2003 ANNUAL MEETING PROGRAM

AMERICAN ASSOCIATION FOR THORACIC SURGERY
2002 – 2003

President Fred A. Crawford, Jr., Charleston, SC
Vice-President Joel D. Cooper, St. Louis, MO
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Douglas J. Mathisen (2003) Boston, MA
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Valerie W. Rusch (2005), New York, NY
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2003 ANNUAL MEETING COMMITTEES

LOCAL ARRANGEMENTS

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Katherine Vernot-Jonas, Chair
Kathy Vlahakes
Linda Sugarbaker
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GRAHAM EDUCATION & RESEARCH FOUNDATION

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John A. Elefteriades, Director (2003)	New Haven, CT

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Timothy J. Gardner (2007)	Philadelphia, PA

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Richard A. Jonas (2003)	Boston, MA
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Mark J. Krasna	Baltimore, MD
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Alex G. Little	Las Vegas, NV
Jeffrey B. Rich	Norfolk, VA
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Sidney Levitsky	Boston, MA
Joe B. Putnam, Jr.	Houston, TX
Peter K. Smith	Durham, NC
Cameron D. Wright	Boston, MA

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY REPRESENTATIVES 2002-2003

AMERICAN ASSOCIATION OF BLOOD BANKS

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AMERICAN COLLEGE OF SURGEONS ADVISORY COUNCIL FOR CARDIOTHORACIC SURGERY

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ASSOCIATION OF PHYSICIANS ASSISTANTS IN CARDIOVASCULAR SURGERY
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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY REPRESENTATIVES 2002-2003

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NATIONAL ASSOCIATION FOR BIOMEDICAL RESEARCH (NABR)
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Clifford H. Van Meter (2003) New Orleans, LA

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David N. Helman (2004) Boston, MA
Bassem N. Mora (2004) Boston, MA

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Lawrence H. Cohn..... Boston, Massachusetts
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Marc R. de Leval..... London, United Kingdom
Davis C. Drinkwater, Jr..... Nashville, Tennessee
M. Arisan Ergin..... Englewood, New Jersey
J. William Gaynor..... Philadelphia, Pennsylvania
Donald D. Glower, Jr..... Durham, North Carolina
Alden H. Harken..... Denver, Colorado
David H. Harpole..... Durham, North Carolina
Axel Haverich..... Hannover, Germany
Valluvan Jeevanandam..... Chicago, Illinois
Larry R. Kaiser..... Philadelphia, Pennsylvania
Harold L. Lazar..... Boston, Massachusetts
Antoon E. M. R. Lerut..... Leuven, Belgium
Michael A. Maddaus..... Minneapolis, Minnesota
Roger B. B. Mee..... Cleveland, Ohio
Robert M. Mentzer, Jr..... Lexington, Kentucky
Marc R. Moon..... St. Louis, Missouri
Ivan M. Rebeyka..... Edmonton, Alberta, Canada
Thomas W. Rice..... Cleveland, Ohio
Robert C. Robbins..... Stanford, California
Valerie W. Rusch..... New York, New York
Hartzell V. Schaff..... Rochester, Minnesota
Frank W. Sellke..... Boston, Massachusetts
Francis G. Spinale..... Charleston, South Carolina
David J. Sugarbaker..... Boston, Massachusetts
Marko I. Turina..... Zurich, Switzerland
Willem Van Oeveren..... Groningen, Netherlands
William G. Williams..... Toronto, Ontario, Canada

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
192M922	Washington, DC	Samuel Robinson
1922-1923	Chicago, IL	Howard Lilienthal
1923-1924	Rochester, MN	Carl A. Hedblom
1924-1925	Washington, DC	Nathan W. Green
1925-1926	Montreal, QUE	Edward W. Archibald
1926-1927	New York, NY	Franz Torek
1927-1928	Washington, DC	Evarts A. Graham
1928-1929	St. Louis, MO	John L. Yates
1929-1930	Philadelphia, PA	Wyman Whittemore
1930-1931	San Francisco, CA	Ethan Flagg Butler
1931-1932	Ann Arbor, MI	Frederick T. Lord
1932-1933	Washington, DC	George P. Muller
1933-1934	Boston, MA	George J. Heuer
1934-1935	New York, NY	John Alexander
1935-1936	Rochester, MN	Carl Eggers
1936-1937	Saranac Lake, NY	Leo Eloesser
1937-1938	Atlanta, GA	Stuart W. Harrington
1938-1939	Los Angeles, CA	Harold Brunn

1939-1940	Cleveland, OH	Adrian V.S. Lambert
1940-1941	Toronto, ONT	Fraser B. Gurd
1943-1944	Chicago, IL	Frank S. Dolley
1945-1946	Detroit, MI	Claude S. Beck
1946-1947	St. Louis, MO	I. A. Bigger
1947-1948	Montreal, QUE	Alton Ochsner
1948-1949	New Orleans, LA	Edward D. Churchill
1949-1950	Denver, CO	Edward J. O'Brien
1950-1951	Atlantic City, NJ	Alfred Blalock
1951-1952	Dallas, TX	Frank B. Berry
1952-1953	San Francisco, CA	Robert M. Janes
1953-1954	Montreal, QUE	Emile Holman
1954-1955	Atlantic City, NJ	Edward S. Welles
1955-1956	Miami Beach, FL	Richard H. Meade
1956-1957	Chicago, IL	Cameron Haight
1957-1958	Boston, MA	Brian Blades
1958-1959	Los Angeles, CA	Michael E. DeBakey
1959-1960	Miami Beach, FL	William E. Adams
1960-1961	Philadelphia, PA	John H. Gibbon, Jr.
	(Deceased 1/11/61)	Richard H. Sweet
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, CA	David J. Dugan
1976-1977	Toronto, ONT	Henry T. Bahnson
1977-1978	New Orleans, LA	J. Gordon Scannell
1978-1979	Boston, MA	John W. Kirklin
1979-1980	San Francisco, CA	Herbert Sloan
1980-1981	Washington, DC	Donald L. Paulson
1981-1982	Phoenix, AZ	Thomas B. Ferguson
1982-1983	Atlanta, GA	Frank C. Spencer
1983-1984	New York, NY	Dwight C. McGoon
1984-1985	New Orleans, LA	David C. Sabiston
1985-1986	New York, NY	James R. Malm
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1987-1988	Los Angeles, CA	Paul A. Ebert
1988-1989	Boston, MA	W. Gerald Austen
1989-1990	Toronto, ONT	F. Griffith Pearson
1990-1991	Washington, DC	Keith Reemtsma
1991-1992	Los Angeles, CA	John A. Waldhausen
1992-1993	Chicago, IL	John L. Ochsner
1993-1994	New York, NY	Aldo R. Castaneda
1994-1995	Boston, MA	Robert B. Wallace
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1996-1997	Washington, DC	David B. Skinner

1997-1998	Boston, MA	Floyd D. Loop
1998-1999	New Orleans, LA	Lawrence H. Cohn
1999-2000	Toronto, ONT	Delos M. Cosgrove
2000-2001	San Diego, CA	James L. Cox
2001-2002	Washington, DC	Timothy J. Gardner

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 3, 2003 12:00 NOON - 5:00 P.M.

(3.5 ACCME HOURS) HYNES CONVENTION CENTER ROOM 306

Co-Chairmen: Ralph J. Damiano, Jr. and Larry R Kaiser

1:00 p.m. Keynote Address

Do No Harm! Is it Immoral to Utilize Living Persons as Sources of Organs and Tissues for Transplantation?

Arthur Caplan, Ph.D., Trustee, Professor and Chairman of the Department of Medical Ethics, University of Pennsylvania.

1:20 p.m. Panel Discussion: Randomized Clinical Trials vs Observational Studies: "Surgical Research or Comic Opera"

Moderator: Larry R Kaiser, Hospital of the University of Pennsylvania, Philadelphia, PA

Panel Members: Joel D Cooper, Washington University, St. Louis, MO

Eugene H Blackstone, Cleveland Clinic Foundation, Cleveland, OH

Eric Rose, Columbia University, Dept. of Surgery, New York, NY

2:05 p.m. The Peer-Review Process in Medical Publishing

An Editor's Perspective:

L. Henry Edmunds, Hospital of the University of Pennsylvania, Philadelphia, PA

A Reviewer's Perspective:

Frank W Sellke, Beth Israel Deaconess Medical Center, Boston, MA

2:50 p.m. Thinking Financially as well as Surgically: Implications of Reimbursement on an Academic Surgical Practice

Daniel Cooper, Chief Operating Officer, Department of Surgeiy, University of Pennsylvania

3:15 p.m. Break

3:35 p.m. Panel Discussion:

Ethical Dilemmas in Cardiothoracic Surgery Role of Private Industry, Patient Confidentiality, Implementation of New Technology, Malpractice Issues

Moderator: Ralph J. Damiano, Jr., Washington University School of Medicine, St. Louis, MO

Panel Members: Arthur Caplan, University of Pennsylvania, Philadelphia, PA

Martin F McKneally, University of Toronto, Toronto, Ontario

Richard D Weisel, Toronto General Hospital, Toronto, Ontario

Bruce W Lytle, Cleveland Clinic Foundation, Cleveland, OH

4:20 p.m. An Educator's Perspective on Academic Surgery: A Paradigm Shift in Training
Ross Ungerleider, Doernbecher Children's Hospital, Portland, OR

4:40 p.m. Looking at Quality: Assessing One's Own Results in the Context of National or Regional Benchmarks
William C Nugent, Dartmouth-Hitchcock Medical Center, Lebanon, NH

5:00 p.m. ADJOURN

SATURDAY, MAY 3

5:00 - 7:00 p.m.

GENERAL THORACIC BIOLOGY CLUB

St. Botolph Club, 199 Commonwealth Ave., Boston

5:00 - 7:00 p.m.

ADULT CARDIAC BIOLOGY CLUB

Provincetown/Orleans Rooms, Marriott Hotel

COURSE 1: ADULT CARDIAC SURGERY SYMPOSIUM

SUNDAY, MAY 4, 2003 8:00 A.M. - 5:00 P.M.

(7.75 ACCME HOURS) HYNES CONVENTION CENTER BALLROOM

Chairman: Patrick M McCarthy, Cleveland Clinic Foundation, Cleveland, OH

SESSION 1: EVOLVING CONCEPTS AND NEW SURGICAL APPROACHES FOR ATRIAL FIBRILLATION

8:00 a.m. What the Surgeon Needs to Know About the Epidemiology and Pathophysiology of Atrial Fibrillation
Robert A Schweikert, Cleveland Clinic Foundation, Cleveland, OH

8:20 a.m. Catheter Based Ablation for AF

Fred Morady, University of Michigan Medical Center, Ann Arbor, MI

8:40 a.m. Indications, Surgical Techniques, and Late Results of the Classic Maze Procedure (Video)
Kartell V Schaff, Mayo Clinic, Rochester, MN

9:10 a.m. Minimally Invasive Maze Procedure (Video)
James L Cox, The World Heart Foundation, Washington, DC

9:40 a.m. Discussion 10:00 a.m.

