndrome: An Alternative Approach

John W. Brown, Christopher Edwards, Andrew C. Fiore, Paul N. Uhlig*, Mark

Ruzmetov*, Yuji Okada", Mark W. Turrentine*; Indianapolis, IN; Wichita, KS;

St.Louis, MO

Discussant: Thomas L. Spray

OBJECTIVE: The scimitar syndrome (SS) is a congenital anomaly that consists mainly of total or partial anomalous venous drainage of the right lung to the inferior vena cava (IVC). Surgical approaches to SS have varied depending on the anatomical as well as the pathological features presented in each case. The aim this study is to present an alternative approach to surgical correction of the SS.

METHODS: Nine patients with SS were seen in the period between 1990 and 2000. There was 1 male and 8 female. Two different types of scimitar vein was recognized: simple classical vein running from the middle of the right lung to the IVC below the diaphragm (5 patients), and double arched vein in the upper and lower lung zone, with ample drainage into the left atrium and inferior caval vein (4 patients). All of the patients underwent to operation involved direct anastomosis of the scimitar vein to the left atrium via right lateral thoracotomy without the aid cardiopulmonary bypass. Three patients demonstrated SS in association with pulmonary sequstration of the right lower lobe. In these cases, treatment involved surgical resection of the right lower lobe. In all cases the atrial septum appeared intact.

RESULTS: The mean age at the operation was 11.5 years (range 7 month to 42 years). All patients survived repair and remain asymptomatic during a follow-up of 12 to 108 months (mean 48.3 months). There were no late death or reoperation. All patients have Echocardiography evidence of a patient anastomosis without obstructions.

CONCLUSIONS: Based on our experience, we feel that the best approach to surgical correction of SS is the direct anastomosis of the scimitar vein to the posterior aspect of the left atrium via a right thoracotomy without to use of cardiopulmonary bypass. If the atrial septal defect is to be surgically corrected, cardiopulmonary bypass is necessary, and the procedure may be performed also via a right thoracotomy. This represents a unique and viable alternative to the previously described tunnel approaches using cardiopulmonary bypass.

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

Room 6A-C, San Diego Convention Center

7:00 p.m. ATTENDEE RECEPTION - NAUTICAL PORTS OF CALL

San Diego Convention Center Outdoor Terrace
(Separate Subscription)

**By Invitation*

WEDNESDAY MORNING, MAY 9, 2001

7:00 a.m. EMERGING TECHNOLOGIES AND TECHNIQUES FORUM

Room 6A-C, San Diego Convention Center

Moderators: Ralph T. Damiano; Patrick M. McCarthy

T1 Development of Vacuum Assisted Lung Reducer for Lung Volume Reduction Surgery in Emphysema

Blanding U. Jones*, †Xavier Gonzalez*, Matthew Brenner*, Tanya L. Burney*, Linda D. Martino*, H. P. Ha*, Arthur Gelb*, Kathy Osann*, Jeffrey C. Milliken*; Orange, CA; Redmond, WA

OBJECTIVE: Lung volume reduction surgery(LVRS) is most commonly performed with resection by linear mechanical stapling. Other methods of LVRS include stapling with tissue or material buttresses, laser ablation, and suture ligation. Air leak is the most frequent significant complication of LVRS. The purpose of this study was to develop a vacuum assisted bio-compatible lung reducer device that would be simple, rapid and effective, with less complications than current methods

METHODS: An implantable, bio-compatible, silicon elastomer sleeve prototype device was developed. The appliance was designed to mechanically reduce the volume of targeted lobes and prevent their re-expansion. A vacuum column device was developed to deploy the appliance. Emphysema was induced with repeated nebulizations of porcine elastase in 10 NZW rabbits. A median sternotomy was performed. The sleeves were deployed onto selected lobes then fixed in position by a purse string suture proximally and a "U" stitch apically.

RESULTS: Selective LVRS was achieved by deployment of this device. During the peri-operative time period, there were no air leaks, no hemorrhage, and no deaths. At sacrifice, there was no infection or abscess formation, however, there was moderate formation of granulation tissue and some mild pleural effusions.

CONCLUSIONS: This new, surgically deployable appliance may be effective in achieving therapeutic LVRS for emphysema and may eliminate the development of peri-operative and late post-operative air leaks. Further studies are needed to evaluate the long- term safety and physiologic effectiveness of this device for LVRS, as well as investigate other potential clinical applications such as rapid sealing of air leaks.

†*Author has a relationship with Spiration, Inc.*

**By Invitation*

T2. Over 3000 Days of Continuous Flow Support with the DeBakey VAD™: Clinical Trial Update

†George P. Noon*, Roland Hetzer, Ernst Wolner, Marko Turina, Jean-Noel Fabiani*, Hans H. Scheld*, Ettore Vitali*, Herman Reichenspurner*, Bruno Reichart*, †Deborah L. Morley*, †Michael E. Debakey; Berlin, Germany; Vienna, Austria; Zurich, Switzerland; Paris, France; Muenster, Germany; Milan, Italy; Munich, Germany; Houston, TX

OBJECTIVE: TheDeBakey VAD™ Clinical Trial is being conducted to demonstrate the safety and effectiveness of the DeBakey VAD™ as a left ventricular assist device in patients with end-stage heart failure.

METHODS: A multicentre trial in Europe and an IDE approved trial in the US enroll patients with Class IV heart failure and profoundly abnormal hemodynamics or the need for extraordinary inotropic support. Patients must be transplant candidates. After implant, anticoagulation is achieved with warfarin and platelet inhibitors. Patients are monitored for pump function, hemodynamics, clinical status and any complications.

RESULTS: Fifty-eight patients have been implanted with the DeBakey VAD™, 16 of the 58 patients are ongoing. The average age of the 50 males and 8 females implanted is 47 yrs. The average BSA is 1.8 m^2 (range 1.5 to 2.5 m^2. Forty-five percent of patients had idiopathic and 43% had ischemic cardiomyopathies. Sixteen patients have been successfully bridged to transplant, with the outcome of the ongoing 15 patients yet to be determined. Twenty patients died prior to transplant of causes not directly related to the device, the most notable cause of death being multiorgan failure. One patient was bridged to recovery and 6 patients experienced an event resulting in device exchange or ligation of the outflow graft. Patients maintained a pump flow between 4-6 L/min throughout support. Complications observed include: late (>14 days postimplant)bleeding(55%)related to anticoagulation; hemolysis (21%)which was related to transfusions, transient, seen > 16 days post-implant and not occurring at all sites; and renal failure

(18%)noted in patients with mulitorgan failure. Only 1 device related infection (exit site)has occurred in 3184+ days of cumlative support.

CONCLUSIONS: The DeBakey VAD™ has provided adequate left ventricular support as a bridge to transplant in patients with end-stage heart failure. Continuous flow (>200 days) has not resulted in neurologic or end organ dysfunction and demonstrates pulsatility with native heart funtion. While some complications have occurred, the risk-benefit ratio remains in favor of implantation of the DeBakey VAD™.

†*Author has a relationship with MicroMed Technology*

**By Invitation*

T3. Percutaneous Transluminal Replacement of the Aortic Valve: An Experimental Study

Georg Lutter*, Ulrich Matern*, Georg Berg*, Kim Ameer*, Daniela

Kuklinski*, Tim Attmann*, Alexander Thiem*, Kerstin Schlegel*, Friedhelm

Beyersdorf; Freiburg, Germany

OBJECTIVE: In 1996, we constructed a new biological heart valve in combination with an artificial stent (valve stent) designed for implantation by transluminal catheter technique without thoracotomy. This abstract describes the new valve stent, *in vitro* measurements, the implantation technique *in vivo.*

METHODS: The new foldable heart valve was prepared by mounting a porcine aortic valve, taken from 62-75kg slaughtered pigs (German Lan-drace), into a self-expandable metallic stent (Memory-, Nitinol-). The aortic valve was carefully dissected and cleaned manually. The diameter, thickness and height of the cleaned annulus depended on the weight and height of the recipient pig. Before implantation, the valve stent was tested in an *in vitro* -hydrodynamic pulsatile-flow testing to exclude any stenosis or mild to moderate regurgitation. Different techniques for the endovascular heart valve ablation were also tested.

RESULTS: The introducer-device of the stent valve had an outer diameter of 21 Fr and was inserted via the common left iliac artery exposed retroperitoneally. The valve stents used for implantation did not show any signs of stenosis or regurgitation *in vitro.* Furthermore, the analysis of these valves *in vivo* (n=7) revealed neither greater stenosis (controlled by millar catheter measurement; dp_{max}: 5.61±3.42mmHg) nor regurgitation (trans-esophageal echo control (TEE), trivial regurgitation in one) in this prolonged acute model (observation period: 3-5hours). Visualisation and positioning was assessed by TEE and CT scan. Sub- (n=l) and supracoronary (n=6) implantation was successfully performed in the anesthetized pigs. Anatomic evaluation which was finally performed demonstrated the correct positions of the valve stents. Holmium:YAG- and CO_2-laser ablation tests indicated a moderate embolic rate of debris.

CONCLUSIONS: This preliminary study demonstrates that aortic valve stents can be successfully implanted by transluminal catheter technique, without the need of opening the chest.

**By Invitation*

T4. Beating Heart Mitral Valve Repair: Initial Results

Stephen W. Downing*, Timothy B. Gilbert*, William W. Herzog, Joseph S. Mclaughlin; Baltimore, MD

OBJECTIVE: It is our hypothesis that mitral valve repair can be performed on the beating heart without cardiopulmonary bypass. As a first phase, we tested the feasibility of suturing the anterior and posterior mitral leaflets under echocardiographic guidance through a pressurized atrial port, mimicking a bow-tie valve repair.

METHODS: In a water bath model, imaging approaches, port design, and suturing techniques were tested. Then, in 6 pigs, the left atrium was cannu-lated with a customized 15mm port via a left thoracotomy. Arterial blood was shunted into the atrium via the port to maintain a positive pressure at all times to minimize air induction. Blood pressure and cardiac rhythm were monitored. A multiplane transesophageal echo (TEE) probe was evaluated in the intraesophageal, epicardial and intracardiac positions. Utilizing a commercial suturing device, sutures were placed through the anterior and posterior mitral leaflets under echo guidance. The animals were sacrificed, and accuracy evaluated by measuring the distance from the target area in the center of the anterior and posterior mitral leaflets. Air induction was monitored by echo and graded as minimal to severe.

RESULTS: There was no hemodynamic instability or significant arryth-mia. The most effective imaging plane was a short axis view utilizing the TEE probe inside of the pericardium at the heart base. Air introduction was minimal in 2 animals, mild in 3, and moderate in 1. Nine of 12 sutures were successfully placed, with a mean error of .8 ± .5 cm. Technical difficulties included off -axis imaging, acoustic shadowing from the device, large instrument size relative to valve orifice, and snaring in chordae.

CONCLUSIONS: Rudimentary beating heart mitral valve surgery is possible. Utilizing this paradigm, with improved instrumentation, the performance of beating-heart mitral valve repairs including annuloplasty, leaflet plication, and chordal replacement will be feasible.

**By Invitation*

T5. The Cryosurgical Maze Procedure: A. Simplified Technique for Atrial Fibrillation

Niv Ad*, Terry Palazzo*, James L. Cox; Washington, DC

OBJECTIVE(s): The Standard Maze Procedure (SMP) has proven to be a safe and effective treatment for atrial fibrillation (AF). However, the major deterrent to its wide application has been its complexity. In order to simplify the procedure we developed the cryosurgical Maze Procedure (CMP) in which the atrial incisions of the SMP are replaced by linear cryolesions. The present study compares the safety and efficacy of this new CMP with that of the Standard Maze Procedure.

METHODS: A total of 362 patients underwent The Maze procedure for the treatment of atrial fibrillation, with 335 having some type of the SMP and 27 having the CMP. Of the 27 patients having the CMP, 21 patients had the CMP performed as their only operation, four patients had the CMP plus mitral valve repair and two patients had the CMP plus CABG. Three patients had the

CMP performed by minimally-invasive techniques and two patients underwent the entire procedure without the aid of car-diopulmonary bypass.

RESULTS: The mean aortic cross-clamp time was dramatically less with the CMP than with the SMP primarily because in 12 of 27 patients having the CMP, the aorta was not cross-clamped at all. Since there were few if any atrial suture lines following the CMP, the procedure was technically much easier to perform than the SMP. There were no early or late deaths in the 27 patients undergoing the CMP. One patient had a prolonged hospital stay following a minimally-invasive CMP due to respiratory problems related to preoperative treatment with amiodarone. Atrial fibrillation was successfully ablated in 26 of 27 patients (96%) undergoing the CMP with a mean follow-up of 5.1±3 months. Atrial contraction was documented to be restored in both the right and left atria in all patients.

CONCLUSIONS: The CMP is technically Less demanding than the SMP and is just as safe and effective in abolishing AF. Because of the speed, safety and simplicity of this new cryosurgical technique, the indications for applying the concepts of the Maze procedure for the treatment of refractory AF should be liberalized, especially when the atrial fibrillation is present in patients who require mitral valve surgery.

**By Invitation*

8:00 a.m. GENERAL THORACIC SURGERY FORUM SESSION

Room 1A/B, San Diego Convention Center

Moderators: G. Alexander Patterson; David H. Harpole, Jr.

F1. Chemosensitization of NF-KB-Deficient Non-Small Cell Lung Cancer Cells is Mediated by Increased Cytochrome c Release and Caspase Activation

David R. Jones*, R. Michael Broad*, Sarah J. Parsons*, Marty W. Mayo*;

Charlottesville, VA

OBJECTIVE: We have previously shown that chemotherapy-induced NF-kB activation is a novel mechanism of chemoresistance in non-small cell lung cancer (NSCLC). While inhibition of NF-kB sensitizes NSCLC cells to chemotherapy-mediated cell death, the cell-signaling mechanisms regulating this process are unknown. The purpose of this study was to determine if the loss of NF-kB in NSCLC cells was pro-apoptotic secondary to increased mitochondrial cytochrome c release and caspase activation.

METHODS: H157 NSCLC cells were stably transfected with the dominant-negative form of NF-kB, which inhibits all NF-kB-dependent gene transcription, (named H157I) or a vector control (H157V). These cells were treated with gemcitabine (ImM) over time and DNA fragmentation determined by ELISA. Caspase-3, -6, -7, and -9 activity was determined in cytoplasmic extracts by flourometric analysis of cleavage of a fluorophore from the appropriate tetrapeptide. Mitochondrial-free extracts of similarly treated cells were subjected to SDS-PAGE analysis with an antibody to cytochrome c and then quantitated using densitometry. To determine if cell death was a caspase-dependent process, the caspase inhibitor, Boc-D, was added to the H157I cells and apoptosis assessed. All experiments were performed in triplicate and the significance of the data determined by ANOVA.

RESULTS: NSCLC cells lacking functional NF-kB (H157I)were significantly more apoptotic following treatment with gemcitabine than vector controls (H157V)($p < 0.05$). H157I cells had significantly more activation of caspases-3 and -9 than H157V cells ($p < 0.05$). There was no significant caspase-6 or -7 activation. Inhibition of caspase activity rescued cell death in H157I cells . There was significantly more cytochrome c release post-chemotherapy in the H157I cells ($p < 0.05$).

CONCLUSIONS: Chemosensitization by inhibition of NF-kB in NSCLC cells occurs via an increased cytochrome c release and caspase-3 and -9 activation. This suggests a mitochondrial-mediated death process. NF-kB and/or its inhibition may be a novel pharmaceutical target in patients with NSCLC.

**By Invitation*

F2. Hyperbaric Oxygen as a Chemotherapy Adjuvant in Lung Tumors

Frank A. Baciewicz, Paul M. Petre*, Richard J. Spears*; Detroit, MI

OBJECTIVE: Catheter-based delivery of Aqueous Oxygen (AO) has been recently shown to allow the local delivery of hyperoxemic blood at pO2 s in the hyperbaric range (>1000mm Hg). The purpose of this study is to demonstrate the utility of high oxygen levels as an adjuvant for lung tumors chemotherapy in a rat model.

METHODS: MCA-2 cells (rat sarcoma cell-line) were treated with increasing concentrations of Doxorubicin(0.2m to 5m). A dose-dependent toxic-ity curve at 12 hours after treatment was obtained. The cells were also treated with Hyperbaric Oxygen (HBO, 40psi for 1.5 hours); the Doxoru-bicin solution was added to the culture medium immediately after HBO, and a second curve was obtained. For the in vivo study, 8 Sprague Dawley rats were injected with 10^6 MCA-2 cells in 1ml saline in the jugular vein, and the lung tumors were allowed to maturate for a 10-day period. At that time, the rats were divided into 4 groups: No Treatment, 2 mg/kg Doxorubicin, HBO + Doxorubicin, and HBO only. The high levels of oxygen obtained were achievable by either of two methods: 1). AO infused directly in the pulmonary artery by a trans- ventricular catheter which allowed a solution with 1ml O2/g to be given at a rate of 0.03 g per 1ml blood flow (arterial pO2 level in the 700-1000 mm Hg range). 2). 25 psi of oxygen for 1.5 hours in a pressure chamber. Seven days after the treatment, the total number of nodules in both lungs was counted and the lung weight determined.

RESULTS: MCA-2 cells shown a significantly greater cytolysis ($p<0.01$) if treated with Hyperbaric Oxygen and Doxorubicin, in comparison with the cells treated with Doxorubicin only. The high levels of oxygen in conjunction with Doxorubicin decreased significantly the lung weight and the number of lung metastasis in comparison with animals treated with Doxorubicin only, or with untreated animals. Hyperoxemia did not enhance tumor growth by itself.

CONCLUSIONS: High levels of oxygen given before chemotherapy enhanced the tumor cell lysis, both in cell culture and in the rat model. Catheter infusion of hyperoxemic blood (method developed by our group) can be a promising technique in achieving local high oxygen levels as a chemotherapy adjuvant in solid tumors.

**By Invitation*

F3. Antisense Therapy for Malignant Mesothelioma Utilizing Antisense Oligonucleotides Targeting the Bclxl Gene Product

W. Roy Smythe*, Imran T. Mohiuddin*, Xiaobo X. Cao*; Houston, TX

OBJECTIVE: Malignant mesothelioma is resistant to all conventional therapies. The bcl-2 gene family is a major determinant of apoptotic ho-meostasis. Mesothelioma cell lines and tumors rarely express significant anti-apoptotic (AAP) bcl-2 protein, but routinely express AAP bclXL. In addition, expression of the pro-apoptotic proteins (PAP), bax and bak are common. We have previously shown that pharmacologic inhibition of bclXL expression in mesothelioma can lead to apoptosis, and sought to determine whether or not antisense oligonucleotides (ASO) directed at the bclXL mRNA would engender apoptotic cell death, possibly via a forced imbalance of bcl-2 family protein expression.

METHODS: Mesothelioma cell lines REN (epithelial) and I-45 (sarcoma-tous) were utilized. Cells exposed to phosphorothioate-modified bclXL ASO (0-100 nM) directed at mRN A start sequence. Untreated cells and bclXL sense oligonucleotides (SO) were controls. Dose-response survival was measured by XTT assay. REN and 1-45 were also exposed to unmodified ASO and SO (0 -1000 nM) combined with a liposomal delivery system. Dose-response survival was again measured with XTT assay. Apoptosis was evaluated with a morphologic assay.

RESULTS: Significant cellular killing was achieved with phosphorothioate-modified ASO when compared to untreated and SO treated cells (p = 0074). At 100 nM, 80% of the modified ASO-treated cells were killed vs. 20% of the ASO group. Morphologic evidence of apoptosis was identified. Using the unmodified ASO, significant cellular killing and morphologic apoptosis was again noted (1000 nM dose: REN 61.4%, I-45 48% cellular death). Non-specific liposomal effect narrowed the therapeutic margin between ASO and SO, but the difference was still significant in REN (p = .05).

CONCLUSIONS: In summary, we have demonstrated that ASO targeting of the critical anti-apoptotic gene, bclXL, can engender apoptotic cellular death. In vivo studies and radiation sensitization evaluation are ongoing. The use of bclXL ASO may be clinically useful for treatment of this difficult disease.

**By Invitation*

F4. Apo2L/TRAIL-lnduced Apoptosis in NSCLC Cell Lines is Enhanced by Chemotherapeutic Agents but not Correlated to the Expression Level of c-FLIP

Steffen Frese*, Thomas Brunner*, Mathias Gugger*, Aima Uduehi*, Ralph A.

Schmid*; Berne, Switzerland

OBJECTIVE: Apo2L/TRAIL (TNF-related apoptosis-inducing ligand) is a potential anticancer drug which promotes apoptosis specifically in tumor cells. Because not all cancer cells are susceptible to Apo2L/TRAIL the aim of our study was to determine if NSCLC cells can be sensitized by chemotherapeutic agents for Apo2L/TRAIL-induced apoptosis. In addition, endogenous expression level of the caspase-inhibiting protein c-FLIP (cellular FLICE-inhibitory protein) were measured in order to investigate partial resistance to Apo2L/TRAIL.

METHODS: Six human lung cancer cell lines (A549, NCI-H358, Calul, Calu6, SkMesl, SkLul) were incubated with soluble Apo2L/TRAIL and two different concentrations of cisplatin, paclitaxel, doxorubicin, 5-fluo-rouracil, and camptothecin. After 24 hours the rate of apoptosis was measured by AnnexinV/PI staining followed by FACScan analysis. Expression level of c-FLIP in cell lines and lung cancer biopsies was determined by Western Blot.

RESULTS: Treatment of lung cancer cells with Apo2L/TRAIL alone resulted in apoptotic cell death in four cell lines ($p<0.001$). Combined treatment of Apo2L/TRAIL with chemotherapeutic agents enhanced the rate of apoptosis significantly. Statistical analysis revealed a syergistc effect of Apo2L/TRAIL in combination with 1.8mM camptothecin, or 100mM cisplatin, each in four of the six cell lines ($p<0.002$). Western Blot analysis showed that sensitization to Apo2L/TRAIL does not correlate with the expression of c-FLIP. Furthermore, no increased protein levels could be found in NSCLC of 12 patients compared to normal lung tissue.

CONCLUSIONS: Apo2L/TRAIL-induced apoptosis in NSCLC cell lines is significantly enhanced by chemotherapeutic agents. Resistance and sensitization to Apo2L/TRAIL is not correlated to the endogenous expression level of c-FLIP, implicating that in NSCLC other mechanism are responsible for inhibition of the Apo2L/TRAIL pathway. Even the molecular mechanism remains unclear our data suggest that the combination of Apo2L/TRAIL with chemotherapy may be a promising treatment modality for NSCLC.

**By Invitation*

F5. Recipient Intramuscular Cotransfection of Naked Plasmid TGFp, and IL-10 Ameliorates Lung Graft Ischemia-Reperfusion Injury.

Niccolo Daddi*, Takashi Suda*, Franco D'Ovidio*, Tsutomu Tagawa*,

Benjamin D. Kozower*, Same: A. Kanaan*, Nelson S. Yew*, Kathleen

Grapperhaus*, T. Mohanakumar*, G. A. Patterson; Saint Louis, MO; Bologna,

Italy; Framingham, MA

OBJECTIVE: Multiple gene transfer may permit modulation of concurrent biochemical pathways involved in lung graft ischemia-reperfusion injury. We sought to determine whether recipient intramuscular (IM) naked plasmid co-transfection of Transforming Growth Factor (TGF-b,) and Interleukin 10 (IL-10) would result in a synergistic amelioration of lung graft ischemia-reperfusion injury.

METHODS: Forty-eight hours prior to transplant six groups (n=6 each) of inbred F344 recipient rats received IM injection of: naked plasmid encoding CAT, CAT+bGal, $TGFb_1$,IL-10 or $TGFT_1$+IL-10 or control (no treatment). Donor lungs were flushed with LPDG and stored for 18 hrs at 4°C prior to transplantation. Twenty-four hours later, graft function was assessed immediately prior to sacrifice. Graft wet to dry ratio (W/D) and myeloperoxidase (MPO) activity were measured.

RESULTS: The table reports the median and range of PaO_2mmHg, W/D and MPO. Statistical analysis (one-way analysis of variance) was after square root correction of the data.

CONCLUSIONS: Recipient intramuscular naked plasmid co-transfection of TGF-b_1 and IL-10 provide a synergistic effect to reduce lung reperfusion injury following prolonged ischemia.