10:00 a.m. Break

10:15 a.m. New Tools for Atrial Fibrillation Surgery (Video)
Ralph J Damiano, Jr., Washington University School of Medicine, St. Louis, MO

10:45 a.m. New Operative Approaches and Results in Surgery for AF (Video)
A Marc Gillinov, Cleveland Clinic Foundation, Cleveland, OH

11:15 a.m. Panel Discussion
Moderator: Patrick M McCarthy, Cleveland Clinic Foundation, Cleveland, OH

Case Presentations:

Patient #1: Tachycardia mediated cardiomyopathy

Patient #2: Myxomatous mitral regurgitation with dilated left atrium and chronic atrial fibrillation

Patient #3: 82 year old with three-vessel coronary artery disease and atrial fibrillation

12:00 noon Lunch

SESSION 2: STATE-OF-THE-ART TREATMENTS OF CORONARY ARTERY DISEASE

12:45 p.m. Early and Late Results with Coronary Artery Bypass Surgery: The Gold Standard
Bruce W Lytle, Cleveland Clinic Foundation, Cleveland, OH

1:05 p.m. Drug Eluting Stents: How Much Will Referrals for Coronary Artery Bypass Change?
Martin B Leon, Lenox Hill Hospital New York, NY

1:25 p.m. New Devices for Proximal and Distal Coronary Anastomoses: Less Morbidity with Smaller Incisions? (Video)
Randall K Wolf, Ohio State University Medical Center, Columbus, OH

1:45 p.m. Experimental Approaches to Coronary Revascularization (Video)
Stephen Oesterle, Medtronic, Inc., Minneapolis, MN

2:05 p.m. Panel Discussion

Moderator: Kent W Jones, Salt Lake City, UT

2:30 p.m. Break

SESSION 3: ISCHEMIC MITRAL REGURGITATION: AN ONGOING SURGICAL CHALLENGE

2:45 p.m. Ventricular Changes Associated with Ischemic Mitral Regurgitation: Echocardiographic Insights, and Potential Device Solutions
Robert A Levine, Massachusetts General Hospital, Boston, MA

3:15 p.m. Mitral Valve Repair or Replacement for Ischemic Mitral Regurgitation: Surgical Techniques (Video)
Antonio M Calafiore, University of Chieti, Chieti, Italy

3:35 p.m. Coronary Artery Bypass in a Patient With Moderate Mitral Regurgitation: The Case for Concomitant Mitral Valve Repair (Video)
[1]David H Adams, Mount Sinai Medical Center, New York, NY

3:55 p.m. Coronary Artery Bypass in a Patient With Moderate Mitral Regurgitation: The Case Against Valve Repair
John A Elefteriades, Yale University School of Medicine, New Haven, CT

4:15 p.m. Panel Discussion

Moderator: D. Craig Miller, Stanford University Medical School, Stanford, CA

5:00 p.m. ADJOURN -

WELCOMING RECEPTION

EXHIBIT HALL

COURSE 2: GENERAL THORACIC SURGERY SYMPOSIUM

Sponsored in cooperation with The General Thoracic Surgical Club

SUNDAY, MAY 4, 2003 7:50 A.M. - 5:00 P.M.

(7 ACCME HOURS) HYNES CONVENTION CENTER ROOM 302

Chairman: Douglas E. Wood, University of Washington Medical Ctr., Seattle, WA

SESSION 1 EARLY STAGE LUNG CANCER
Moderator: Douglas E. Wood

8:00 a.m. Current status of lung cancer screening: Current Challenges and Potential Solutions

Phillip M Boiselle, Beth Israel Deaconess Medical Center, Boston, MA

8:15 a.m. Endoscopic lung cancer detection and staging

Armin Ernst, Beth Israel Deaconess Medical Center, Boston, MA

8:30 a.m. PET as a predictor of lung cancer prognosis

Hubert Vesselle, University of Washington, Seattle, WA

8:45 a.m. How I Do It: Cardiopulmonary function testing and risk assessment

[1]Mark K Ferguson, University of Chicago, Chicago, IL

8:55 a.m. Controversy: Management of stage INSCLC in the high-risk patient

Radiation is preferred

Walter J Curran, Jr., Thomas Jefferson University, Philadelphia, PA

Surgical results are worth the risks

Joe B Putnam, Jr., Anderson Cancer Center, Houston, TX

9:15 a.m. Discussion

9:45 a.m. BREAK

SESSION 2 LOCALLY ADVANCED LUNG CANCER

Moderator: Valerie W. Rusch

10:15 a.m. Development and organization of a multidisciplinary thoracic oncology clinic

Frank C Detterbeck, University of North Carolina, Chapel Hill, NC

10:30 a.m. How I Do It: Carinal resection

Paolo Macchiarini, Ph.D., Hannover, Germany

10:40 a.m. Controversy: Management of solitary station N2 disease

Primary resection

Steven M Keller, Bronx, New York

Induction therapy and surgery

Valerie W Rusch, Memorial Sloan Kettering Cancer Center, New York, NY

11:10 a.m. Controversy: Adjuvant therapy for resected N1/N2 NSCLC

Radiation

Walter J Curran, Jr., Thomas Jefferson University, Philadelphia, PA

Chemotherapy

Tom J Lynch, Massachusetts General Hospital, Boston, MA

[1]*1986-88 Research Scholar*

11:30 a.m. Discussion

12:00 noon Lunch

SESSION 3 END-STAGE LUNG DISEASE

Moderator: G Alexander Patterson

1:00 p.m. Long term results of LVRS

Joel D Cooper, Washington University School of Medicine, St. Louis, MO

1:15 p.m. Technical advances in lung reduction

[1]Tom K Waddell, University of Toronto, Toronto, Ontario

1:25 p.m. How I Do It: Selection of patients for bullectomy

Henning A Gaissert, Massachusetts General Hospital, Boston, MA

1:35 p.m. Pulmonary endarterectomy for chronic embolic pulmonary hypertension

[2]Michael S Mulligan, University of Washington, Seattle, WA

1:50 p.m. Controversy: Lung transplant for non- infectious lung disease

Single lung is preferred

Walter Klepetko, Vienna, Austria

Bilateral lung is preferred

Shaf Keshavjee, Toronto General Hospital, Toronto, Ontario

2:10 p.m. Inununologic advances in lung transplantation

James S Allan, Massachusetts General Hospital, Boston, MA

2:25 p.m. How I Do It: Management of secondary pneumothorax

John D Mitchell, University of Colorado, Denver, CO

2:35 p.m. Discussion

3:00 p.m. Break

SESSION 4 ESOPHAGUS

Moderator Keith S Naunheim

3:30 p.m. Photodynamic therapy and endoscopic mucosal resection for early stage esophageal cancer

Kenneth K Wang, Mayo Clinic, Rochester, MN

3:45 p.m. Controversy: Barrett's with high-grade dysplasia

Surveillance

Brian J Reid, University of Washington, Seattle, WA

Esophagectomy

Richard I Whyte, Stanford University, Palo Alto, CA

4:15 p.m. How I Do It: Endoscopic Zenker's Diverticulotomy

Alex G Little, University of Nevada, Las Vegas, NV

4:30 p.m. Discussion

5:00 p.m. ADJOURN -

WELCOME RECEPTION EXHIBIT HAIL

[1]2000-02 *Research Scholar*

[2]*2002-04 Research Scholar*

COURSE 3: CONGENITAL HEART DISEASE SYMPOSIUM

SUNDAY, MAY 4, 2003 8:00 A.M. - 5:30 P.M.

(7.75 ACCME HOURS) HYNES CONVENTION CENTER ROOM 304

Chairman: Ross M Ungerleider, Doernbecher Children's Hospital, Portland, OR

8:00 a.m. Introduction and Welcome

Ross M Ungerleider

SESSION 1 Immediate Outcomes Following Norwood's Procedure for HLHS

8:05 a.m. Audience response questions to generate reflection of practice patterns by participants

8:15 a.m. "Conventional" Management

Vaughn A Starnes, University of Southern California, Los Angeles, CA

8:30 a.m. Alpha Blockade

Glen S Van Arsdale, University of Toronto, Toronto, Ontario

8:45 a.m. Routine Postoperative Ventricular Assist

Irving Shen, Doernbecher Children's Hospital, Portland, OR

9:00 a.m. RV-PA Shunt

Frank L Hanley, Stanford University, Stanford, CA

9:15 a.m. Mixed Venous 02 as a Reflection of Outcome Following Variations on the Norwood Procedure

Scott M Bradley, Medical University of South Carolina, Charleston, SC

9:30 a.m. Discussion

9:45 a.m. BREAK

SESSION 2 Complex Right and Left Ventricle Outflow Obstruction

10:10 a.m. Complex LVOT Obstruction

Thomas L Spray, Children's Hospital of Philadelphia, Philadelphia, PA

10:30 a.m. Mitral Valve Replacement with Pulmonary Autograft Monocusp and Bovine Jugular Vein Reconstruction of the RVOT

John W Brown, Indiana University, Indianapolis, IN

10:50 a.m. Non-homograft valved conduits

Kirk R Kanter, Emory University School of Medicine, Atlanta, GA

11:10 a.m. Stented Bioprostheses

James J. Jaggers, Duke University Medical Center, Durham, NC

11:30 a.m. Surgery for Congenital Heart Disease in the Adult Age: A multicentric European Study

Giovanni Stellin, Policlinico Universita, Padova, Italy

11:50 a.m. Discussion

12:00 noon Lunch

SESSION 3 Difficult Problems

(to include video as part of presentation when appropriate)

1:00 p.m. Management of TGA in infants who present at ago2 wks

Pedro J Del Nido, Children's Hospital, Boston, MA

1:20 p.m. Tetralogy of Fallot-Indications for Transatrial Repair

Edward L Bove, University of Michigan Medical Center, Ann Arbor, MI

1:40 p.m. Repair of Ebstein's Anomaly in Neonates and Young Infants

[1]Christopher J Knott-Craig, H.S.C./ Oklahoma University Medical Center, Oklahoma City, OK

2:00 p.m. TOP with Absent Pulmonary Valve

J William Gaynor, Children's Hospital of Philadelphia, Philadelphia, PA

2:20 p.m. Tricuspid Atresia with Normally Related Great Vessels

David Ashburn, Toronto, Ontario

2:40 p.m. Single Lung Fontan

Christo I Tchervenkov, The Montreal Children's Hospital, Montreal, Quebec

3:00 p.m. BREAK

3:20 p.m. Difficult Problems

PANEL WITH AUDIENCE RESPONSE
This session will convene a panel of "experienced" congenital heart surgeons who will help provide opinions about some of the difficult problems currently being encountered by congenital heart surgeons. The panel will help generate the questions but the floor will be open to the participants for other questions as well. Topics can range from those discussed as a part of the program to others that have not been covered, but will all relate to timely and important issues in the field.