	GROUP	PaO_2 mmHg	W/D	MPO ^†OD/mg/min
I	***No Treatment***	**86.3** (39.4-228)	**87** (6.1-11.6)	**0.57** (0.23-0.83)
II	***CAT***	**54.5** (31.1-142.5)	**7.3** (6.5-10.8)	**0.60** (-0.20-1.78)
III	***CAT + Î²Gal***	**52.8** (36.1-151.5)	**8.7** (6.3-10.6)	**0.62** (0.20-0.98)
IV	***TGFÎ²1***	**130** (76.4-193.2)	***5.9** (4.9-6.7)	**0.47** (0.26-0.65)
V	***IL-10***	***150.7** (104.8-346	***6.1** (4.4-7.1)	**0.31** (0.0003-0.66)
VI	***TGFÎ²1+IL-10***	****298.1** (162.4-524)	***5.5** (4.9-6.2)	***0.26** (0.04-0.44)

*p‰¤0.05 vs Group I, II and III

**p<005 vs Group I, II, III, IV and V

**By Invitation*

F6. Induction of Apoptosis in Malignant Pleura! Mesothelioma Cells by Activation of the Fas (APO-1/CD95) Death Signal Pathway.

John H. Stewart*, Dao M. Nguyen*, G. Aaron Chen*, David S. Schrump*;

Bethesda, MD

OBJECTIVE: The Fas death signaling pathway has been implicated in antitumor immunity. This project was undertaken to evaluate the cytotoxic effects of Fas receptor activation by recombinant soluble Fas ligand (sFasL) or FasL-bearing lymphokine-activated killer (LAK) cells in malignant pleural mesotheliomas.

METHODS: Fas expression in primary normal human cells and 5 MPM cell lines was quantified by flow cytometry. Components of the Fas-mediated apoptotic pathway were evaluated by western blot techniques. sFasL-mediated cytotoxicity, and apoptosis were evaluated by MTT, and TUNEL assays respectively. LAK-mediated cytolysis of MPM cells was evaluated by chromium release assays. The FasL-dependent component was examined following inhibition of perforin function under calcium-free conditions, and blocking residual cytotoxicity with sFas (100 ng/ml).

RESULTS: Three of five MPM cell lines exhibited high level Fas expression, and 2 of these 3 lines were susceptible to sFasL-mediated cytotoxicity (sFasL IC_{50} <10 ng/ml). The sFasL-refractory H2052 cell line, expressing high levels of bcl-2, was sensitized to sFasL cytotoxicity by pretreat-ment with cisplatin or the bcl-2 antagonist Tetracarcin A (reduction of sFasL IC_{50} from µ to 4.17±0.14 ng/ml and 5.85±0.09 ng/ml respectively). The contribution of Fas ligand during LAK mediated cytolysis was readily detectable in Fas-ligand sensitive H2373 cells. In contrast, LAK induced minimal cytolysis in H2052 cells. However, following cisplatin treatment, H2052 cells were lysed via perforin as well as Fas ligand pathways.

CONCLUSIONS: Apoptosis mediated by chemotherapeutic agents or immune effector cells can be enhanced in mesotheliomas via restoration of a functional Fas death pathway. These preclinical data support further evaluation of strategies to enhance Fas-mediated apoptosis as a means to augment efficacy of chemotherapy or immunotherapy in MPM patients.

**By Invitation*

F7. Autologous Tissue Engineered Trachea Using Sheep Nasal Chondrocytes

Koji Kojima*, Lawrence J. Bonassar*, Amit K. Roy*, Charles A. Vacanti*,

Joaquin Cortiella*; Worcester, MA

OBJECTIVE(s): Extensive tracheal resection is often required in patients with malignant and benign disease. Several approaches for tracheal replacement have been attempted with limited success. This study was designed to evaluate the ability of autologous tissue engineered cartilage shaped in a helix to form the structural component of a functional tracheal replacement.

METHODS: A septoplasty was performed on 6 sheep (1-year-old, 32Kg weight each). Chondrocytes were harvested from sheep nasal septum. Cells were suspended in Ham's F-12 culture media, concentrated to density of 50 x 10^6 / ml and seeded onto a 100mm x 10mm x 2mm matrix of non-woven mesh of PGA fibers. Cell-polymer constructs were incubated in vitro for 2 week and then wrapped around a 20mm diameter x 50mm length helical template and implanted into subcutaneous pocket for in the neck. After 6 weeks, the coil structure of tissue engineered trachea was harvested and placed in a 50mm defect in the trachea of the same sheep.

RESULTS: All six sheep were able to survive with minimum respiratory symptoms. Bronchoscopy showed minimal tracheal stenosis. Histologi-cal evaluations of engineered trachea using H&E, safranin-O stains, showed the presence of mature cartilage. Proteoglycan and hydroxypro-line contents were similar to native cartilage levels. The morphology and histology of tissue engineered trachea resembled those of sheep trachea.

CONCLUSIONS: This study demonstrated the ability to create a tissue engineered trachea from autologous Chondrocytes obtained via simple septoplasty. This approach provides a benefit to individuals needing trachea resection.

**By Invitation*

F8 Replication-Competent Herpes Virus NV1020 for Direct Treatment of Pleural Cancer in a Rat Model

Michael I. Ebright*, Sandeep Malhotra", Jonathan S. Zager*, Keith A.

Delman*, Tracey L. Weigel*, Paul Johnson*, Yuman Fong*; New York, NY,

San Diego, CA

OBJECTIVE: Innovative treatments are needed for metastatic disease involving the pleura. NV1020 is a novel, multi-mutated, replication-restricted herpes-simplex virus (HSV) under investigation for its ability to selectively kill tumors by direct cell lysis. This study examines NV1020 in a rat model of pleural-based lung cancer.

METHODS: Cytotoxicity was evaluated *in vitro* by exposure of the human non-small cell lung cancer cell line A549. NV1020 was also tested in an *in vivo* pleural-based cancer model established by injecting 1x10^7 A549 cells into the thoracic cavity of nude rats. Intrapleural treatments

($1x10^7$ viral particles) were given 3h or 3d after tumor injection to model treatment of microscopic or macroscopic disease (n=9/group). Tumor was assessed at 5 weeks.

RESULTS: *In vitro,* at multiplicities of infection (viral particles/tumor cell) of 0.01, 0.1, and 1.0, cell kill of A549 by NV1020 was 66%, 90%, and 97%, respectively, at 7 days post-infection. Intrapleural treatment was also effective when given at 3h ($p<0.001$)(Figure) and 3d ($p<0.05$).

CONCLUSIONS: NV1020 is not only highly cytotoxic to the human lung cancer line A549 *in vitro,* but can be delivered in a clinically relevant fashion to effectively treat pleural-based tumor *in vivo.*

**By Invitation*

F9. Adenosine A2A Receptor Activation Decreases the Severity of Lung Transplant Reperfusion Injury Following High Flow Reperfusion

Steven M. Fiser*, †Curtis G. Tribble, Aditya K. Kaza*, Stewart M. Long*,

David C. Cassada*, Andrew J. Matiskoff*, John A. Kern*, †Irving L. Kron;

Charlottesville, VA

OBJECTIVE: High pulmonary artery flow rates can result in severe reperfusion injury following lung transplantation. Our hypothesis was that selective activation of the adenosine A_{2A} receptor, using a highly specific analogue (DWH), would inhibit leukocyte activation and decrease reperfusion injury following high flow reperfusion.

METHODS: Using our isolated, ventilated, blood perfused rabbit lung model, all groups (n=8/group) underwent lung harvest, 4 hr cold storage, and blood reperfusion for 30 minutes. Measurements of pulmonary artery pressure (PAP, mmHg), arterial oxygenation (PO2, mmHg), peak inspiratory pressure and wet to dry weight ratio (WTD) were obtained. Groups I (High Flow) and II (High Flow DWH) underwent reperfusion at 120 cc/min for 30 minutes. Groups III (Controlled High Flow) and IV (Controlled High Flow DWH) underwent controlled reperfusion using an initial reperfusion rate of 60 cc/min for the first 5 minutes, followed by a rate of 120 cc/min for 25 minutes. During reperfusion, groups II and IV received DWH at 4 ug/min.

RESULTS: DWH significantly improved lung physiologic measurements under both high flow (group I vs II) and controlled high flow (Group III vs IV) conditions (Table 1).

CONCLUSIONS: Inhibition of circulating leukocytes using the adenosine A_{2A} receptor analogue DWH significantly decreases reperfusion injury in the setting of both high flow and controlled high flow reperfusion.

	‡High Flow	**High Flow DWH**		**‡Controlled Flow High**	**Controlled High Flow DWH**	
PO2	41.0±4.1*	73.3±7.8*	*p=0.002	87.0±9.7†	123±7.0†	†p‰¤0.001
PAP	80.1±4.4—Š	66.0±2.6—Š	—Šp=0.01	62.9±2.8□□	50.3±2.3□□	□□p=0.003
WTD	13.7±0.6™¦	12.3±0.7™¦	™¦p=0.02	6.8±0.4□□	5.9±0.5□□	□□p=N.S.

‡Additionally, PO2 (p‰¤0.001), PAP (p=0.004) and WTD (p=0.01) were significantly improved in the Controlled High Flow Group compared to the High Flow Group

†*Author has a relationship with ATL*

**By Invitation*

F10. A Respiratory Gas Exchange Catheter: In Vitro and In Vivo Tests in Large Animals

Brack G. Hattlei, Joseph Golob*, Heide Russian*, Michael F. Lann*, Thomas L. Merrill*, Laura W. Lund*, Brian Frankowski*, William Federspiel*;

Pittsburgh, PA

OBJECTIVE: Acute respiratory failure is associated with a mortality of 40-50% in spite of advanced ventilator support and ECMO. To improve survival, a respiratory gas exchange catheter (the HC) has been developed as an oxygenator and CO2 removal device for placement in the vena cava and right atrium of patients with severe respiratory failure.

METHODS: Differing from a previously clinically-tested intravenous gas exchange device (i.e. IVOX), it incorporates a small pulsating balloon surrounded by hollow fibers. The pulsating balloon redirects blood toward the fibers, enhances red cell contact with the membrane, and significantly improves gas exchange so that smaller catheter devices are still efficient upon insertion and can be inserted through the femoral vein.

RESULTS: Using ex-vivo mock vena cavas and comparing the HC with the IVOX reveals up to threefold increases in O2 and CO2 exchange. In vivo performance in calves has shown O2 transfer rates of 336 mL/minute/ m2 and CO2 removal rates of 402 mL/minute/m2. Devices n=8 with layers of thrombo-resistant heparin bonded coating have been tested up to five days in calves with gas exchange rates maintained over this time interval. Plasma-free hemoglobin levels at the end of five days have been 4.8 ± 3.2 mg%. Hemodynamic measurements have shown no changes over the experimental intervals. Autopsies show no end-organ damage. The device linearly increases its CO2 output with progressive hypercapnea predicting its ability to meet tidal volume reduction in the therapy of respiratory failure.

CONCLUSIONS: Progress has been made toward developing an easily inserted intravenous gas exchange catheter to provide temporary pulmonary support for patients in acute respiratory failure.

**By Invitation*

8:00 a.m. ADULT CARDIAC SURGERY FORUM SESSION

Room 6A-C, San Diego Convention Center

Moderators: Edward D. Verier; Pedro T. Del Nido

F11. The Insulin Cardioplegia Trial: Reaching the Limits of Myocardial Protection for CABG.

Vivek Rao*, George T. Christakis, Richard D. Weisel, Joan Ivanov*, Michael A. Borger*, Gideon Cohen*; Toronto and North York, ON, Canada

OBJECTIVE: Previous reports suggest insulin (INS)enhanced cardiople-gia (CPG) improves metabolic and functional recovery following CABG. We performed a large prospective, randomized trial to assess the clinical impact of this intervention in high risk pts undergoing CABG.

METHODS: Pts requiring isolated CABG for unstable angina were randomized to 8:1 blood CPG enhanced with INS (n=557) or the non-active vehicle solution (placebo, n=570). The route and temperature of CPG infusion was determined by surgeon preference. All persons with direct patient contact were blinded to treatment group. The study was stratified for surgeon and institution and was designed to have 80% power to detect a 25% treatment effect for the primary composite outcome of low output syndrome (LOS: prolonged inotropic or IABP support) ± enzymatic MI.

RESULTS:

Outcome	Insulin	Placebo	p-value
LOS	10%	10%	0.9
Enzymatic MI	21%	19%	0.4
LOS±MI	30%	26%	0.2
Temp. Pacing	31%	39%	**0.005**
Hypoglycemia	5%	3%	**0.05**
ICU Stay	40±77 hrs	30±19hrs	0.1
PostOp Stay	8±13 days	6±3 days	0.1
Death	2.2%	2.3%	0.6

CONCLUSIONS: Despite previously encouraging results from smaller non-randomized studies, this large prospective randomized trial failed to demonstrate any clinically meaningful benefits of INS cardioplegia in high risk pts undergoing CABG. The contemporary results of surgery are now more likely to be influenced by technical factors rather than the method of myocardial protection, especially in pts undergoing isolated CABG.

**By Invitation*

F12. Tissue Engineering with Genetically Modified Autologous Endothelial Progenitor Cells Isolated from Peripheral Blood

Afshin Ehsan*, Daniel P. Griese*, Michael J. Mann*, Luis G. Melo*, Lunan Zhang*, Richard E. Pratt*, Richard C. Mulligan*; Victor J. Dzau*, Boston, MA

OBJECTIVE: Endothelial progenitor cells have been shown to originate from adult bone marrow, however, efforts at autologous endothelial cell harvest have been plagued by cumbersome, invasive methods. We therefore developed a highly efficient yet technically simplified method of endothelial lineage cell isolation that yields an abundant number of cells capable of undergoing rapid genetic modification, and applied this method in two models of therapeutic endothelialization.

METHODS: Venous blood was obtained from New Zealand White rabbits and mononuclear cells were separated by density gradient centrifugation. Without further enrichment, cells were plated on fibronectin coated dishes in the presence of endothelial specific growth factors. Cells were characterized via immunohistochemical detection of vWF, uptake of acetylated LDL, and the formation of vascular tubes on matrigel. LacZ gene transfer was performed using a VSV-G

pseudotyped retrovirus. Cells were either seeded onto a denuded carotid artery or sodded onto PTFE grafts prior to implantation. Both models were evaluated for transgene expression and endothelial coverage.

RESULTS: A relatively pure population of confluent autologous endothelial cells was obtained within 10-14 days. A highly efficient transduction of these cells (70-80%) was achieved using the concentrated pseudotyped vector without a need for antibiotic selection. Carotid arteries demonstrated >90% endothelial coverage with an equivalent degree of transgene expression at week 1, and retained 70-80% coverage with 10-15% expression by week 2. Unseeded control carotids had only 20% endothelial coverage at week 2 ($P<0.05$). PTFE grafts demonstrated 40-60% coverage with 10-15% expression at 4 weeks, while unsodded grafts demonstrated only 5% coverage at that time ($P<0.05$).

CONCLUSIONS: Endothelial lineage cells can be obtained from peripheral blood in a clinically feasible manner and can generate a population of autologous endothelial cells that is amenable to efficient genetic modification and that can accelerate endothelialization of denuded arteries and prosthetic vascular grafts.

**By Invitation*

F13. Cardiac Surgery in Infants Using Deep Hypothermic Circulatory Arrest Produces Less Systemic Inflammatory Response than Low-Flow Cardiopulmoary Bypass

Peter M. Tassani*, Andras Barankay*, Felix Haas*, Sun U. Pack*, Martina Heilmaier*, John Hess*, Ruediger Lange*, Josef A. Richter*; Muenchen, Germany

OBJECTIVE: Deep hypothermic circulatory arrest (DHCA) has been used with excellent clinical results. However, low-flow cardiopulmonary bypass (CPB) is nowadays favored because of a possible lower incidence of neurological complications. We hypothesized that less systemic inflammatory response (SIRS) may have contributed to the well known good clinical outcome with DHCA. The aim of the study was to compare the two techniques with respect to SIRS and postoperative course.

METHODS: In a randomized, controlled study 23 infants, weighing <10 kg scheduled for repair of congenital malformations (TGA, TAG, CAVSD) were enrolled. In 11 patients deep hypothermic circulatory arrest was used (Group CA) and low-flow CPB was performed in 12 patients (Group LF). As mediators of inflammatory response: interleukin-8, anaphylatoxin-C3a were measured at six time points perioperatively. Also clinical parameters and a radiological soft tissue index were compared. Statistics: Mean±SEM,U-test, $p<0.05$ significant.

RESULTS: All patients had an uneventful clinical course. Duration of DHCA was 40+4 min vs zero in Group LF, the CPB-time was shorter in Group CA (85±8 vs 130±19 min). However, the duration of surgery was similar in both groups (245±30 vs 246± 30 min). During CPB (rewarming) the concentration of C3a (3751±388 vs 5761±1688 ng/mL) was lower in Group CA then in Group LF. IL-8 was also lower in Group CA as compared to Group LF during and also 0.5h after CPB, when the peak concentrations were reached (64±12 vs 116±44 pg/mL). The weight gain on the first postoperative day was less in Group CAas compared to LF (65±61 vs 408±118 g). Modified ultrafiltration was used in all patients. Reduced edema formation Group CA patients could also be

demonstrated in a radiological soft tissue index. Two hours after CPB mean arterial pressure was higher in Group CA (60±2 vs 54±3 mmHg).

CONCLUSIONS: DHCA produces less SIRS compared to low-flow CPB. The consequences are less vasodilatation, higher mean arterial pressure, and reduced tissue edema postoperatively. Therefore, DHCA may have advantages in special subgroups of infants.

**By Invitation*

F14. Dynamic Balance of the Aorto-Mitral Junction

Carlos M. G. Duran, Emmanuel Lansac*, Khee Hiang Lim*, Yu Shomura*,

Wolfgang Goetz*, Hou Sen Lim*, Joon Hock Yeo*, Christophe Acar*,

Missoula, MT; Singapore, Singapore; Paris, France

OBJECTIVE: Aortic and mitral valves have been studied in isolation as if their function was independent. Sharing a common myocardial pump and orifice we hypothesized that both valves works in synchrony.

METHODS: 3-D Sonomicrometry crystals were implanted on the mitral annulus of 7 sheep: anterior and posterior trigone T1-T2; antero-posterior diameter AM-PM; longitudinal diameter (P1-P2). Three crystals (8 sheep) were implanted in the aortic root at the base of the left, right and non coronary sinus of Valsalva. Under stable hemodynamic conditions, geometric changes were time related to LV and aortic pressure.

RESULTS: From mid diastole to end systole MA area contracts by -16.1±1.9 % (mean±SEM), whereas the base of the aortic root expands by +29.8±3.3% during systole. Mitral annulus deformation is heterogeneous. The anterior mitral annulus (T1-T2: +11.5±2.3%) expands whereas the posterior MA (P1-P2:-12.1±1.5%) contracts in systole. The inter-trigonal distance (T1-T2) corresponds to the length between the base of the left and non coronary sinus of Valsalva which expands similarly (+12.9±2.0%) during systole. The mitral annulus antero-posterior diameter (AM-PM:-23.6±2.5%) is reduced more than twice the longitudinal diameter (P1-P2: -12.1±1.5%). This disparity of reduction is explained by the posterior displacement of the anterior mitral annulus. Therefore the inter-trigonal portion of the mitral annulus bulges towards the posterior part of the annulus during systole, in relation to the aortic root expansion.

CONCLUSIONS: Mitral annulus deformation during the cardiac cycle is closely related to aortic root dynamics. During systole the posterior movement of the mitral annulus allows for aortic root expansion in order to maximize ejection. While during diastole the aortic root volume reduction participates in the mitral annulus dilatation. This findings should impact both mitral annuloplasty and aortic valve replacement.

**By Invitation*

F15. Results of Tissue Engineered Auto-Xenograft Implanted in The Juvenile Sheep Model.

Pascal M. Dohmen*, Shigeyuki Ozaki*, Jessa Yperman*, Willem Flameng,

Wolfgang Konertz*; Berlin, Germany; Leuven, Belgium

OBJECTIVE: The auto-xenograft, a new generation heart valve prosthesis, was developed and evaluated to improve the durability of stentless valves without loosing the excellent hemodynamic function.

METHODS: In 8 juvenile sheep 6 weeks before implantation of the valves, a piece of vein was harvested. After endothelial cell (EC) expansion and decellularization of a porcine pulmonary valve, this matrix was reendothelialized. These tissue engineered valves were implanted into the RVOT of juvenile sheep. Valves were explanted after 7 days, 3 or 6 months and evaluated macroscopically, by X-ray, light microscopy (HE, Masson, Von Giesson, Von Kossa, PTAH strains), scanning and transmission electron microscopy. Quantitative determination of the calcium content of the cusps was performed by atomic absorption spectrometry.

RESULTS: All juvenile sheep showed fast recovery without any problems during the observation period after valve implantation. Echocardiography showed excellent function without regurgitation. Light microscopy showed monolayer of viable EC at all explanted auto-xenografts, confirmed by scanning electron microscopy. X-ray examination of explanted valves showed no cusp calcification, confirmed by atomic absorption spectrometry (2.17±0.24; 2.37±0.20; 0.34 ±0.14 mg/mg/min at respectively 7 days, 3 and 6 months).

CONCLUSIONS: In this novel tissue engineered auto-xenograft valve no cusp calcification occurred in the juvenile sheep model. Valve function showed to be normal over the entire observation period.

**By Invitation*

F16. Total Right Ventricular Exclusion Procedure: An Operation For Isolated Congestive Right Ventricular Failure

Shunji Sano*, Kozo Ishino*, Masaaki Kawada*, Shingo Kasahara*, Takushi

Kohmoto*, Mamoru Takeuchi*, Shin-Ichi Ohtsuki*; Okayama, Japan

OBJECTIVE(s): In order to prevent possible opposite effect of right ventricular (RV) volume overload on cardio-respiratory function, we developed a total RV exclusion procedure for the treatment of end-stage isolated congestive RV failure.

METHODS: Since 1996, this procedure has been performed in 5 patients in New York Heart Association (NYHA) class IV; 2 adults, aged 27 and 44 years, with arrhythmogenic RV dysplasia (ARVD) and 3 children, aged 6 months, 1 and 5 years, with Ebstein's anomaly. The entire RV free wall was resected along its attachment to the interventricular septum and then along the tricuspid annulus, sparing the pulmonary valve and a skeletonized right coronary artery. The tricuspid valve was excised and its orifice was closed with a polytetrafluoroethylene (PTFE) patch. The defect of RV free wall was covered with the PTFE patch in 2 ARVD patients and directly closed with the remnant of RV free wall in 3 Ebstein children. Coronary sinus blood flow was rerouted into the left atrium through an atrial septal defect. After resection of redundant right atrial wall, total cavopulmonary connection was constructed in 4 patients and bidirectional Glenn anastomosis in 1 infant.

RESULTS: There were no deaths. The patients were extubated at a mean of 14 hours postoperatively. At follow-up ranging from 4 to 50 months, mean cardio-thoracic ratio decreased from 74±7% before operation to 53±7% ($p<0.01$) and all patients are in NYHA class I.

CONCLUSIONS: The RV exclusion procedure provides effective decompression of the lung as well as the left ventricle, and may result in more effective volume loading of a surgically created single ventricle with incresed systemic output. We believe that this new surgical option offers excellent treatment for isolated congestive RV failure in critically ill patients.

**By Invitation*

F17. Capillary Leak Syndrome after Cardiopulmonary Bypass: Does it Exist?

Peter M. Tassani*, Hubert Schad*, Claudia Winkler*, Gregory P. Rising*,

Siegmund L. Braun*, Eberhard Kochs*, Ruediger Lange*, Josef A. Richter*;

Muenchen, Germany

OBJECTIVE: Operations using Cardiopulmonary bypass (CPB) may provoke a complex inflammatory response, which is claimed to induce capillary leakage of proteins, edema formation, and organ failure. This capillary leak syndrome, however, is a clinical diagnosis and has not been verified as yet by determination of protein leakage from the circulation. Therefore, the transcapillary transition of labelled protein, plasma cytokines, and complement were measured before and after CPB.

METHODS: Sixteen patients scheduled for elective CABG were enrolled in a prospective controlled study. The CPB circuit was primed with crystalloids only. Interleukin (IL)-6, tumor necrosis factor (TNF)-a, terminal complement complex (C5b9) were assessed before, during, and 3 hours after CPB. Transcapillary transition (TT) of intravenously injected Evans Blue (EB, a dye completely bound to protein), plasma volume (PV, from dilution of injected amount of EB) were measured before and during the 3^{rd} hour after CPB. Hemodynamic monitoring included mean arterial pressure (MAP), and cardiac index (CI). Statistics: Friedman's two-way ANOVA, Wilcoxon matched pairs signed rank test, * indicates significance of differences for $p<0.05$ (two-tailed). Data are given as mean±SEM.

RESULTS: All patients (age 59±2.8y) had an uneventful clinical course. CPB time was 84±7 min, OP time was 226±14 min. TT-EB did not change (table).