It will certainly include the questions about CPB strategies and about Outcome measures. Panel members will be asked to generate questions that can be educational for the audience or which can serve to sample practice patterns of the audience. All questions will be posed as audience response questions and then discussed by the panel.

The experts' answers to questions will be recorded separately and experts will be asked to discuss their answers for the audience.

This session is designed to be lively, pertinent to whatever issues are current in May, 2003, and should generate a lot of participation from the audience.

[1]*1989-90 Graham Fellow*

PANEL:

Ross M Ungerleider, Doernbecker Children's Hospital, Portland, OR, *Moderator*

Edward L Bove, University of Michigan Medical Center, Ann Arbor, MI

John W Brown, Indiana University School of Medicine, Indianapolis, IN

Constantine Mavroudis, Children's Memorial Hospital, Chicago, IL

Pedro J Del Nido, Children's Hospital, Boston, MA

Thomas L Spray, Children's Hospital of Philadelphia, Philadelphia, PA

Tom R Karl, University of California at San Francisco, San Francisco, CA

Frank L Hanley, Stanford University, Stanford, CA

Martin J Elliott, The Great Ormond Street Hospital for Children, London, UK

Frank A. Pigula, Children's Hospital of Pittsburgh, Pittsburgh, PA

SESSION 4 Outcomes and Education

4:00 p.m. How to Interpret Data-an Overview for Clinical Surgeons

Eugene H Blackstone, Cleveland Clinic Foundation, Cleveland, OH

4:20 p.m. Risk Adjusted Mortality for Congenital Heart Surgery

Kathy Jenkins, Children's Hospital, Boston, MA

4:40 p.m. The Aristotle Score for Congenital Heart Surgery

Francois Lacour-Gayet, Eppendorf University Hospital, Hamburg, Germany

5:00 p.m. Discussion

5:30 p.m. ADJOURN-

WELCOME RECEPTION

EXHIBIT HALL

83RD ANNUAL MEETING

Hynes Convention Center

Boston, MA * May 4-7, 2003

PROGRAM OUTLINE

MONDAY, MAY 5, 2003

7:45 a.m. Business Session

(Limited to Members Only)

Ballroom, Hynes Convention Center

8:00 a.m. SCIENTIFIC SESSION

(8 minutes presentation, 12 minutes discussion)

Ballroom, Hynes Convention Center

Moderators: Fred A Crawford, Jr.

Tirone E. David

1. Prospective, Randomized Trial Reveals No Changes in Neurocognitive Function Between On- and Off-Pump Patients

Aftab R Kherani*, Ronald Lazar*, David D'Alessandro*, Eric Rose, Barry Esrig, Joanne Festa*, Paul Kurlansky*, Michael Parides*, Lopa Gupta*, Annetine Gelijns*, Faisal Cheema*, Alan Moskowitz*, Minoo Kavarana*, [1]Mehmet C Oz, New York, NY; Miami, FL.

Discussant: John D Puskas

BACKGROUND: Proponents have argued that off-pump coronary artery bypass grafting (CABG) may result in fewer neurocognitive changes than traditional on-pump surgery. The purpose of this study was to compare preoperative neurocognitive function with that at 30 days following either on- or off-pump CABG to determine whether cardiopulmonary bypass was associated with neurocognitive impairment.

METHODS: This prospective, randomized trial enrolled 56 consecutive patients who were assigned to undergo on- (n=27) or off-pump (n=29) CABG. All but one off-pump patient underwent a baseline neurocognitive examination comprised of 13 neurological tests assessing seven areas: mental status, language, verbal memory, visual memory, visual construction, attention, and executive skills. An identical battery of tests was repeated at 30 days following surgery. The scores were standardized according to age-specific criteria.

RESULTS: In none of the seven areas was there a significant change in neurocognitive performance in either the on- or off-pump groups. Table 1 summarizes the results*. Additionally, the changes from baseline to 30-days did not differ between groups for any cognitive area. The randomized groups were also compared with respect to the proportion of patients experiencing a decline of at least one standardized unit for each cognitive area using Fisher's exact test (no statistically significant differences).

Table 1 : Summary of the Standardize means*

Neurocognitive Area	Mental Status	Verbal Memory	Visual Construction	Visual Memory	Language	Attention	Executive Function
Off Pump CABG: Baseline	0.93	-0.53	0.15	-0.54	0.16	0.62	0.94
Off Pump CABG: Post-Op day 30	0.42	0.86	-0.04	0.54	0.36	0.01	-0.92
On Pump CABG: Baseline	0.70	-0.82	-0.50	-1.03	-0.55	0.90	026
On Pump CABG: Post-op day 30	0.38	0.30	0.31	0.46	0.46	-0.20	-0.29
p-value	0.65	0.72	0.82	0.26	0.18	0.40	0.11

CONCLUSIONS: At 30 days following surgery, neurocognitive performance was unchanged compared to baseline in both on- and off-pump patient groups. Cardiopulmonary bypass was not associated with increased risk to any of seven major areas of neurocognitive ability.

[1]*1994-96 Research Scholar*

**By Invitation*

2. A Decade of Living Lobar Transplantation: Recipient Outcomes

Vaughn A Starnes, Mark L Barr*, Marlyn Woo*, Felicia A Schenkel*, Monica V Horn*, Michael E Bowdish*, Renzo Pessotto*, CraigJ Baker*, Ross M Bremner*, Robbin G Cohen, Winfield J Wells, Richard Barbers*, Los Angeles, CA.

Discussant: G. Alexander Patterson

OBJECTIVE: Living lobar transplantation was developed as a procedure for patients considered too ill to await cadaveric transplantation. We report recipient outcomes during our first decade of living lobar transplantation.

METHODS: 128 living lobar transplants were performed in 123 patients between 1993 and 2002. 84 patients were adults (age 27 ± 7.7 yrs) and 39 were pediatric (age 13.9 ± 2.9 yrs). Overall follow-up is now 343 patient-years (range 0-9.3 yrs/patient). During the same period, 81 cadaveric pulmonary transplants (71 bilateral and 10 heart-lung) were performed.

RESULTS: The primary indication for transplantation in lobar recipients was cystic fibrosis (n = 108). At the time of transplantation, 83 (67%) patients were hospitalized and 23 (19%) were intubated (p = 0.0001 and 0.001 vs cadaveric cohort). 1, 3, and 5 year actuarial survival among living lobar recipients was 70, 53, and 44% respectively, which did not differ from the cadaveric cohort (KM, p = 0.35), or the 2002 ISHLT Registry. There was no difference in actuarial survival between adult and pediatric living lobar recipients (KM, p = 0.73). There were 63 deaths among living lobar recipients, with infection being the predominant cause (n = 31), followed by non-compliance (n = 8), graft dysfunction (n = 5), OB (n = 4), and others (n = 15). The overall incidence of acute rejection was 0.02 episodes/patient-month, representing 0.8 episodes/patient. OB was diagnosed in 13 (10.5%) patients. Post-transplant mean percent predicted FVC, FEV_1 and FEF_{25-75} were comparable in adult bilateral cadaveric and living lobar recipients who survived greater than 90 days.

CONCLUSIONS: These results support the continued use of living lobar transplantation, as survival and pulmonary function are comparable to cadaveric transplantation. Additionally, these results are important if this procedure is to be considered an option at more pulmonary transplant

centers given continued organ shortages and differences in philosophical and ethical acceptance of live organ donors.

**By Invitation*

3. Left sided lesions after anatomic repair of Transposition of the Great Arteries, VSD and Coarctation. Surgical factors

[1]Alain Serraf*, Siamak Mohammadi*, Emre Belli*, Francois Lacour-Gayet, Bertrand Aupecle*, Regine Roussin, Anita Touchot*, Jacqueline Bruniaux*, Claude Planche, Le Plessis-Robinson, France.

Discussant: Christo I Tckervenkov

OBJECTIVE: To evaluate if anatomic factor such the aorto pulmonary size mismatch and if various surgical techniques to repair TGA-VSD and CoA affect the late occurrence of left sided lesions, namely reCoA and aortic insufficiency (AI)

METHODS: From 1985 to 2002, 110 pts underwent anatomic repair of TGA, VSD and CoA. One stage repair was performed in 69 and 2-stage repair in 41. Prior to repair, aortic and pulmonary anuli, as well as sino tubular aortic junction diameters were recorded. CoA was repaired by EEEA in 70 and homograft patch augmentaion in 40. The VSD was closed through the native PA in 66, the RA in 11 and the RV in 33.Neoaortic discongruence was treated by resection of the posterior sinus of Valsalva in 7, anterior splitting of ascending aorta (Asc Ao) in 71 and aortic patch extension down to the asc Ao in 32. Before hospital discharge neoaortic anulus and asc Ao diameters, AI, gradient across the aortic arch were recorded. The mean follow-up was 67±51 months.

RESULTS: PA to Ao mismatch was significantly higher in single versus two stage repair (1.9±0.2 vs 1.3±0.1, $p<0.05$). Risk factors for reCoA (n=18) were single stage repair ($p<0.05$) and EEEA ($p<0.05$).Risk factors for evolutive AI were closure of VSD through the PA ($p<0.01$), EEEA ($p<0.05$) and residual asc Ao discongruence with turbulences and no gradient($p<0.01$).Reccurrent CoA was treated by patch augmentation (n=9) or balloon dilatation (n=9). Four pts had an AYR, 3 had a Bentall procedure and 3 other had both Bentall and aortic arch repair. Although two stage repair provided better results with no reCoA and no AI, it carried out a higher overall mortality ($p<0.05$).

CONCLUSIONS: Neonatal single stage repair of TGA, VSD and CoA remains the optimal approach. Avoidance of long term reCoA and/or AI requires aortic arcch patch augmentation, no residual neo ascending aorta turbulences and careful precaution for VSD closure.

[1]1993-94 Graham Fellow

**By Invitation*

4. Intramural Hematoma Caused by Penetrating Atherosclerotic Ulcer

Kwang Ree Cho*, Anthony W Stanson*, D Dean Potter*, Kenneth J Cherry, Harzell V Schaff, ‘Thoralf M Sundt III, Rochester, MN.

Discussant: John A Elefteriades

OBJECTIVE: Although it is widely accepted that intramural hematoma (IMH) of the descending thoracic aorta may be managed expectantly, it has been argued that the identification of an associated penetrating atherosclerotic ulcer (PAU) predicts more unstable behavior and should encourage operative intervention. To better define the clinical behavior of this entity, we reviewed cases of IMH with PAU at our institution.

METHODS: The diagnostic radiology database was searched from 1961 to 2002 for the diagnoses of IMH or PAU. All available imaging studies were reviewed by a cardiovascular radiologist (AWS) to confirm the diagnosis and, when serial studies were present, to perform serial measurements. For this analysis, only cases with both entities were considered.