CONCLUSIONS: The data confirm the systemic inflammatory response following CPB. Contrary to expectation, TT-EB showed a tendency to decrease and PV remained unchanged despite elevated MAP and CI postCPB as compared to preCPB. Therefore, these data do not support the concept of increased protein leakage in the exchange vessels following CPB. Capillary leak syndrome did not occur.

	TT-EB	PV	IL-6	TNF□	C5b9	MAP	CI
Control	20±1.2	53±3	6±0.3	6±1	88±8	79±2	2.9±0.2
CPB+3h	14±1.7	53±2	178±43*	12±2*	260±30*	58±2*	3.8±0.2*

**By Invitation*

F18. Deep Hyypothermic Circulatory Arrest and Global Reperfusion Injury: Avoidance by Making a Pump Prime Reperfusion; A New Concept

Bradley S. Allen, Jeffrey S. Veluz*, Gerald D. Buckberg, Ernesto Aeberhard*,

Louis J. Ignarro*; Oak Lawn, IL; Los Angeles, CA

OBJECTIVE: Determine if the damage which occurs to all organs following deep hypothermic circulatory arrest (DHCA) can be diminished by changing pump prime components for reperfusion when reinstituting CPB.

METHODS: Fifteen pigs (2-3 months old) were cooled to 19°C using a-stat ph strategy. Five were cooled and rewarmed without ischemia (controls). The other 10 underwent 90 minutes of DHCA. Five had CPB reinstituted and were rewarmed without altering the pump blood prime. In the other 5 animals, the CPB blood prime was changed to ph-stat and modified (leukodepleted, hypocalcemic, hypermagnesemic, normoxic, and a sodium/hydrogen ion exchange inhibitor) during circulatory arrest before starting warm reperfusion. Oxidant injury was assessed from conjugated dienes (CD); vascular changes by endothelin (ET-1) levels; myocar-dial function by cardiac output (CO), and dopamine need; lung injury by pulmonary vascular resistance (PVR); and cellular damage by release of creatine kinase (CK) and transaminase (SCOT).

RESULTS: Compared to animals undergoing CPB without ischemia (controls), DHCA without modification of the reperfusate produced an oxi-dant injury (CD increased 0.91 vs 1.71 Abs 240nM/.5ml)*, depressed CO (6.0 vs 4.0 L/min)*; prolonged dopamine need*, elevated PVR (74% vs 197%)*; and increased release of CK (2695 vs 7267 u/L)*, SCOT (144 vs 170 u/L), and ET-1 (1.02 vs 2.56 pg/ml)*. This oxidant injury was markedly limited (CD only 0.90±0.07 Abs 240nM/.5ml)** with CPB prime modification, resulting in normal CO (5.1±0.9 L/min)**; minimal dopamine need**, no increase in PVR (28±31%)** or ET-1 levels (0.83±0.08 pg/ml)**; and lower release of SCOT (124±23u/L) and CK (3480±1089)**.

CONCLUSIONS: A global reperfusion injury after DHCA was identified and changed. The injury is mediated by oxygen free radicals resulting in cellular and vascular damage, with associated pulmonary and cardiac dysfunction. This injury is substantially avoided if the blood prime in the CBP circuit is modified to control the global reperfusate before reinstituting bypass.

*p< 0.05 vs controls; ** p<0.05 vs unmodified pump prime.

**By Invitation*

F19. Gene Transfer of Human Prostacyclin Synthase Transfected into the Liver is Effective for a Treatment of Pulmonary Hypertension.

Hitoshi Suhara*, Yoshiki Sawa*, Shigeaki Ohtake*, Motonobu Nishimura*,

Chieko Yokoyama*, Tadashi Tanabe*, Hikaru Matsuda; Suita, Osaka, Japan

OBJECTIVE(s): Although the efficacy of continuous administration of prostacyclin analogue has been reported as a treatment of primary and secondary pulmonary hypertension (PH), continuous intravenous administration is not ideal for QOL of patients because of the long-term administration.

Further excellent procedures are desired. We have tested the hypothesis that the gene transfection of PGIS into liver could be for a treatment of PH as a continuous delivery system of prostacyclin.

METHODS: PH was induced by subcutaneous injection of monocrotaline (MCT;60mg/kg) in rat PH model. Four weeks after the injection of MCT, human PGIS gene was transfected into the liver using HVJ liposome method. HVJ liposome vector complex without PGIScDNA was used for the control group. Hemodynamic indices and blood samples were obtained 5 days after HVJ injection. The rats were sacrificed on the same day. The expression of PGIS by ELISA, immunohistological stainning and western blot were examined in liver and lung tissue. Amount of total collagen and endothelin-1 of the lung tissues were also analyzed by ELISA.

RESULTS: RV/FApressure ratio decreased significantly in the PGIS group as compared with the control group (PGIS 0.60 ± 0.10 vs control; 0.85 ± 0.10, p = 0.0036). The plasma level of 6ketoPGFlawas significantly higher in the PGIS group than that in the control group (PGIS vs control: 3.43 ± 0.42ng/ml vs 22.34 ± 0.33ng/ml, p =0.0436). The amount of total collagen and endothelin-1 in lung showed less in the PGIS group than that in the control group. PGIS positive cells were detected in the liver in the PGIS group immunohistologically while not in the control group. Survival ratio was significantly higher in the PGIS group as compared with the control group (Logrank, p = 0.0375). These data indicated that the continuous delivery system of prostacyclin from the liver to the lung by gene transfection of PGIS might be effective for the treatment of pulmonary hypertension, in association with pulmonary arterial remodeling.

CONCLUSIONS: Thus, gene transfer of PGIS into the liver may be effective for *a* treatment of pulmonary hypertension.

**By Invitation*

F20. The Beneficial Vortex in Total Extracardiac Cavopulmonary Connection

Antonio Amodeo*, Mauro Grigioni*, Guido Oppido*, Carla Daniele*,

Giuseppe D'Avenio*, Giovanni Pedrizzetti*, Roberto Di Donate*; Rome and

Trieste, Italy

OBJECTIVE: Total extracardiac cavopulmonary connection (TECPC) is an established procedure but what is the best spatial arrangement remains controversial. We performed quantitative and qualitative analysis of the TECPC flow field to identify the most favorable hydrodynamic pattern among different arrangements applied to our patients.

METHODS: We selected two main groups among 110 patients who underwent a TECPC with an extracardiac conduit in the last 12 years: (1) with facing superior vena cava (SVC) and inferior vena cava (IVC) anastomoses; (2) with leftward offset of the IVC anastomosis. Glass blown TECPC phantoms were constructed based on NMR and angiographic images of our patients. In the phantom simulating group *2,* the IVC had a 22° inclination with respect to the vertical axis towards the left pulmonary artery (PA), and the offset between IVC and SVC anastomoses was 6 mm. Flow measurements were performed by a Quantel twins-switched laser and a (newly introduced) particle imaging velocimetry (PIV) system.The seeding of the flow was obtained with 10 micron silvered glass particles. A power dissipation study in different caval flow settings was also accomplished.

RESULTS: When applying a SVC/IVC flow distribution of respectively 40% and 60% of total systemic venous return, a vortex is visualized in the phantom of group 2, which rotates counter-

clockwise at the junction of caval streams. We demonstrate that this vortex, compared to the presence of competitive flows in the phantom of group 1, has a beneficial effect as it reduces energy dissipation, by modulating the flow distribution into the PAs. When a PA stenosis is simulated, the vortex shifts away from the stenotic PA until it dissipates.

CONCLUSIONS: TECPC with leftward offset of the IVC anastomosis is characterized by a central vortex at the junction of caval streams which regulates the flow partitioning through the PAs reducing the energy dissipation. This arrangement provides a more favourable hydrodynamic pattern than TECPC with facing cavopulmonary anastomoses.

**By Invitation*

9:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY

Room 6A-C, San Diego Convention Center

Introduction: Martin F. McKneally

Topic: Is it ethical to advertise surgical results to increase referrals?

Pro: Delos M. Cosgrove

Con: D Craig Miller

Moderator Tirone E. David

10:30 a.m. ACQUIRED CARDIAC CONTROVERSIES

Topic: Off-pump CABC is safer for the brain

Pro: Michael J. Mack

Con: Timothy J. Gardner

Moderator: Andrew S. Wechsler

11:15a.m.

Topic: Sixty year old patients requiring aortic valve replacement should have a mechanical prosthesis.

Pro: Robert W. Emery

Con: G. Michael Deeb

Moderator: Verdi J. Di Sesa

12:00 p.m. ADJOURN

10:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY GENERAL THORACIC CONTROVERSIES

Room 1A/B, San Diego Convention Center

Topic: Repair of giant intrathoracic stomach is best performed via laparoscopy

Pro: Daniel L. Miller

Con: Andre C. H. Duranceau

Moderator: Thomas W. Rice

11:15 a.m.

Topic: Wedge excision is optimal for the subcentimeter lung cancer.

Pro: Robert J. Keenan

Con: Joe B. Putnam, Jr

Moderator: Robert J. Ginsberg

12:00 p.m. ADJOURN

**By Invitation*

10:30 a.m. CONTROVERSIES IN CARDIOTHORACIC SURGERY CONGENITAL HEART CONTROVERSIES

Room 6D/E, San Diego Convention Center

10:30 a.m.

Topic: Homograft arch augmentation is the preferred technique for Norwood reconstruction in HLHS.

Pro: Thomas L. Spray
Con: Michael D. Black

Moderator: Vaughn Starnes

11:15 a.m.

Topic: Late pulmonary valve insertion is beneficial for tetrology of flow

Pro: Carl L. Backer

Con: William G. Williams
Moderator: Francisco J. Puga

12:00 p.m. ADJOURN

AMERICAN ASSOCIATION FOR THORACIC SURGERY
2000

GEOGRAPHICAL ROSTER

NECROLOGY

Reeve H. Belts, M.D., Asheville, North Carolina
Allan E. Bloomberg, M.D., Brooklyn, New York
H. Williams Clatworthy, M.D., Delaware, Ohio
Rene G. Favaloro, M.D., Buenos Aires, Argentina
Walter W. Fischer, M.D., Southern Pines, North Carolina
John P. Heaney, M.D., San Antonio, Texas
Bobby J. Heath, M.D., Jackson, Mississippi
Earle B. Kay, M.D., Cleveland, Ohio
Manuel E. M. Machado Macedo, M.D., Lisbon, Portugal
Joseph L. Mangiardi, M.D., Marathon, Florida
Edgar P. Mannix, M.D., Escondido, California
Rush E. Netterville, M.D., Marietta, Georgia
Maruf A. Razzuk, M.D., Dallas, Texas
Richard D. Sautter, M.D., Marshfield, Wisconsin
Stewart M. Scott, M.D., Asheville, North Carolina
Hawley H. Seiler, M.D., Tampa, Florida
Gabriel P. Seley, M.D., Boca Raton, Florida
Francis M. Woods, M.D., Needham, Massachusetts

GEOGRAPHICAL ROSTER

UNITED STATES

ALABAMA

Birmingham
Holman, William L
Kahn, Donald R
Kessler, Charles R
Kirklin, James K
Kirklin, John W
McGiffin, David C
Pacifico, Albert D

Montgomery
Simmons, Earl M

ARIZONA

Green Valley
McClenathan, James E

Paradise Valley
Nelson, Arthur R

Phoenix
Cornell, William P
Vaughn, Cecil C

Arcadia
Lindesmith, George G

Berkeley
Young, J Nilas

Bonita
Gonzalez-Lavin, Lorenzo

Burlingame
Ullyot, Daniel J

Capistrano Beach
Flynn, Pierce J

Chico
Becker, Ronald M

Coronado
Silver, Arthur W

Covina
Wareham, Ellsworth E

Del Mar
Fosburg, Richard G

El Macero

Scottsdale
Fisk, R Leighton
Pluth, James R
Shields, Thomas W
Sun City
Read, C Thomas
Tucson
Copeland, Jack G, III
Gulshan, Sethi K
Sanderson, Richard G