RESULTS: Sixty cases of IMH with PAU were confirmed. The mean age was 72 ± 8 years and males predominated (41 M, 19 F). Comorbidities were common with hypertension in 57 (95%), tobacco use in 48 (80%), abdominal aortic aneurysm in 34 (57%), coronary artery disease in 29 (48%), chronic obstructive pulmonary disease in 14 (23%) and renal failure in 14 (23%). Thirty-seven (62%) cases had localized IMH at the time of diagnosis. Extensive IMH involving the entire descending thoracic aorta was more likely to be symptomatic at presentation, (22/ 23 vs. 28/37; $p<0.05$). Complications of the PAU (localized aortic dissection, pseudoaneurysm, or rupture) were identified at presentation in 32% (19/60). Of non-operated IMH, the mean thickness decreased significantly by one month and completely resolved by one year in all patients. There were 2 deaths (5%) within 30-days in the medically treated group. Twenty patients (33%) underwent operative intervention with an operative (30-day) mortality rate of 25% ($p<0.05$ vs medically treated). The mean age of survivors (medical or surgical) was lower (71 ± 7 vs 78 ± 8 years, $p=0.039$) and mean systolic pressure on admission higher (153 ± 31 vs 126 ± 18, $p=0.028$) than of non-survivors. Neither ulcer size or depth, nor extent of hematoma predicted adverse outcome (surgical intervention or mortality).

CONCLUSIONS: In most cases, even in the presence of PAU, IMH of the descending thoracic aorta can be managed nonoperatively in the acute setting.

9:20 a.m. Second Alfred E. Blalock Research Scholar Presentations

Abbas Ardehali, UCLA School of Medicine Thomas K Waddell, University of Toronto and Toronto General Hospital

Evarts A. Graham Memorial Traveling Fellowship Presentation

Cliff K C Choong, Auckland, New Zealand

9:35 a.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

10:20 a.m. Thoracic Surgery Foundation for Research and Education

Ballroom, Hynes Convention Center John R Benfield, President

[1]1994-96 Research Scholar

**By Invitation*

10:25 a.m. SCIENTIFIC SESSION

Moderators: Joel D Cooper

Tirone E David

5. Tricuspid Valve Repair In Hypoplastic Left Heart Syndrome

Richard G Ohye*, Carlen A Gomez*, EricJ Devaney*, Edward L Bove, Ann Arbor, MI.

Discussant: Thomas L Spray

OBJECTIVE: The outlook for patients with hypoplastic left heart syndrome (HLHS) has dramatically improved over the past two decades. However, the development of tricuspid regurgitation (TR) remains a significant obstacle to successful staged repair in a subset of these patients. The results of tricuspid valve repair in this challenging patient population remains largely unknown.

METHODS: From August 1995-April 2002, 25 patients with classic HLHS presented to the C. S. Mott Children's Hospital at the University of Michigan with significant (3-4+) tricuspid regurgitation following the Norwood procedure necessitating tricuspid valve repair. The clinical and Doppler/echocardiographic data were retrospectively reviewed to determine the efficacy of the repair and patient outcome.

RESULTS: The mean patient age was 24 months +/- 31 months and mean weight 10.0 kg +/-6.9 kg. All 25 patients had undergone a Norwood procedure, 14 a hemi-Fontan operation and 3 a Fontan operation. Operative repair was tailored to the individual valve pathology. There were 2 operative deaths (8%), one from ventricular dysfunction and one from an intra-opera-tive coronary injury Of the early survivors, post-operative Doppler/echocardiography revealed 0-2+ regurgitation in 18 (72%) and 3+ in 5 (20%). Follow-up was 96% complete (24/25) at a mean of 27 months. There were 6 late deaths (26%, 6/23), 6 re-repairs or replacements (26%, 6/23), and 1 transplantation (4%, l/23).0f the 17 late survivors, 15 were in NYHA class I, 1 in NYHA class II and one was lost to follow-up. 13 of the 16 followed survivors have not required further intervention and remain with 0-2+ TR in 11, 3+ and 4+ in 1 each.

CONCLUSIONS: Tricuspid valve repair can be accomplished in this challenging patient population with satisfactory results Successful repair, as defined by TR of 0-2+, was initially achieved in 72% (18/25) of patients. While 44% (11/25) maintained a durable result, the significant need for re-intervention and late mortality due not only to tricuspid valve dysfunction, but also ventricular dysfunction and pulmonary vascular disease, underscore the difficulties in caring for the suboptimal HLHS patient.

**By Invitation*

6. Results of Lung Volume Reduction Surgery in High Risk Patients

Bryan F Meyers*, Roger D Yusen*, Stephen S Lefrak*, TraceyJ Guthrie*, Gail Davis*, G Alexander Patterson, Joel D Cooper, St. Louis, MO.

Discussant: Douglas E Wood

OBJECTIVE: A recent report from the National Emphysema Treatment Trial (NETT) indicated that lung volume reduction surgery candidates with both an FEV_1 and a DLCO £20% predicted were at high risk for postoperative mortality and were unlikely to benefit from the surgery. Furthermore, projected 3 year survival for this group was less than 40%.

METHODS: We reviewed 280 patients who underwent bilateral lung volume reduction surgery at our institution between January 1993 and December 2001 (Group A). All patients met our

previously published criteria including a heterogeneous distribution of emphysematous destruction. Of these 280 patients, 19 patients (6.8 %) had both a preop FEV_1 and DLCO £20% predicted, meeting the NETT criteria for high risk (Group B). Objective measures of preop function and improvement at six months postop are reported as mean values and % of predicted. The Kaplan-Meier survival at 3 years was determined for each group.

RESULTS: Ninety day operative mortality was 3.6% for Group A and 5.3% for Group B. For Group A the FEV_1 improved from .72 L (26%) to 1.11 L (40%), a 54% improvement; DLCO improved from 44% to 52%, a 15% improvement; RV declined from 5.90 L (294%) to 4.03 L (202%), a 32% improvement; and room air Pa02 improved from 64 mm Hg to 72 mm Hg. For Group B, the FEV_1 improved from 0.49 L (18%) to 0.81 L (29%), a 65% improvement; DLCO improved from 16% to 27%, a 67% improvement; RV declined from 6.51 L (316 %) to 4.33 L (208%), a 34% improvement; and room air Pa02 improved from 53 mm Hg to 64 mm Hg. Kaplan-Meier survival for Group A was 84% and for Group B 82% at three years.

CONCLUSIONS: Although caution is indicated with LVRS candidates having both a FEV_1 and a DLCO £20% predicted, selected patients in this category with heterogeneous disease can undergo LVRS with significant benefit and without a significant increase in risk.

**By Invitation*

7. Tight Glycemic Control In Diabetic Patients During Coronary Artery Bypass Graft Surgery Increases Long-Term Survival And Decreases Recurrent Ischemic Events.

Harold L Lazar, Stuart R Chipkin*, Carmel A Fitzgerald*, Yusheng Bao*,

Howard Cabral*, Carl A Apstein*, Boston and Springfield, MA.

Discussant: Anthony P Furnary

OBJECTIVE: Our previous studies have shown that tight glycemic control in diabetic coronary artery bypass graft (CABG) patients with a modified glucose-insulin-potassium (GIK) solution reduces postoperative morbidity and shortens hospital stay. This study sought to determine whether the early beneficial effects achieved with tight glycemic control would result in increased long-term survival and a decreased incidence of recurrent ischemic events.

METHODS: One hundred forty one diabetic patients undergoing CABG were prospectively randomized to tight glycemic control (serum glucose 120-200mg/dl) with GIK or standard therapy (serum glucose <250mg/dl) using intermittent subcutaneous insulin beginning prior to anesthesia and continuing for 12 hours following surgery.

RESULTS: There were no 30 day mortalities in either group. Results are expressed as the number of occurrences in each group and the mean values± standard error. Three year follow-up was obtained in 85% of patients.

	GIK(N=72)	NO GIK(N=69)	P Value
Glucose(mg/dl) 6 hours postop	138±4	260±6	<0.0001
Free Fatty Acids(mEq/L) 6 hours postop	0.32±0.03	0.57±0.05	<0.001
Lactates(mg/dl) 6 hours postop	1.82±0.14	2.33±0.31	0.04

Death	1	6	0.06
Median time to death(years)	2.3	2.1	0.04
Angina Class 0	56	44	0.03
Recurrent wound infection	1	7	0.03
Recurrent ischemic events	4	13	0.01

CONCLUSIONS: Tight glycemic control with GIK in diabetic CABG patients enhances long-term survival and decreases the incidence of ischemic events and wound complications.

11:25 a.m. PRESIDENTIAL ADDRESS

Thoracic Surgery Education - Responding to a Changing Environment

Fred A Crawford, Jr., Charleston, South Carolina

Introduced by: Joel D Cooper

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

Exhibit Hall, 2nd Floor, Hynes Convention Center

**By Invitation*

MONDAY AFTERNOON, MAY 5, 2003

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION -ADULT CARDIAC SURGERY
(8 minutes presentation, 12 minutes discussion)

Ballroom, Hynes Convention Center

Moderators: Timothy J Gardner,

O Wayne Isom

8. Propensity Case Match Analysis of Off Pump CABG in Patients with Atheromatous Aortic Disease
Ram Sharony*, Eugene A Grossi M.D., Paul C Saunders*, Charles F Schwartz*, Aubrey C Galloway, Greg Ribakove*, Alfred T Culliford, Marc Kanchuger*, Robert M Applebaum*, Itzhak Kronzon*, Stephen B Colvin, New York, NY.

Discussant: Nicholas T Kouchoukos

OBJECTIVE: Atheromatous aortic disease (AAD) is a strong risk factor for excessive mortality and stroke with CABG. Initial studies have demonstrated some benefits of off-pump CABG (OPCAB) surgery in high-risk patients. We used propensity case-match analysis to compare the outcomes of OPCAB and cardiopulmonary bypass (CPB-CABG) techniques in patients with severe AAD.

METHODS: Routine intra-operative transesophageal echocardiography (TEE) on 5737 patients undergoing isolated CABG identified 913 (15.9%) study subjects with severe AAD in the arch or ascending aorta. OPCAB was performed on 235 (25.7%) patients by surgeon preference; the remainder underwent CPB-CABG. Data was collected prospectively, and greedy propensity

matched-pairs analysis was used to construct a control cohort from the CPB-CABG patients. Variables used for matching included age, gender, ejection fraction, history of stroke, CHF, urgent operation, diabetes, previous cardiac surgery, renal disease, and acute Ml.
RESULTS: 229 OPCAB patients were case-matched with 229 CPB-CABG patients. Hospital mortality was significantly lower in OPCAB compared with CPB-CABG (12/229, 5.2% vs. 25/229, 10.9%; p=0.03), as was stroke incidence (5/229, 2.2% vs. 14/229,6.1%; p<0.03). Freedom from any postoperative complication was improved in OPCAB compared with CPB-CABG (210/ 229, 91.7% vs. 180/229, 78.6%; p<0.01). Multivariant analysis of all patients revealed increased mortality was associated with CPB-CABG (OR=3.4; p<0.01), acute MI (OR=8.1; p<0.01), dialysis (OR=4.4; p<0.01), and peripheral vascular disease (OR=1.2; p=0.01).