ARKANSAS

Little Rock
Campbell, Gilbert S
Read, Raymond C

CALIFORNIA

Alameda
Ecker, Roger R
Anaheim
Main, F Beachley
Loma Linda
Bailey, Leonard L
Gundry, Steven R
Razzouk, Anees J
Long Beach
Bloomer, William E
Stemmer, Edward A
Los Angeles
Benfield,John R
Buckberg, Gerald D
Cohen, Robbin G
Davis, Lowell L
DeMeester, Tom R
Holmes, E. Carmack
Kay, Jerome H
Khonsari, Siavosh
Laks, Hillel
Longmire, William P, Jr
Maloney, James V, Jr
Matloff, Jack M
McKenna, Robert J, Jr
Mulder, Donald G
Starnes, Vaughn A
Trento, Alfredo
Wells, Winfield J
Los Osos
Aronstam, Elmore M
Marina del Rey
Nelson, Ronald J
Martinez
Guernsey, James M
Mill Valley
Turley, Kevin
Montebello
Lui, Alfred H F
Oakland
Iverson, Leigh I G
Orange
Gazzaniga, Alan B
Ott, Richard A
Oxnard
Dart, Charles H, Jr
Palo Alto
Jamplis, Robert W
Peters, Richard M
Wilson, John L
Palos Verdes Estates
Stiles, Quentin R
Andrews, Neil C
Flintridge
Penido, John R F
Fresno
Evans, Byron H
Indian Wells
Carter, P Richard
Salyer, John M
Inglewood
Lee, Myles E
Irvine
Connolly, John E
La Canada
Meyer, Bertrand W
Lajolla
DeLaria, Giacomo A
Hutchin, Peter
Moreno-Cabral, Ricardo J
Pebble Beach
Ebert, Paul A
Miller, George E, Jr
Ramsay, Beatty H
Portola Valley
Fogarty, Thomas J
Rancho Mirage
Rubin, Morris
Ranchos Palos Verdes
Mandal, Ashis K
Sacramento
Berkoff, Herbert A
Delius, Ralph E
Follette, David M
Harlan, Bradley J
Hurley, Edward J
San Bernardino
Misbach, Gregory A
San Diego
Baronofsky, Ivan D
Daily, Pat O
Dembitsky, Walter P
Jamieson, Stuart W
Miller, Fletcher A
Mountain, Clifton F
Trummer, Max J
San Francisco
Ellis, Robert J
Hanley, Frank L
Hill, J.Donald
Reddy, Vadiyala M
Roe, Benson B
Thomas, Arthur N
Yee, Edward S
San Jose
Oakes, David D
San Marino
Tsuji, Harold K
Santa Ana
Pratt, Lawrence A
Wakabayashi, Akio
Santa Barbara
Jahnke, Edward J
Love, Jack W
Santa Cruz
Fishman, Noel H
Santa Monica
Chaux, Aurelio

Pasadena
Hughes, Richard K
Newman, Melvin M

Santa Rosa
Neville, William E
Sausalito
Zaroff, Lawrence I
Sonoma
Richards, Victor
Spring Valley
Long, David M, Jr
Stanford
Mark, James B. D
Miller, D. Craig
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
Victorville
Jurado, Roy A
Walnut Creek
May, Ivan A
Woodside
Mitchell, R. Scott

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

Fonkalsrud, Eric W
Morton, Donald L
Robertson, John M

CONNECTICUT

Bridgeport
Rose, Daniel M
Essex
Jaretzki, Alfred, III
Hartford
Kemler, R Leonard
Middlefield
Blumenstock, David A
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
Mainwaring, Richard D
Norwood, William I
Pecora, David V

DISTRICT OF COLUMBIA

Washington
Cox, James L
Katz, Nevin M
Keshishian, John M
Lefemine, Armand A
Midgley, Frank M
Simmons, Robert L

FLORIDA

Atlantic Beach
Stranahan, Allan
Aventura
Bregman, David
Bal Harbour
Grondin, Pierre R
Belleair
Lasley, Charles H
Coconut Grove
Center, Sol
Coral Gables
Cooke, Francis N

Delray Beach
Shumacker, Harris B, Jr
Gainesville
Alexander, James A
Jacksonville
Edwards, Fred H
Koster J Kenneth, Jr
Stephenson, Sam, Jr
Jupiter
Gerbasi, Francis S
Smeloff, Edward A

St Petersburg
Daicoff, George R
Tallahassee
Kraeft, Nelson H
Tamarac
Mendelssohn, Edwin
Tampa
Angell, William W
Robinson, Lary A
Winter Haven
Maurer, Elmer P R

Lady Lake
Fuller, Josiah
Lakeland
Brown, Ivan W, Jr
Largo
Wheat, Myron W, Jr
Marco Island
Conrad, Peter W
Miami
Bello, Alexis G
Bolooki, Hooshang
Daughtry, Dewitt C
Greenberg, Jack J
Jude, James R
Kaiser, Gerard A
Papper, Emanuel M
Ripstein, Charles B
Salerno, Tomas A
Subramanian, S
Thurer, Richard J
Wilder, Robert J
Miami Beach
Reis, Robert L
Spear, Harold C
Naples
Battersby, James S
Linberg, Eugene J
MacGregor, David C
Mundth, Eldred D
Smyth, Nicholas P D
Orlando
Scott, Meredith L
Ponte Vedra Beach
Barnhorst, Donald A
Gilbert, Joseph, Jr
Punta Gorda
Taber, Rodman E
Winter Park
Sherman, Paul H

GEORGIA

Atlanta
Graver, Joseph M
Gott, John P
Guyton, Robert A
Hatcher, Charles R, Jr
Hopkins, William A
Jones, Ellis L
Kanter, Kirk R
King, Richard
Lee, Arthur B Jr
Mansour, Kamal A
Miller, Joseph I, Jr
Rivkin, Laurence M
Symbas, Panagiotis
Williams, Willis H
Augusta
Ellison, Robert G
Rubin, Joseph W
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

IDAHO

Boise
Herr, Rodney H

ILLINOIS

Burr Ridge
Blakeman, Bradford P
Chicago
Amato, Joseph J
Backer, Carl L
Barker, Walter L
Breyer, Robert H
Campbell, Charles D
DiSesa, Verdi J
Faber, L. Penfield
Ferguson, Mark K
Fullerton, David A
Geha, Alexander S
Goldin, Marshall D
Hanlon, C. Rollins
Head, Louis R
Hunter, James A
Ilbawi, Michel N
Jeevanandam, Valluvan
Karp, Robert B
Kittle, C Frederick
Mavroudis, Constantine
Michaelis, Lawrence
Montoya, Alvaro
Najafi, Hassan
Raffensperger, John
Replogle, Robert L
Peoria
DeBord, Robert A
Springfield
Hazelrigg, Stephen R
Willowbrook
Leininger, Bernard J
Winnetka
Mackler, S Allen

INDIANA

Bloomington
O'Neill, Martin J, Jr
Fort Wayne
Ladowski, Joseph S
Indianapolis
Brown, John W
King, Harold
King, Robert D
Mandelbaum, Isidore
Siderys, Harry

IOWA

Cedar Rapids
Lawrence, Montague S
Levett, James M
Council Bluffs
Sellers, Robert D
Des Moines
Dorner, Ralph A
Zeff, Robert H

Snow, Norman J
Tatooles, C. J
Thomas, Paul A, Jr
Vanecko, Robert M
Warren, William H
Zajtchuk, Rostik

Elk Grove Village
Sullivan, Henry J

Evanston
Fry, Willard A
Rosengart, Todd K

Glencoe
Rubenstein, L H

Maywood
Mason, G. Robert
Pifarre, Roque

Oak Brook
Javid, Hushang
Jensik, Robert J
Nigro, Salvatore L

Oak Lawn
Allen, Bradley S

Park Ridge
Weinberg, Milton, Jr

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

Urbandale
Phillips, Steven J

KANSAS

Cunningham
Allbritten, Frank F, Jr

Lawrence
Miller, Don R

Mission Hills
Ashcraft, Keith W

Prairie Village
Holder, Thomas M

Shawnee Mission
Adelman, Arthur
Padula, Richard T

Wichita
Tocker, Alfred M

KENTUCKY

Lexington
Crutcher, Richard R
Mentzer, Robert M, Jr
Sanchez, Juan A
Todd, Edward P

Louisville
Austin, Erie H, III
Dowling, Robert D
Gray, Laman A, Jr
Mahaffey, Daniel E
RansdeU, Herbert, Jr

LOUISIANA

Baton Rouge
Berry, B Eugene
Beskin, Charles A

Campti
Bloodwell, Robert D

Metairie
Ochsner, Alton, Jr

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 Michael
Mills, Noel L
Moulder, Peter V
Ochsner, John L
Schramel, Robert J
VanMeter, Clifford H
Webb, Watts R

Shreveport
Mancini, Mary C

MAINE

Portland
Bredenberg, Carl E
Morton, Jeremy R

Windham
Gott, Vincent L
Greene, Peter S
Haller, J. Alex, Jr
Hankins, John R
Krasna, Mark J
McLaughlin, Joseph S
Michelson, Elliott
Watkins, Levi, Jr

Bethesda
Swain, Julie A

Glenarm
Turney, Stephen Z

Lutherville
Salomon, Neal W

Reisterstown
Heitmiller, Richard F

Worton
Walkup, Harry E

MASSACHUSETTS

Boston
Akins, Gary W
Aranki, Sary F
Austen, W. Gerald
Bougas, James A
Burke, John F
Collins, John J Jr
Couper, Gregory S
Daggett, Willard M
Daly, Benedict D T
Del Nido, Pedro J
Ellis, F. Henry, Jr
Folkman, M Judah
Grillo, Hermes C
Hilgenberg, Alan D
Jonas, Richard A
Lahey, Stephen J
Lazar, Harold L
Levitsky, Sidney
LoCicero, Joseph, III
Madsen, Joren C
Mathisen, Douglas J
Mayer, John E
Mentzer, Steven J

Hiebert, Clement

MARYLAND

Baltimore
Attar, Safuh
Baker, R. Robinson
Baumgartner, William A
Cameron, Duke Edward
Moncure, Ashby C
Sellke, Frank W
Shemin, Richard J
Sugarbaker, David J
Thurer, Robert L
Torchiana, David F
Vlahakes, Gus J
Wain, John C J.
Weintraub, Ronald M
Wright, Cameron D

Boylston
Moran, John M
Okike, Okike N

Brookline
Adams, David H
Berger, Robert L
Cohn, Lawrence H
Frank, Howard A

Burlington
Shahian, David M
Svensson, Lars G

Cambridge
Malcolm, John A
Whitman, Glenn J R

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
Vander Salm, Thomas J

MICHIGAN

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

Beverly Hills
Timmis, Hilary H

Detroit
Arbulu, Agustin
Baciewicz, Frank A, Jr
Pass, Harvey, I
Silverman, Norman A
Steiger, Zwi
Stephenson, Larry W
Walters, Henry L, III
Wilson, Robert F

Grand Rapids
Harrison, Robert W
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
Bolman, R. Morton, III
Emery, Robert W
Foker, John E
Garamella, Joseph J
Helseth, Hovald K

Joyce, Lyle D Maddaus, Michael A
Molina, J. Ernesto

St Charles
Codd, John E

Nicoloff, Demetre M
Shumway, Sara J
Ward, Herbert B

Rochester
Allen, MarkS
Bernatz, Philip E
Danielson, Gordon K
Deschamps, Claude
McGregor, Christopher G A
Mullany, Charles J
Olsen, Arthur M
Orszulak, Thomas A
Pairolero, Peter C
Puga, Francisco J
Schaff, Hartzell V
Trastek, Victor F

Shorewood
Kiser, Joseph C

Stillwater
Kaye, Michael P

Waubun
DeNiord, Richard N

MISSISSIPPI

Carthage
Logan, William D, Jr

Jackson
Johnston, J. Harvey, Jr

Madison
Hardy, James D

MISSOURI

Chesterfield
Bergmann, Martin

Columbia
Curtis, Jack J
Jones, James W
Silver, Donald
Walls, Joseph T

Frontenac
Penkoske, Patricia A
Strevey, Tracy E, Jr

Kansas City
Borkon, A Michael
Killen, Duncan A
Mayer, John H Jr
Piehler, Jeffrey M
Reed, William A
VanWay, Charles W, III

St Louis
Earner, Hendrick B
Connors, John P
Cooper, Joel D
Damiano, Ralph J, Jr
Ferguson, Thomas B
Fiore, Andrew C
Flye, M Wayne
Gay, William A Jr
Huddleston, Charles B
Johnson, Frank E
Johnson, Robert G
Kouchoukos, Nicholas T
Lewis, J Eugene, Jr
McBride, Lawrence R
Naunheim, Keith S
Pasque, Michael K
Patterson, G. Alexander
Roper, Charles L
Sasser, William F
Sundt,Thoralf M

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
Little, Alex G

NEW HAMPSHIRE

Center Harbor
Aaron, Benjamin L

Franconia
Taylor, Warren J

Hanover
Baldwin, John C

Lebanon
Marrin, Charles A. S
Nugent, William C
Plume, Stephen K
Sanders, John H, Jr

Peterborough
Glenn, William W. L

Stratham
Gaensler, Edward A

Windham
Burbank, Benjamin

NEW JERSEY

Alpine
Holswade, George R

Basking Ridge
Lewis, Ralph J

Belleville
Gerard, Franklyn P

Browns Mills
McGrath, Lynn B

Camden

Tenafly
Gerst, Paul H

Wyckoff
Adler, Richard H

NEW MEXICO

Albuquerque
Dietl, Charles A
Edwards, W. Sterling
Wernly, Jorge A

Buena Vista
Thai, Alan P

Santa Fe
Davila, Julio C

Santa Teresa
Glass, Bertram A

Silver City

Camishion, Rudolph C
DelRossi, Anthony J
Englewood
Ergin, M. Arisan
Fort Lee
Conklin, Edward F
Hackensack
Hutchinson, John E, III
Jersey City
Demos, Nicholas J
Long Branch
Fernandez, Javier
Millburn
Parsonnet, Victor
Moorestown
Morse, Dryden P
Morristown
Parr, Grant V S
Neptune
Roberts, Arthur J
New Brunswick
MacKenzie, James W
Scholz, Peter M
Newark
Donahoo, James
Gielchinsky, Isaac
Swan, Kenneth G
Pittstown
Garzon, Antonio A
Short Hills
Hochberg, Mark S

Fayetteville
Bugden, Walter F
Effler, Donald B
Fishers Island
Baue, Arthur E
Floral Park
Crastnopol, Philip
Honeoye Falls
Graver, William L
Larchmont
Steichen, Felicien M
Lido Beach
Hines, George L
Millerton
Green, George E
New York
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
Galloway, Aubrey C, Jr
Griepp, Randall B
Grossi, Eugene A
Isom, O. Wayne
King, Thomas C
Kirschner, Paul A
Krieger, Karl H
Lamberti, John J

Waddell, William R

NEW YORK

Albany
Canver, Charles C
Moores, Darroch W. O
Bay Shore
Ryan, Bernard J
Bellport
Finnerty, James
Bronx
Altai, Lari A
Ford, Joseph M
Prater, Robert W M
Gold, Jeffrey P
Hirose, Teruo
Veith, Frank J
Brooklyn
Acinapura, Anthony J
Bloomberg, Allan E
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
East Amherst
Andersen, Murray N
East Quogue
McCormack, Patricia M

Waters, Paul F
Wichern, Walter, Jr
Wolff, William I
Plattsburgh
Potter, Robert T
Rochester
DeWeese, James A
Hicks, George L
Schwartz, Seymour I
Stewart, Scott
Roslyn
Thomson, Norman B, Jr
Wisoff, George
Saranac Lake
Decker, Alfred M Jr
Slingerlands
Kausel, Harvey W
Staten Island
Adams, Peter X
Stony Brook
Bilfinger, Thomas V
Soroff, Harry S
Syracuse
Brandt, Berkeley, III
Kohman, Leslie J
Meyer, John A
Parker, Frederick, Jr
Valhalla
Moggio, Richard A
Reed, George E
Voorhees Ville
Foster, Eric D

NORTH CAROLINA

Asheville

Lansman, Steven L
Litwak, Robert S
Martini, Nael
McCord, Colin W
Mosca, Ralph S
Oz, Mehmet C
Quaegebeur, Jan M
Redo, S. Frank
Reemtsma, Keith
Rose, Eric A
Rusch, Valerie W
Skinner, David B
Smith, Craig R
Spencer, Frank C
Spotnitz, Henry M
Subramanian, Valavanur A
Tice, David A
Tyras, Denis H

Durham
Anderson, Robert W
Glower, Donald D
Harpole, David H, Jr
Jones, Robert H
Lowe, James E
Oldham, H. Newland, Jr
Sabiston, David C, Jr
Smith, Peter K
Wolfe, Walter G
Young, W. Glenn, Jr

Greensboro
Van Trigt, Peter, III

Greenville
Chitwood, W. Randolph, Jr
Sealy, Will C

High Point
Mills, Stephen A

Winston-Salem
Cordell, A. Robert
Hammon, John W, Jr
Hudspeth, Allen S
Kon, Neal D
Meredith, Jesse H
Pennington, D. Glenn

OHIO

Chagrin Falls
Ankeney, Jay L
Cross, Frederick S

Cincinnati
Albers, John E
Callard, George M
Flege, John B, Jr
Gonzalez, Luis L
Helmsworth, James A
Hiratzka, Loren F
Ivey, Tom D
Wilson, James M
Wright, Creighton B

Cleveland
Blackstone, Eugene H
Cosgrove, Delos M
Groves, Laurence K
Kirby, Thomas J
Loop, Floyd D
Lytle, Bruce W
McCarthy, Patrick M
Mee, Roger B. B
Betts, Reeve H
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

Meckstroth, Charles
Michler, Robert E
Williams, Thomas E, Jr

Dayton
DeWall, Richard A

Grove City
Kilman, James W

OKLAHOMA

Jenks
LeBeck, Martin B

Oklahoma City
Elkins, Ronald C
Felton, Warren L, II
Fisher, 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
Waldhausen, John A

Pettersson, Gosta B
Rice, Thomas W
Van Heeckeren, Daniel W

Columbus
Davis, J. Terrance
Kakos, Gerard S

Lancaster
Bonchek, Lawrence I
Rosemond, George P

Philadelphia
Acker, Michael A
Addonizio, V. Paul
Bavaria, Joseph E
Bowles, L Thompson
Diehl, James T
Edie, Richard N
Edmunds, L. Henry, Jr
Fineberg, Charles
Gardner, Timothy J
Goldberg, Melvyn
Guerraty, Albert J
Hargrove, W. Clark, III
Jacobs, Marshall L
Kaiser, Larry R
Karl, Tom R
MacVaugh, Horace
Mannion, John D
Spotnitz, William D
Spray, Thomas L
Wechsler, Andrew S

Pittsburgh
Bahnson, Henry T
Griffith, Bartley P
Hardesty, Robert L
Hattler, Brack G, Jr
Keenan, Robert J
Kormos, Robert L
Landreneau, Rodney J
Luketich, James D
Magovem, George J
Magovern, George J, Jr
Magovern, James A
Pontius, Robert G
Rams, James J
Siewers, Ralph D

Rosemont
Sink, James D

Rydel
Frobese, Alfred S

Sewickley
Clark, Richard E

Wayne
Lemmon, William M

Wilkes-Barre
Cimochowski, George E

Wynnewood
Wallace, Herbert W

Yardley
Sommer, George N, Jr

TEXAS

Austin
Tyson, Kenneth R T

Bedford
McPhail, Jasper L

Johnstown
Kolff, Jacob

RHODE ISLAND

Providence
Hopkins, Richard A
Moulton, Anthony L
Singh, Aran K

SOUTH CAROLINA

Charleston
Bradham, R Randolph
Crawford, Fred A, Jr
Kratz, John M
Reed, Carolyn E
Sade, Robert M
Swenson, Orvar

Columbia
Almond, Carl H

Hilton Head Island
Humphrey, Edward W

Isle of Palms
Mullen; Donald C

Landrum
Stayman, Joseph W

Spartanburg
Utley, Joe R

TENNESSEE

Jonesborough
Bryant, Lester R

Knoxville
Blake, Hu Al
Brott, Walter H
Domm, Sheldon E

Memphis
Cole, Francis H
McBurney, Robert P
Pate, James W
Robbins, S Gwin, Sr
Rosensweig, Jacob
Shochat, Stephen J
Watson, Donald C
Weiman, Darryl S

Nashville
Alford, William, Jr
Bender, Harvey W, Jr
Drinkwater, Davis C
Gobbel, Walter G, Jr
Merrill, Walter H
Pierson, Richard N, III,
Randolph, Judson G
Rankin, J. Scott
Sawyers, John L
Stoney, William S
Thomas, Clarence, Jr

Pearsall
Hood, Richard H, Jr

San Antonio
Calhoon, John H
Cohen, David J

Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Jessen, Michael E
Lambert, Gary J
Mack, Michael J
Mills, Lawrence J
Platt, Melvin R
Ring, W Steves
Seybold, William D
Urschel, Harold C, Jr

Galveston
Conti, Vincent R
Derrick, John R
Zwischenberger, Joseph B

Horseshoe Bay
Sutherland, R. Duncan

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
Reardon, Michael J
Reul, George J, Jr
Roth, Jack A
Safi, Hazim J
Walker, William E
Wukasch, Don C

Kemp
Davis, Milton V

Lubbock
Bricker, Donald L
Feola, Mario
Wallsh, Eugene

Marble Falls
Hood, R Maurice

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

Friday Harbor
Lawrence, G Hugh

Dooley, Byron N
Treasure, Robert L

Temple
Brindley, G. Valter, Jr

UTAH

Salt Lake City
Doty, Donald B
Jones, Kent W
Karwande, Shreekanth V
Liddle, Harold V
McGough, Edwin C
Mortensen, J D
Nelson, Russell M

VERMONT

Burlington
Leavitt, Bruce J

Richford
Grondin, Claude M

West Dover
Humphreys, George H, II

VIRGINIA

Altavista
Pierucci, Louis, Jr

Annandale
Akl, Bechara F
Burton, Nelson A
Lefrak, Edward A

Arlington
Klepser, Roy G

Aylett
Gwathmey, Owen

Charlottesville
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

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

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
Hill, Lucius D
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
Chopra, Paramjeet S
Cochran, Richard P
Young, William P
Marshfield
Myers, William O
Ray, Jefferson F, III
Mequon
Narodick, Benjamin
Milwaukee
Almassi, G. Hossein
Haasler, George B
Litwin, S Bert
Olinger, Gordon N
Tector, Alfred J
West Bend
Gardner, Robert J

WYOMING

Teton Village
Kaunitz, Victor H

OTHER COUNTRIES

ARGENTINA

Buenos Aires
Kreutzer, Guillermo O

AUSTRALIA

QUEENSLAND
Brisbane
O'Brien, Mark F, FRCS
SOUTH AUSTRALIA
Beaumont
Sutherland, H D'Arcy, M.S. FRCS

AUSTRIA

Leonding
Bruecke, Peter E
Salzburg
Unger, Felix H
Vienna
Wolner, Ernst

BELGIUM

Bertem
Sergeant, Paul T
Leuven
Flameng, Willem J
Lerut, Antoon E. M. R

BRAZIL

Rio de Janeiro
Meier, Milton A
Sanjose do RioPreto
Braile, Domingo M
Sao Paulo
Jatene, Adib D

CANADA

ALBERTA
Calgary
Bharadwaj, Baikunth
Miller, George E
Edmonton
Gelfand, Elliot T
Koshal, Arvind
Rebeyka, Ivan M
Sterns, Laurence P
BRITISH COLUMBIA
Vancouver
Ashmore, Phillip G
Jamieson, W. R. Eric
Tyers, G. Frank O
Victoria
Field, Paul
Stenstrom, John D
MANITOBA
Winnipeg
Barwinsky, Jaroslaw
Cohen, Morley
NOVA SCOTIA
Halifax
Murphy, David A
Ross, David B
ONTARIO
Collingwood
Heimbecker, Raymond
London
McKenzie, F Neil
Menkis, Alan H
Novick, Richard J
Mansfield
Pearson, F. Griffith
North York
Goldman, Bernard S

Oakville
Allen, Peter

London
Braimbridge, Mark V

Ottawa
Hendry, Paul J
Keon,Wilbert J
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
Chiu, Chu-Jeng (Ray)
Cossette, Robert
Dobell, Anthony R C
Duranceau, Andre C H
MacLean, Lloyd D
Morin, Jean E
Mulder, David S
Pelletier, L. Conrad
Scott, Henry J
Shennib, Hani
Tchervenkov, Christo I
Sainte-Foy
DesLauriers, Jean

ENGLAND

Bath
Belsey, Ronald
Cambridge
Kennedy, John H
Wallwork, John
Herts
Lennox, Stuart C
de Leval, Marc R
Goldstraw, Peter
Lincoln, Christopher R
Ross, Donald N
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
Bordeaux-Pessac
Baudet, Eugene M
Creteil
Loisance, Daniel
Le Plessis Robinson
Binet, Jean-Paul
Dartevelle, Philippe G
Lyon
Champsaur, Gerard L
Marseille
Merras, Dominique R
Montpellier
Thevenet, Andre A
Paris
Bachet, Jean E
Blondeau, Philip
Cabrol, Christian E. A
Carpentier, Alain F
Chauvaud, Sylvain M
Menasche, Philippe
Piwnica, Armand H
Planche, Claude
Weldon, Clarence S
Pessac
Couraud, Louis