CONCLUSIONS: Patients with severe AAD undergoing OPCAB have significantly lower rates of operative mortality, perioperative stroke, and overall complications than with CPB-CABG. Routine intra-operative TEE allows identification of these patients and directs choice of surgical technique.

**By Invitation*

9. Emergency Conversion To Cardiopulmonary Bypass During Attempted Off-Pump Revascularization Results In Increased Morbidity And Mortality

Nirav C Patel*, Nilesh U Patel*, John McCabe, V A Subramanian, New York, NY.

Discussant: Irving L Kron

OBJECTIVE: Off-pump coronary revascularisation (OPCAB) is increasingly performed and many reports have shown reduced morbidity in comparison to conventional CABG on cardiopulmonary bypass. However very little has been reported about conversion to cardiopulmonary bypass (CPB) during attempted OPCAB. The aim of this study was to evaluate outcomes and predictors of emergency conversions to CPB during attempted OPCAB.

METHODS: From January 1999 to July 2002, 1678 consecutive isolated CABG were performed at single institution with intention to treat all patients with OPCAB. Fifty (2.97%) needed urgent conversion to CPB. All the pre, intra and postoperative variables were collected and analyzed in accordance with previously reported model. Multivariate regression analysis was performed to determine predictors for conversion.

RESULTS: In hospital mortality and major morbidity was significantly higher in the converted group compared to OPCAB patients (table). The annual incidence of conversion decreased from 4.8%(n=16), 3.8%(n=19), 2.6%(n=13) and 0.7%(n=2) for 1st to 4th year of experience respectively. None of the preoperative variables were independent predictors of conversion on multivariate regression analysis. There was significant reduction in incidence of conversion after routine use of cardiac positioning device for performing lateral and inferior wall grafts (4.2%:n=27 vs 2.3%:n=23-p=.04).

Outcomes	OPCAB without conversion	Emergency Conversion to CPB	P Value
In-hospital Mortality	1.47%(24)	12%(6)	0.001
Stroke	1.1%(18)	6%(3)	0.022
Deep sternal wound infection	1.54%(25)	8%(4)	0.009

Renal failure	1.23%(20)	6%(3)	0.028
Respiratory failure	3.75%(61)	28%(14)	<0.0001
Freedom from complications	91.09%(1483)	64%(32)	<0.0001

CONCLUSIONS: As emergency conversion to CPB during attempted OPCAB results in significantly higher morbidity and mortality, studies comparing OPCAB to conventional CABG should include converted patients in OPCAB group. In our experience urgent conversion is an unpredictable event. The incidence of conversion decreases with increasing experience of surgeons in performing OPCAB and use of cardiac positioning device.

**By Invitation*

10. Left Ventricular Reconstruction: Early and Late Results

Lynda L Mickleborough, Naaem Merchant*, Joan Ivanov*, [1]Vivek Rao*,

Susan Carson*, Toronto, ON, Canada.

Discussant: Lorenzo A Menicanti

OBJECTIVE: In patients with coronary disease and poor LV function, ventricular reconstruction (VR) combined with revascularization is a surgical option. Details of patient selection and optimal surgical technique are still debated. This study reports results achieved with VR in 279 patients who had akinesia or dyskinesia associated with wall thinning.

METHODS: Data were prospectively collected. Reconstruction on the beating heart was accomplished using a modified linear closure plus septoplasty when indicated. Preop 232 (83%) were in symptom class 3 or 4 with CHF in 171(61%), angina in 156(56%), VT in 107(38%). Average EF was 27±10% and 138(49%) had preop MR ‰¥2+. Operative procedure included CABG 256(92%), septoplasty 61(22%), VT ablation 100(36%) and mitral valve procedure 5(2%).

RESULTS: OR mortality was 2.9%. Perioperative support included IABP 48(17%) and inotropes 151 (54%). During mean followup 64±47 months, 8 patients required transplantation (interval of 49±41 months) 2 MVR and 9 AICD for VT. At 1,5 and 10 years actuarial survival was 92%, 82% and 61%. Freedom from sudden death was 99%, 99% and 94%. Among survivors, symptom class improved in all but 45(16%) mean improvement 1.4±0.9 functional class per patient. Average increase in EF postop was 9±11%

CONCLUSIONS: Using wall thinning as a criteria for patient selection, LV reconstruction can be performed with low operative mortality, provides good control of symptoms, excellent long-term survival and freedom from sudden death. This approach should be considered in all patients with coronary disease, poor LV function and wall thinning.

[1]2003-05 Research Scholar

**By Invitation*

11. Optimal Timing of Cardiac Transplantation after Ventricular Assist Device Implantation

James S Gammie*, Leah B Edwards*, Bartley P Griffith, Richard N Pierson III,

Lana Tsao*, Baltimore, MD; Richmond, VA; Boston, MA.

Discussant: Francis D Pagani

OBJECTIVE: Timing of cardiac transplantation after ventricular assist device (VAD) implantation is influenced by donor organ availability and the clinical status of the recipient. In October 1999, UNOS allocation criteria were revised. A patient with a VAD is considered status 1A for the first 30 days after implantation and status IB thereafter. Device-related complications after 30 days permit an upgrade to status lA(b). A policy change under consideration would permit listing as a status 1A for a 30 day period to start at the discretion of the treating physician. We have examined UNOS/OPTN data to examine the effect of time between VAD implantation and cardiac transplantation on survival after transplantation.

METHODS: UNOS data on 2692 adult heart transplants in the United States between 10/99 and 3/01 were reviewed. Post-transplant survival rates were computed using the Kaplan-Meier method; comparisons were performed with the log-rank test. A multivariable proportional hazards model was developed to determine factors affecting post-transplant survival.

RESULTS: Seventeen % (471/2692) of the population were supported with a VAD at the time of transplantation. VADs were present in 24 % (471/1986) of status 1 recipients. At the time of transplantation, 36 % (170/471) were status lA(a) (VAD-flrst 30 days), 48 % (225/471) were status 1A(b) (VAD > 30 days with complications) and 16 % (76/471) were status 1B(a) (VAD > 30 days). One-year survival after cardiac transplantation was 78.6 % for status 1A(a), 76.4% for status 1A(b), and 91.5 % for status 1B(a) recipients. One-year survival for status 2 transplant recipients was 86.5 %. Data on the interval from VAD implantation to transplantation were available for 72 % (340/471) of patients (Table). Multivariate modeling demonstrates a significant independent effect of the time interval from implantation to transplantation on post-transplant mortality, with a rapid drop in the relative risk in the first 30 days after implantation and a window of lowest relative risk between 30 and 70 days after implantation.

Time implant to transplant	n	1 month survival (%)	1 year survival (%)
0-2 weeks	60	84.9	73.0*
2-4 weeks	58	96.6	83.8
4-6 weeks	73	97.2	92.4
6-12 weeks	35	88.2	85.2
12 weeks-6 months	72	94.2	83.9
> 6 months	42	83.1	75.8

* p = 0.02 for 0 - 2 weeks versus > 2 weeks

CONCLUSIONS: Survival after cardiac transplantation is influenced by the time interval from VAD insertion to transplantation. Survival is significantly worse when transplantation is performed in the first two weeks after VAD implantation. Mortality risk is lowest between 30 and 70 days after implantation. Allocation policies that maximize donor organ availability during a window of lowest relative risk for post-transplantation mortality are likely to result in the most efficient use of donor hearts.

*By Invitation

12. LVAD Destination Therapy: An Extended Follow-Up of Outcomes

Soon J Park, O H Frazier, William Piccioni Sr.*, Edward Raines*, William Holman*, Eric Rose, [1]Mehmet Oz, Annetine Gelijns*, Nuala Ronan*, Satoshi Furukawa*, Walter Dembitsky, Minneapolis, MN; Houston, TX; Chicago, IL; Lincoln, NE; Birmingham, AL; New York, NY; Philadelphia, PA; San Diego, CA.

Discussant: Stephan Westaby

BACKGROUND: A prospective randomized trial (REMATCH) comparing Optimal Medical Management (OMM) v. LVAD as a long-term support for patients with end stage heart failure showed promising results for LVAD patients at the end of enrollment period. Since then, OMM patients were allowed to cross over to surgical therapy, and we have continued to follow all patients until their death. Extended follow-up of this unique cohort could provide important insight into benefits (survival and quality of life) and risks of LVAD application as destination therapy device.

METHODS: Sixty-eight patients were randomly assigned to LVAD therapy as compared to 61 patients to Optimal Medical Management (OMM) between May 1998 and June 2001. An independent morbidity and mortality committee adjudicated all events. Survival was analyzed by the product-limit method of Kaplan and Meier. The frequency of adverse events was analyzed by means of Poisson regression. The "close" date of the adjudicated data was 6/20/02.

RESULTS: Survival rates for LVAD patients were 51% (95% CL; 40-63%) at one year and 26% (95% CL 14-38%) at two years according to the intention-to-treat analysis. However, 19 were living on LVAD support out of the total 21 survivors in the trial. (Included are 3 of 5 OMM survivors who crossed-over to surgical therapy.) Minnesota Living with Heart Failure scores (MLHF, 0-105) improved significantly from 75 (+/-18) at baseline to 42 (+/-20) at 1 year and 37(+/-14) at 2 years. Patients were clinically depressed at baseline [Beck's Depression Inventory (BDI) 19 (+/-9)], but their BDI scores became normal while being supported on LVAD, 9 (+/-9) at 1 year and 7 (+/-7) at 2 years. The rate of nonspecific neurological dysfunction (events/patient-year) was 0.44, and the thromboembolism rate was 0.1. Freedom from device failure was 100% at 1 year and 85% at 2 years. LVAD malfunction, typically manifested by device alarms was 0.87, while the rate of LVAD system failure was 0.06. LVAD inflow/outflow insufficiency rate was 0.93, and the rate of infection at driveline/pocket was 0.35.

CONCLUSIONS: Our extended follow-up data seem to support that LVAD therapy renders significant survival and quality of life benefits as compared to OMM for patients with end stage heart failure. Also, our reported incidences of adverse events and device failures could serve as benchmarks for future trials testing new management approaches and new devices for destination therapy.

3:25 p.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

[1]1994-96 Research Scholar

*By Invitation

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

Ballroom, Hynes Convention Center

Moderators: Timothy J Gardner
O Wayne Isom

13. The Long-Term Outcome of Patients with Coronary Disease and Atrial Fibrillation Undergoing the Cox-Maze Procedure

Sydney L Gaynor*, Sunil Prasad*, James L Cox, John P Boineau*, Richard B Schuessler*, Ralph J Damiano, Jr., Saint Louis, MO
Discussant: Joseph A Dearani

OBJECTIVE: The Cox-Maze procedure has been used to treat patients with lone atrial fibrillation (AF) and AF associated with vavular disease with excellent long-term success. However, it is unknown whether treating permanent AF concurrently in patients with coronary artery disease (CAD) undergoing coronary revascularization (CABG) would have similar results. This study evaluated the late results of 47 consecutive patients undergoing both a Cox-Maze procedure and coronary revascularization.

METHODS: From 1992 to 2002, 47 consecutive patients with AF underwent CABG with a Cox-Maze procedure. All the procedures were performed with cardiopulmonary bypass. Complications included renal failure, reoperation for mediastinal bleeding, mediastinitis, neurological events, postoperative myocardial infarction, and balloon pump insertion. Patient follow-up was conducted by questionnaire and electrocardiographic documentation.

RESULTS: Mean duration of preoperative AF was 8.2 ± 6.9 years. Perioperative results are shown in table below. Long-term follow-up was obtained in 94% (44/47) of patients. There were 98% (43/44) of patients free of AF with a mean follow-up time of 5.5 ± 2.8 years. There was no difference in long-term outcomes in patients with paroxysmal AF (PAF) or persistent AF (PTAF) (p=0.3). There were no perioperative or late strokes.

CONCLUSIONS: The Cox-Maze procedure is safe and effective in patients who have AF and CAD. This procedure returns the great majority of patients to normal sinus rhythm and eliminates the risk of stroke in this high-risk population. These data suggest that all patients with AF undergoing coronary surgery should be considered for a Cox-Maze procedure.

**By Invitation*

14. Separated Graft Technique and En Bloc Technique for Arch Vessels Reimplantation During Surgery of the Aortic Arch: A Retrospective Comparative Study

T Keavi*, Marco Di Eusanio*, Marc Schepens*, Wim Morshuis*, Karl Dossche*, Teruhisa Kazui*, Kazuhiro Ohkura*, Naoki Washiyama*, Roberto Di Bartolomeo*, Davide Pacini*, Angelo Pierangeli*, Ancona, Italy; Nieuwegein, Netherlands; Hamamtsu, Japan; Bologna, Italy.

Discussant: Axel Haverich

OBJECTIVE: To compare the results of the separated graft technique and the en bloc technique as a method of arch vessels reimplantation during surgery of the aortic arch. To determine predictive risk factors associated with hospital mortality and adverse neurologic outcome during aortic arch repairs.

METHODS: Between October 1995 and March 2002, 352 patients (mean age 64.9± 11.3 years; urgent status: 49/352; 13.9%) underwent surgery of the aortic arch using the separated graft technique (Group A: n=230; 653%) and the en bloc technique (Group B: n=122; 34.7%)to reimplant the arch vessels. An aortic arch replacement was performed in 32 patients (9.1%), an ascending aorta and arch replacement in 222 patients (53.1%), an aortic arch and descending aorta replacement in 16 patients (4.5%) and a complete replacement of the thoracic aorta in 82 patients (23.3%). Brain protection was achieved by means of Antegrade Selective Cerebral Perfusion (ASCP) in all cases. The mean Cardio Pulmonary Bypass (CPB) time was 204.8±61.9 minutes (group A: 199.7±57.0 min; group B: 214.5±69.4 min; p= 0.033), the mean myocardial ischemic time was 121.5±43.2 minutes (group A: 116.7±38.9 min; group B: 130.80±49.4 min; p=0.003), the mean ASCP time was 84.5±36.4 (group A: 91.3±36.3 min; group B: 70.6±32.7 min).

RESULTS: Overall hospital mortality was 6.8% (group A: 6.5%; group B: 7.4%; p=ns). The Permanent Neurological Dysfunction (PND) rate was 3.5% (group A: 4.0%; group B: 2.5%; p=ns). The Transient Neurological Dysfunction (TND) rate was 5.4% (group A: 5.5%; group B: 5.2%, p=ns). Postoperative systemic morbidity was similar in the two groups (table 1). A logistic regression analysis revealed preoperative cardiac tamponade (p=0.035; OR=7.1) to be independent predictor of hospital mortality. None of the analysed preoperative variables were associated with an increased risk of PND. CABG (p=0.001; OR=14.1) and CPB>240 minutes (p=0.013;OR=3.9) were indicated as independent predictors of TND by logistic regression.

Results	All patients; n(%)	Group A; n(%)	Group B; n(%)	P
Hospital mortality	24(6.8)	15(6.5)	9(7.4)	ns
Permanent neurologic dysfunction	12/345(3.5)	9(4.0)	3(2.5)	ns
Transient neurologic dysfunction	18/333(5.4)	12(5.5)	6(5.2)	ns
Postop dyalisis	16(4,5)	10(4.4)	6(5.0)	ns
Postop respiratory insufficiency	61(17.6)	41(18.1)	20(16.7)	ns
Postop myocardial infarction	4(1.2)	2(0.9)	2(1.7)	ns
Bleeding requiring rethoracot-omy	32(9.7)	18(8.3)	14(12.6)	ns

CONCLUSIONS: ASCP confirmed to be a safe method of cerebral protection allowing complex aortic arch operations to be performed with acceptable results in terms of hospital mortality and neurologic outcome. The separated graft technique had no adverse impact on hospital mortality and morbidity.

**By Invitation*

15. Aortic Root Surgery in Marian's Syndrome: Comparison of Aortic Valve Sparing ReimPlantation Versus Composite Grafting

Matthias Karck*, Klaus Kallenbach*, Christian Hagl*, Thorsten Walles*, Christine Rhein*, Rainer G Leyh*, Axel Haverich, Hannover, Germany.

Discussant: Tirone E David

OBJECTIVE: To compare long term results of two methods of aortic root repair in patients with Marfan's syndrome.

METHODS: Between 3/79 and 4/02, 121 patients with clinical evidence of Marian's syndrome underwent either Composite graft replacement (C) of the aortic valve and ascending aorta (n=76) or aortic valve reimplantation (AYR) (n=45). Underlying etiology was aortic dissection type A in 47, seventy-four were operated due to aneurysms of the ascending aorta.

RESULTS: Patients with AVR were younger, when compared to patients with C (28 vs. 35 yrs, p=.002), had longer intraoperative aortic cross clamp times (127 vs. 79 minutes, p<.001) and extracorporeal circulation times (164 vs. 123 minutes, p<.001). Early postoperative mortality was 5.2 % (n=4) in patients with C and 0% in patients with AVR (p=n.s.). Mean follow up was 32 months for patients with AVR, and 109 months for patients with C. At five years follow up, survival was comparable between groups (86% for C vs. 91% for AVR; p=n.s.) as well as freedom from reoperation (95% vs. 84%, p=n.s.)(all corrected for cohort size and time of follow up). During follow up, there were 15 bleeding or thromboembolic events among patients with C when compared to one event in patients with AVR (p=.043).

CONCLUSIONS: Early postoperative outcome, long term survival and incidence of reoperation are comparable after aortic valve reimplantation versus composite grafting in Marfan patients. The lower incidence of thromboembolic or bleeding complications favor aortic valve sparing reimplantation in these patients.

5:00 p.m. ADJOURN

**By Invitation*

MONDAY AFTERNOON, MAY 5, 2003

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

(8 minutes presentation, 12 minutes discussion

Room 302, Hynes Convention Center

Moderators: Joel D Cooper
Larry R Kaiser

16. Twenty Year Experience of Lung Transplantation at a Single Center: Influence of Recipient Diagnosis on Long-Term Survival

Marc de Perrot*, Cecilia Chaparro*, Karen McRae*, 'Thomas K Waddell*, Denis Hadjiliadis*, Lianne G Singer*, Andrew F Pierre*, Michael Hutcheon*, Shaf Keshavjee, Toronto, Canada.
Discussant: Thomas M Egan

OBJECTIVES: To examine the long-term patient outcomes of lung transplantation in a single center.

METHODS: Between 1983 and 2002, 471 lung transplants were performed in 454 patients. With experience, the indications for lung transplantation for end stage pulmonary failure have continued to expand and evolve. Major indications were: cystic fibrosis (CF, n=115), chronic obstructive pulmonary disease (COPD, n=87), alpha-1 anti-trypsin deficiency (AAT, n=51), idiopathic pulmonary fibrosis (IPF, n=86), primary pulmonary hypertension (PPH, n=32), Eisenmenger¢s syndrome (ES, n=19), and miscellaneous end-stage lung diseases including selected malignancies (n=64). Bilateral lung transplant was performed in 380 cases (80.7%), including 17 re-transplants (3.6%), single lung transplant in 83 (17.6%), and heart-lung transplant in 8 (1.7%).

RESULTS: The 5-, 10-, and 15-year actuarial survival rates for all recipients were: 55.2% (95% confidence interval: ±5.2%), 33.9% (±6.5%), and 26.2% (±11%), respectively. The most common causes of death were: sepsis (n=75, 34.9%), obliterative bronchiolitis (OB, n=69, 32.6%), cancer (n=22, 10.2%), cardiac event (n=14, 6.5%), and primary graft failure (n=12, 5.6%). Despite an increased early postoperative mortality rate, patients with PPH achieved the best sustained long-term survival (72.4% at 3 months, 68.8% at 1 year, 54.4% at 5 years, and 54.4% at 10 years). Fourty-four CF patients infected with Burkholderia cepacia (B cepacia + ve group) experienced a low overall survival rate (69.8% at 3 months, 62.7% at 1 year, 32.6% at 5 years, and 17.4% at 10 years), whereas the71 CF patients without Burkholderia cepacia infection (Bcepacia -ve group) survived longer (92.5% at 3 months, 87.4% at 1 year, 77.4% at 5 years, and 46.9% at 10 years; $p<0.0001$ when compared to B cepacia +ve group). Sepsis was the main cause of death in the B cepacia +ve group (63% of death vs 24% in the B cepacia -ve group, $p=0.005$). The 10-year survival rate was significantly better in patients with COPD (47.3%) than in patients with AAT (22.3%, $p=0.02$) or IPF (21.7%, $p=0.01$). The cause of death was predominantly OB in COPD patients (53% of deaths vs 23% in IPF and 15% in AAT, $p=0.002$), whereas sepsis predominated in AAT and IPF patients (45% and 29% of deaths, respectively, vs 9% in COPD, $p=0.005$).

CONCLUSIONS: While OB and sepsis still limit the durability of the benefit, lung transplantation returns many patients with end stage lung disease to active and productive lives. Differences in the complications and long term survival show the important contribution of the recipient diagnosis to the success of lung transplantation.

[1]2000-02 Research Scholar

**By Invitation*

17. Prolonged Survival in Patients with Resected Non-Small Cell Lung Cancer and Single Level N2 Disease

Steven M Keller*, Mark G Vangel*, Henry Wagner*, Joan H Schiller*, Arnold M Herskovic*, Ritsuko Komaki*, Randolph S Marks*, Michael C Perry*, Robert B Livingston*, David H Johnson*, Bronx, NY; Boston, MA; Tampa, FL; Madison, WI; Arlington Heights, IL; Houston, TX; Rochester, MN; Columbia, MO; Seattle, WA; Nashville, TN.