GERMANY

Aachen
Messmer, Bruno J
Bad Oeynhausen
Korfer, Reiner
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
Pisa
Bortolotti, Uberto
Rome
Marcelletti, Carlo

JAPAN

Kamakura
Suma, Hisayoshi
Kanazawa
Iwa, Takashi
Watanabe, Yoh
Kitakyushushi
Miyamoto, Alfonso T
Kobe
Okita, Yutaka
Osaka
Kawashima, Yasunaru
Kitamura, Soichiro
Matsuda, Hikaru
Sendai
Fujimura, Shigefumi

Borst, Hans G

Neuss

Bircks, Wolfgang H

GREECE

Athens

Palatianos, George M

Sarris, George E

GUATEMALA

Guatemala City

Castaneda, Aldo R

Herrera-Llerandi, Rodolfo

HONG KONG

Shatin

He, Guo-Wei

IRELAND

Dublin

O'Malley, Eoin

ITALY

Bergamo

Parenzan, Lucio

Chieti

Calafiore, Antonio M

Milan

Peracchia, Alberto

Naples

Cotrufo, Maurizio

Mohri, Hitoshi

Shinjuku-ku

Imai, Yasuharu

Tokyo

Koyanagi, Hitoshi

Kurosawa, Hiromi

Naruke, Tsuguo

Wada, Juro J

KOREA

Seoul

Cho, Bum-Koo

MONACO

Monaco

Dor, Vincent

NETHERLANDS

Wassenaar

Brom, A Gerard

NEW ZEALAND

Waiwera Auckland

Barratt-Boyes, Brian G

P.R. OF CHINA

Beijing

Ying-Kai, Wu

PORTUGAL

Carnaxide

Melo, Joao Q

Coimbra

Antunes, Manuel J

ROMANIA

Targu-Mures

Deac, Radu C

RUSSIA

Moscow

Bockeria, Leo A

SAUDI ARABIA

Riyadh

Al-Halees, ZohairY

SCOTLAND

Edinburgh

Logan, Andrew

Glasgow

Wheatley, David J

SPAIN

Barcelona

Aris, Alejandro

Murtra, Marcos

Madrid

Rivera, Ramiro

Santander

Revuelta, Jose Manuel

SWEDEN

Sollentuna

Bjork, Viking

Umea

Aberg, Torkel H

SWITZERLAND

Arzier

Hahn, Charles J

Lausanne

vonSegesser, Ludwig K

Pully

Naef, Andreas P

Zurich

Senning, Ake

Turina, Marko I

SYRIA

Damascus

Kabbani, Sami S

UNITED ARAB EMIRATES

Abu Dhabi

Todd, Thomas R J

VENEZUELA

Caracas

Tricerri, Fernando E

AMERICAN ASSOCIATION FOR THORACIC SURGERY CHARTER MEMBERS

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

AMERICAN ASSOCIATION

FOR THORACIC SURGERY

THE BY-LAWS

ARTICLE I NAME

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

ARTICLE II PURPOSE

The purposes of the Association shall be:

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

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

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

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

ARTICLE III MEMBERSHIP

Section 1. There shall be three classes of members: Honorary, Senior, and Active. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws. Honorary members shall have the privilege of voting but shall not be eligible to hold office.

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

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

Section 4. Active Membership shall be limited to six hundred. A candidate to be eligible must be a physician and a citizen of the United States of America or Canada, unless is 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 III, Section 8.

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

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

ARTICLE V Officers

Section 1. The officers of the Association shall be 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 may be 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 meetings of the Association and at all meetings of the Council.

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

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

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve as Treasurer of the Association.

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 seven members: the President, Secretary, and Treasurer of the Association and four members-at-large, one member being appointed by the President each year to serve a term of four years. The Chairman shall be the member-at-large serving his fourth year. The duties of the Committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and Fellows, past, present, and future.

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

Section 9. The Ethics Committee shall consist of five members appointed by the Council. 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 form 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 derived by the Association subject to the provisions of Section 4, following.

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

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 that 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 no 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 XI Initiation Fees, Dues and Assessments

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

Section 2. Annual dues for Active Members hall 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, 2000

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 Valerie W. Rusch, M.D.
New York, New York

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWSHIP

The Evarts A. Graham Memorial Traveling Fellowship was established in 1951 by The American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young surgeons from abroad who have completed their formal training in general, thoracic, and cardiovascular surgery. The award allows the recipient to study a year to intensify his training in a program of special interest and to travel to several sites to broaden his overall training and increase his contacts with thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 48 young surgeons from 25 countries have completed their training at thoracic surgical centers.

1st	1951 -52	L. L. Whytehead Winnepeg, 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 . Sl im 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 Goto 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	Florentino 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

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 Toralf 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, III

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.

Philadelphia, 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-Adregenic Receptor Based Gene Therapy"

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

Duke University Medical Center

**Charter Member*

+Board of Directors

†Director Emeritus

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

THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION

THIS IS YOUR FOUNDATION

Unlike other organizations to which you make philanthropic contributions, The Thoracic Surgery Foundation works directly for your specialty. The Foundation supports research and education initiatives to increase knowledge and enhance treatment of patients with cardiothoracic diseases; develops the skills of cardiothoracic surgeons as surgeon-scientists and health policy leaders; and, strengthens society's understanding and trust in the profession.

Your Foundation is making a difference in cardiothoracic surgery. This is possible only because of your support. The Foundation is entirely supported through private donations. So, please don't forget your gift to The Foundation. If you have not yet made your annual gift to your Foundation, now is the time! If you make an annual gift to The Foundation of appreciated stocks, bonds or mutual funds, you avoid capital gains tax and earn an income tax deduction by donating rather that selling these assets. This may be better for you than a gift of cash.

If you have been thinking of making a charitable contribution to TSFRE, this may be the time to consider a planned gift. Often, this type of giving enables an individual to give a larger gift at a cost that is actually lower than if the gift were to be made outright. You may also find that planned giving enables you to meet other personal financial goals while making significant charitable gifts.

You may give to The Foundation through a revocable instrument, such as a bequest in your will, or through an irrevocable instrument like a charitable lead trust or a charitable remainder trust. You may also give through a life insurance policy or your retirement plan. For more information about your annual gift or a deferred gift, contact Frank Kurtz, TSFRE Executive Director, 312/464-6100, extension 3425; FAX 312/527-6635; Email frank_kurtz@sba.com.

THE THORACIC SURGERY FOUNDATION

FOR RESEARCH AND EDUCATION

THE THORACIC SURGERY FOUNDATION AWARDS

2001 RESEARCH AND EDUCATION AWARD RECIPIENTS

* Individual Research Investigator Grants
* Research Fellowship Awards
* Career Development Awards
* Alley-Sheridan Scholarships

THE THORACIC SURGERY FOUNDATION RESEARCH GRANT provides operational support of original research projects by cardiothoracic surgeons who have completed their formal training and who are certified or eligible by The American Board of Thoracic Surgery or its equivalent.

Marc R. Moon, M.D., Washington University School of Medicine

Alfred C. Nicolosi, M.D., Medical College of Wisconsin

NINA S. BRAUNWALD RESEARCH FELLOWSHIP provides support to women who wish to acquire investigational skills.

Meena Nathan, MBBS, FRCS (Edin), FRCS (Glas), Brigham and Women's Hospital

ALLEY-SHERIDAN SCHOLAR-IN-RESIDENCE AT HARVARD

The Foundation offers Alley-Sheridan tuition scholarships for cardiothoracic surgeons to pursue a year of study in health care policy at Harvard University. The following individuals have received this award.

William R. Berry, M.D., Napa, CA

Alan J. Spotnitz, M.D., New Brunswick, NJ

PREVIOUS RESEARCH AWARD RECIPIENTS

THE THORACIC SURGERY FOUNDATION RESEARCH FELLOWSHIP provides support to surgeons and surgical trainees who wish to acquire investigational skills.

Edward M. Boyle, Jr., M.D., The University of Washington

Seth Force, M.D., The University of Pennsylvania

Julie R. Glasson, M.D., Stanford University School of Medicine

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

Daniel Kreisel, M.D., University of Pennsylvania

Baiya Krishnadasan, M.D., University of Washington

Paul C. Lee, M.D., University of Pittsburgh

Sang H. Lee, M.D., University of California, San Diego Medical Center

Raja S. Mahidhara, MD, University of Pittsburgh

Steffen Pfeiffer, MD, Vanderbilt University Medical Center

Robert S. Poston, Jr., M.D., Stanford University Medical Center

Andrew J. Sherman, M.D., Northwestern University Medical School

Christopher L. Skelly, M.D., The University of Chicago

Michael A. Smith, M.D., Washington University

Wilson Y. Szeto, MD, Hospital of the University of Pennsylvania

Mohan Thanikachalam, MD, University of Miami

Vinod H. Thourani, **M.D.**, Emory University School of Medicine

Tomasz A. Timek, M.D., Stanford University

Edward Yiming Woo, M.D., University of Pennsylvania

BAXTER HEALTHCARE CORPORATION RESEARCH FELLOWSHIP AWARD provides support to surgeons and surgical trainees who wish to acquire investigational skills.

Richard W. Kim, M.D., Yale University School of Medicine

THE THORACIC SURGERY FOUNDATION RESEARCH GRANT provides operational support of original research projects by cardiothoracic surgeons who have completed their formal training and who are certified or eligible by The American Board of Thoracic Surgery or its equivalent.

James S. Allan, M.D., Massachusetts General Hospital

Richard P. Embrey, M.D., The Medical College of Virginia

Paul M. Kirshbom, MD, Children's Hospital of Pennsylvania

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

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

Si M. Pham, M.D., University of Pittsburgh

Todd K. Rosengart, M.D., The New York Hospital - Cornell Medical Center

David S. Schrump, M.D., National Cancer Institute

Thomas K. Waddell, PhD, MD, Toronto General Hospital and the University of Toronto

THE THORACIC SURGERY FOUNDATION CAREER DEVELOPMENT AWARD provides support for applicants who have completed their residency training and who wish to pursue investigative careers in thoracic surgery.

Kenneth McCurry, MD, University of Pittsburgh

NINA S. BRAUNWALD CAREER DEVELOPMENT AWARD provides salary to women in academic cardiothoracic surgery at early stages of their faculty careers.

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

Mary C. Mancini, M.D., Louisiana State University Medical Center

Lynne A. Skaryak, MD, University of Massachusetts Medical Center

Patricia A. Thistlethwaite, M.D., University of California - San Diego

NINA S. BRAUNWALD RESEARCH FELLOWSHIP provides support to women who wish to acquire investigational skills.

Kathryn Quadracci Flores, M.D., Brigham and Women's Hospital

Melina R. Kibbe, M.D., University of Pittsburgh

Elizabeth N. Morgan, M.D., University of Washington

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

Jennifer Dale Walker, M.D., Medical University of South Carolina

PREVIOUS EDUCATION AWARD RECIPIENTS

ALLEY-SHERIDAN SCHOLAR-IN-RESIDENCE AT HARVARD

The Foundation offers Alley-Sheridan tuition scholarships for cardiothoracic surgeons to pursue a year of study in health care policy at Harvard University. The following individuals have received this award.

Edward J. Dunn, M.D., Milwaukee, WI

Edgar L. Feinberg, III, M.D., Lafayette, LA

Peter P. McKeown, M.D., Tampa, FL

Joseph J. McNamara, M.D., Honolulu, HI

Juan A. Sanchez, M.D., Lexington, KY

Paul N. Uhlig, M.D., Wichita, KS

ALLEY-SHERIDAN EXECUTIVE COURSE SCHOLARS The Alley-Sheridan Fund was established within The Thoracic Surgery Foundation by Mr. David Sheridan on behalf of his life-long friend and collaborator, Dr. Ralph Alley, to provide educational opportunities, especially in health care policy matters for cardiothoracic surgeons. This fund has been used to make a generous grant from The Foundation to the Kennedy School of Government at Harvard University to develop an intensive executive course in management and health care policy, *Understanding the New World of Health Care: A Health Policy Program for Physicians, Trustees and Health Care Leaders.* To date, The Foundation has named 102 individuals to receive Alley-Sheridan Scholarships to attend this course.