Discussant: Garrett L Walsh

OBJECTIVE: In order to test the hypothesis that patients with NSCLC and single N2 level metastases constitute a favorable subgroup of patients with mediastinal metastases, we analyzed the results of the Eastern Cooperative Oncology Group 3590 (a randomized prospective trial of

adjuvant therapy in patients with resected stages II - IIIa NSCLC) by site of primary tumor and pattern of lymph node metastases.

METHODS: Accurate staging was assured by mandating either systematic sampling or complete dissection of the ipsilateral mediastinal lymph nodes. Overall survival of patients with left lung NSCLC and metastases to only one of lymph node levels 5,6 or 7 and right lung NSCLC with metastases to only one of levels 4 or 7 was compared to that of patients with N1 disease originating in the same lobe.

RESULTS: Median survival of the 172 patients with single level N2 disease was 35 months versus 66 months for the 143 patients with N1 disease (P = .01). Median follow-up was 75 months.

Median Survival - months

Lobe of Primary Tumor (#pts N1/N2)	N1	N2	Risk Ratio (95% Cl)	P value log-rank
Left Upper (43/49)	49	51	1.07(0.64-1.79)	0.80
Left Lower (28/28)	56	27	0.50 (0.28-0.92)	0.02
Right Upper (47/61)	85	36	0.48(0.28-0.81)	0.005
Right Lower (25/34)	45	28	0.71(0.39-1.31)	0.27

CONCLUSIONS: The presence of metastases in a single N2 level does not preclude prolonged survival. Survival of patients with left upper lobe NSCLC and metastases to single N2 level lymph nodes is not significantly different from that of patients with N1 disease. Confirmation of the improved survival of patients with left upper lobe tumors and single level N2 disease would suggest the need to modify the current NSCLC staging system.

**By Invitation*

18. Results of the American College of Surgeons Oncology Group Trial Z0050: The Utility of PET in Staging Patients with Potentially Operable Non-Small Cell Lung Carcinoma

Carolyn E Reed, David H Harpole, K E Posther*, S H Jung*, Robert J Downey*, Bryan F Meyers*, Valerie W Rusch, Barry A Siegel*, Charleston, SC; Durham, NC; New York, NY; St. Louis, MO.

Discussant: Robert J Cerfolio

OBJECTIVE: The accurate staging of NSCLC is the critical basis for patient treatment, prognosis and data acquisition. The benefit of adding PET to standard routine staging (CT of chest/ upper abdomen, CT or MRI of the brain, and bone scintigraphy) in potentially operable patients is not certain. ACOSOG Z0050 is a prospective multi-institutional trial with the primary objective of validating the utility of PET scanning in patients found to be surgical candidates by standard imaging procedures.

METHODS: Patients with the presumptive diagnosis of surgically resectable NSCLC (clinical stage I-IIIA) were enrolled after informed consent and completion of the above staging procedures. As of 10/01/02, complete data were available on 175 eligible patients of a planned population of 235. The mean age was 66 years with 101 men and 74 women. Data were analyzed by T stage, N stage, and M stage.

RESULTS: For tumor status, PET correctly identified 142/150 as malignant (sensitivity 95%) and 13/25 as benign (specificity 52%). PET identified 7/141 presumed MO patients as Ml (5%). For N stage, the accuracy of CT alone, PET alone, and combined CT + PET is shown for the 147 patients with NSCLC in the table.

Confirmed Pathology	CT	PET	Combined CT + PET	P-value
N0	78/88(89%)	70/88(80%)	77/88(88%)	p>0.5
N1	4/23(17%)	8/23(35%)	9/23(39%)	0.085
N2/3	17/36(47%)	24/36(67%)	23/36(64%)	0.035

CONCLUSIONS: Although final data analysis (n=235) will be completed by 3/1/03, several important findings are evident: 1. We confirm that PET is highly sensitive in identifying a lesion as malignant but not helpful in substantiating benign disease in patients with presumed NSCLC. 2. PET does not add value if the CT scan reveals NO status. 3. Neither CT nor PET accurately identifies N1 disease. 4. PET may be more accurate than CT in identifying N2/N3 disease. 5. The utility of routine full-body PET may be questionable in this potentially surgically resectable cohort, as it only identified Ml disease in an additional 5% of patients after routine staging.

2:45p.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

**By Invitation*

3:20 p.m. SIMULTANEOUS SCIENTIFIC SESSION -GENERAL THORACIC SURGERY

Room 302, Hynes Convention Center

Moderators: Joel D Cooper
Larry R Kaiser

19. Lung Cancer Resection Combined with Lung Volume Reduction in Patients with Severe Emphysema

[1]Cliff K Choong*, Bryan F Meyers*, [2]Richard J Battafarano*, Tracey J Guthrie*, Gail Davis*, G Alexander Patterson, Joel D Cooper, St. Louis, MO.

Discussant: Larry R Kaiser

OBJECTIVE: Certain patients with resectable lung cancer and severe respiratory limitation due to emphysema may have a suitable operative risk by combining cancer resection with lung volume reduction surgery (LVRS). The purpose of this study is to review our experience with such patients.

METHODS: Between 1994 and 2001, 21 patients with clinically resectable early stage lung cancer in the setting of severe emphysema underwent an operation designed to provide complete cancer resection and a volume reduction effect. The degree of emphysema destruction, as determined by radiologic imaging and objective functional testing in these patients, would ordinarily make them ineligible or at very high risk for a surgical resection of their cancer.

RESULTS: In the 21 patients, the mean age was 66.2 ± 6.7 years. The mean preoperative FEV_1 was 0.7 ± 0.2 liters (29% predicted), residual volume was 5.5 ± 1.0 liters (271%), and DLCO was 8.0 ± 2.2 ml/min/mm Hg (34% predicted). Oxygen supplementation was required continuously in 57% of patients and in 86% during exercise. In 9 patients, the cancer was located in a severely

emphysematous lobe and the volume reduction component of the procedure was accomplished by an anatomic lobectomy. In the remaining 12 patients, the cancer resection lobectomy (n=9) and wedge resection (n=3) was supplemented with additional LVRS. This additional resection was ipsilateral in 3, contralateral in 5, and bilateral in 4 patients. Final pathologic staging was stage I in 16 patients, II in 2 patients, III in 2 patients. One patient was found to have stage IV disease due to multifocal tumors in separate lobes. There were no hospital deaths. All but one patient were extubated immediately following the procedure. Postoperative complications included prolonged air leak (>7 days) in 11 (52%); atrial fibrillation in 6 (29%); and re-intubation for ventilatory assistance in 2 (9%) patients. All patients showed an improvement in measured lung function postoperatively. Survival and functional improvement are shown in the table below.

	Pre-Op	6 Months Post-Op	1 Year Post-Op	3 Years Post-Op	5 Years Post-Op
N	21	21	20	14	7
FEVi L (% Predicted)	0.7 ± 0.2 (29%)	1.0 ± 0.3 (40%)	1.1 ± 0.4 (43%)	0.9 ± 0.2 (34%)	0.8 ± 0.2 (28%)
RV L (% Predicted)	5.5 ±1.0(271%)	3.2 ± 0.9 (152%)	3.4 ± 0.6 (161%)	3.6 ± 0.7 (163%)	4.1 ± 1.2 (195%)
Six-Minute Walk (ft)	906 ±354	1240 ± 171	1292 ± 171	1171 ± 113	1101 ±126
Actuarial Survival	------	100%	100%	73.7%	62.7%
Freedom From Recurrence	------	95.2%	90.5%	84.0%	68.7%

CONCLUSIONS: Patients with severe emphysema and a resectable lung cancer who have a favorable anatomy for LVRS may undergo a combined cancer resection and LVRS with an acceptable risk and good long-term survival.

[1]2002-03 Graham Fellow

[2]2001-03 Research Scholar

**By Invitation*

20. Respiratory Complications after Pneumonectomy. An Analysis of Incidence, Risk Factors and Outcome.

Nicolas Aubree*, Jocelyn Gregoire*, Louis F Jacques*, Michel Piraux*, Liu Guojin*, Yves Lacasse*, Jean Deslauriers, Ste-Foy, QC, Canada.

Discussant: Shaf Keshavjee

OBJECTIVE: Pneumonectomy is associated with an increased incidence of postoperative cardiopulmonary events. Among them, respiratory complications are the most serious and a major cause of postoperative mortality. The objectives of this clinical study were to determine the incidence, risk factors and prognostic implications of these events.

METHODS: A retrospective review of 1045 consecutive patients who underwent pneumonectomy for lung cancer over two decades (1980-2000) was carried out. Respiratory complications occurred in 155 patients (14.8%) and they were classified into bronchial events (n: 39, 3.7%) and pulmonary events (n:116, 11.1%). The records were further analysed to identify possible risk factors. Follow-

up was complete for all patients and dates of death were documented through the Provincial Health Insurance Plan.

RESULTS: Overall operative mortality was 5.3% (55/1045) and for patients with respiratory complications it was 29.7% (46/155). Risk factors and survival figures are shown in the table. If patients who died postoperatively are excluded from analysis, five and 10-year survival rates for those who sustained bronchial or pulmonary complications are 26% and 6% and 25% and 17% respectively.

Incidence, risk factors and survival

	Bronchial events	**Pulmonary events**	**No respiratory complication**
No patients, incidence (%)	39 (3.7%)	116(11.1%)	890 (85.2%)
Mortality (%, No)	7.7% (No=3)	37.1%(No=43)	1%(No=9)
Decade (<1990, >1990)*	N.S.	p<0.001	
Age (<65, >65)*	N.S.	N.S.	
PreopFEV1(<1.5, >1.5)*	N.S.	N.S.	
Side of op. (RT vs LT)*	p<0.001	p<0.001	
pTNM (l-ll vs others)*	N.S.	p=0.05	
Operation (extended vs standard)*	p=0.05	p<0.001	
5 year survival	0.24	0.16	0.33
10 year survival	0.05	0.11	0.23

*: Risk factors with according p value

CONCLUSIONS: In a large study population, the incidence of respiratory complications after pneumonectomy was 14.8%. While right-sided and extended procedures were significant risk factors for all events, respiratory insufficiency occurred much more often during the years 1990-2000 than during the years 1980-1990. For patients who survived a pulmonary event, long term survival was similar to the rest of the cohort. By contrast, a broncho-pleural fistula had a significant negative impact on survival.

**By Invitation*

21. Endotracheal Calcineurin Inhibition Ameliorates Injury in an Experimental Model of Lung Ischemia Reperfusion.

Steven M Woolley*, Alexander S Farivar*, Robert Thomas*, [1]Michael S Mulligan*, Seattle, WA.

Discussant: Robert J Keenan

OBJECTIVE: Calcineurin inhibition has been shown to provide protection in several models of ischemia reperfusion injury e.g. cerebral, gut and hindlimb. VCe have demonstrated in previous studies that the Calcineurin inhibitors cyclosporine A and tacrolimus (FK506) given IV are effective in reducing lung reperfusion injury (LIRI) in our model. Delivering a Calcineurin inhibitor directly into the lung via the airway is a potentially attractive option in donor management. The

purpose of this study was to evaluate the effects of Calcineurin inhibition in LIRI when delivered intratracheally.

METHODS: Left lungs of male Long Evans rats were rendered ischemic for ninety minutes and reperfused for up to four hours. Treated animals received FK506 via the endotracheal tube (ET) at doses of 0.2mg/kg, 0.1mg/kg or 0.025mg/kg 60 minutes prior to ischemia. Injury was quantitated in terms of vascular permeability (^{125}IBSA extravasation). Additional animals treated at a dose of 0.1mg/kg were assessed for tissue neutrophil accumulation (MPO content), and bronchoalveolar lavage (BAL) leukocyte content. BAL fluid was assessed for cytokine and chemokine content by ELISA in a further group of animals treated at this dose. Separate tissue samples were processed for nuclear transcription factors by electromobility shift assay. Serum levels of FK506 were also measured in treated animals.

RESULTS: Left lung vascular permeability was reduced in treated animals in a dose dependent fashion compared to control animals. The reductions in permeability were 63% at 0.2mg/kg, 60% at 0.1mg/kg and 27% at 0.O25mg/kg (0.29 +/- 0.1, 0.32 +/- 0.12,0.58 +/- 0.04 vs 0.79 +/- 0.09) (all p values <0.004). The protective effects of FK506 at 0.1mg/kg correlated with a 47% (0.50 +/- 0.06 vs 0.27 +/- 0.08) reduction in tissue MPO content ($p<0.004$) and marked reductions in BAL leukocyte accumulation. This positively correlated with diminished expression of pro-inflammatory cytokines and chemokines as well as nuclear transcription factors. Serum FK506 levels in treated animals were found to be almost undetectable.

CONCLUSIONS: Calcineurin inhibition by FK506 administered via the ET tube is protective against LIRI in our model. This protection is associated with a decrease in nuclear transcription factors. This route of administration of FK506 broadens its potential clinical use, as it decreases concerns about systemic and renal toxicity, and may prove to be a useful therapy in donors to protect against LIRI.

[1]2002-04 Research Scholar

**By Invitation*

22. Identification of Risk Factors Associated with Atrial Fibrillation after Thoracic Surgery: Prospective Analysis of 2,335Patients

Ara A Vaporciyan*, Arlene M Correa*, David C Rice*, Jack A Roth, W Roy Smythe*, Stephen G Swisher*, Garrett L Walsh*, Joe B Putnam Jr., Houston, TX.

Discussant: Robert J Downey

OBJECTIVE: Atrial fibrillation (afib) remains one of the most common postoperative events after thoracic surgery despite advances in patient selection and perioperative care. Attempts at pharmacologic prevention applied uniformly to all patients undergoing a specific procedure have met with limited success. Identification of risk factors associated with the onset of afib will allow more targeted interventions in the patients with the highest risk.

METHODS: A comprehensive prospective database initiated in January 1998 was used to identify patients undergoing major thoracic surgical procedures (January 1998 through July 2002). Data collection was performed at point-of-contact: at preoperative evaluation, at surgery, and at discharge. All patients undergoing resection of lung, esophagus, chest wall or a mediastinal mass

were included. Univariate and multivariate analysis of factors associated with die development of afib were analyzed.

RESULTS: There were 2335 patients who met inclusion criteria. The overall incidence of afib was 11.6% (271). Categories of disease included primary lung cancer (906), pulmonary metastasis (617), esophageal cancer (285), benign lung disease (133), other intrathoracic metastasis (123), mediastinal tumors (75), mesothelioma (55), chest wall tumors (55), benign esophageal (16) and other (70). Average length of stay was significantly increased in patients with afib (8 days versus 14 days, $p < 0.01$). Univariate analysis of multiple factors (demographics, comorbidities, preoperative therapy, category of disease and procedure) was performed. Ah" significant variables (defined as p £ 0.25) were entered into the multivariate analysis. Significant variables (p £ 0.05) in the multivariate analysis included (relative risk; 95% confidence interval) male gender (1.58; 1.16-2.16), age 60 to 69 (4.58; 2.56-8.23), age > 70 (5.47; 3.04-9.83), diagnosis of benign lung disease (0.17; 0.04-0.72), pulmonary metastasis (0.48; 0.27-0.85), history of congestive heart failure (3.12; 1.19-8.19), history of arrhythmias (2.31; 1.39-3.84), preoperative chemotherapy and radiation (2.12; 1.26-3.56), lobectomy (2.67; 1.68-4.22), bilobectomy (4.62; 1.94-10.99), and pneumonectomy (5.36; 2.91-9.89). Esophagectomy, although significant in univariate analysis, did not reach significance in the multivariate analysis.

CONCLUSIONS: Prospective data collection on a large population of thoracic surgical patients identified multiple factors associated with the occurrence of afib. The association of preoperative chemotherapy and radiation with afib is novel. Preventive therapies in selected populations may reduce the incidence of afib.

**By Invitation*

23. Efficacy of FDG-PET Imaging in Esophageal Cancer

Seth Force*, [1]Richard J Battafarano*, Bryan F Meyers*, Jennifer Bell*, Sara F Hicks*, Joel D Cooper, G Alexander Patterson, St. Louis, MO.

Discussant: Kemp H Kernstine

OBJECTIVE: Recent imaging advances and esophageal ultrasound have improved clinical T staging in esophageal cancer. Accuracy of N and M staging may be enhanced by FDG-PET imaging. This retrospective review was undertaken to evaluate the accuracy of FDG-PET imaging in patients with esophageal cancer referred for surgical evaluation.

METHODS: Between September 1996 and December 2001, 242 consecutive patients with esophageal cancer were evaluated by our General Thoracic Surgery service. All patients received CT and FDG-PET as initial staging investigation for esophageal cancer. FDG-PET images were reported by nuclear medicine physicians who had reviewed CT images in advance. CT and FDG-PET reports were examined for all patients. For resected patients, final pathologic staging was compared to CT and FDG-PET reports.

RESULTS: There were 195 males and 47 females; mean age was 65 years. One hundred and thirty one patients did not undergo surgical resection: metastatic disease in 84 patients (1 discovered at exploration), comorbidity in 41 patients and 6 patients refused surgery. Among the 84 patients with metastatic disease, 18 patients had mediastinal metastases, which were identified only by FDG-PET imaging in 7 patients. In addition, 58 patients had M1b disease, which were identified only by FDG-PET in 25 patients. One hundred and eleven patients underwent resection. For these patients,

in comparison to the final pathology report, CT and FDG-PET sensitivity and specificity for T and N is shown in the table below.

	CT		FDG-PET	
	T	**N**	**T**	**N**
Sensitivity	75%	19%	93%	33%
Specificity	7%	91%	100%	81%

CONCLUSIONS: FDG-PET imaging provides valuable additional information for the staging of esophageal cancer. Although FDG-PET was more sensitive than CT scanning for regional lymph node metastases, it had a high false-positive rate in our experience. FDG-PET is very accurate for the detection of distant metastatic disease.

5:00 p.m. ADJOURN

[1]2001-03 Research Scholar

**By Invitation*

MONDAY AFTERNOON, MAY 5, 2003

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

(8 minutes presentation, 12 minutes discussion)

Room 304, Hynes Convention Center

Moderators: Thomas L Spray
Ross M Ungerleider

24. Incidence of Atrial Arrhythmias After Long-Term Fontan Circulation

Joachim Weipert*, Christian Noebauer*, Christian Schreiber*, Martin Kostolny*, Bernhard Zrenner*, John Hess*, Ruediger Lange, Munich, Germany.
Discussant: Constantine Mavroudis

OBJECTIVE: Intraatrial reentrant atrial tachycardias (IART) can be frequently observed after longtime follow-up in patients with Fontan circulation, but risk factors for their development and therapy are ill defined.

METHODS: Up to Sep. 2002 surgical experience summarises 325 patients with right heart bypass procedures. A subgroup of 162 hospital survivors with tricuspid atresia (TrA, n = 90) and complex malformations (CCM, n = 70) operated on between Jan./1980 to Mai/1995 was studied. Surgical modifications included F.-Bjoerk (n=60), F.-Kreutzer (n=26), lateral tunnel with autologous atrial tissue (n=52) and atrio-pulmonary connection with allograft conduits (n=24). Mean age at the time of Fontan operation was 7.5 ± 6.0 yrs for TrA and 9.3 ± 5.8 yrs for CCM patients. Median Fontan-circulation time was 11.0 yrs (range 1 month to 18.8 yrs) for TrA and 11.8 yrs (range 1 month to 20.2 yrs for CCM patients.

RESULTS: Follow-up is complete for 97% and was closed in Feb. 2002 (1789 patient years). 34 (21.0%) patients died late and IART was seen in three of these patients. 46 (40.0%) survivors were suffering from intermittent or chronic IART. Kaplan-Meyer analysis revealed, that 15 and 20 years

after Fontan operation 65%+ 6% and 16% + 9% of the patients are free from IART. Log-rank test showed no significant difference for the development of IART (neither diagnosis of underlying disease, nor operative modification nor duration of Fontan-circulation) between TrA or CCM patients. Age at Fontan operation was not a risk factor (p=0.86) and the presence of IART did not predict an increased incidence of Fontan failure. Electroanatomical mapping and ablation was performed in 22 patients with IART and was successful in all but two patients.

CONCLUSIONS: Patients with modified Fontan operation exhibit a high incidence of IART with time, regardless of the surgical technique or underlying diagnosis. The presence of IART was without significant influence on late Fontan failure. By means of electrophysilogic therapy IART can be effectively controlled. It has to be shown, that a change in operative technique (e.g. extracardiac conduit) is able to reduce the incidence of IART at long time follow-up.

**By Invitation*

25. Brain MRI Demonstrates that Periventricular Leukomalacia is Common Early After Neonatal Cardiac Surgery

Kristin K Galli*, Robert A Zimmerman*, Gail P Jarvik*, Gil Wernovsky*,

Robert R Clancy*, Lisa M Montenegro*, William T Mahle*, Susan C

Nicolson*, Thomas L Spray, J William Gaynor, Philadelphia, PA; Seattle, WA;

Atlanta, GA; Philadelphia, PA.

Discussant: Ross M Ungerleider

OBJECTIVE: Periventricular leukomalacia (PVL) is necrosis of the white matter adjacent to the lateral ventricles and results from hypoxia/ischemia of immature oligodendroglia. PVL is associated with an increased incidence of developmental delay and attention deficit disorder in infants without congenital heart disease. The incidence of PVL and the risk factors for development of PVL after infant cardiac surgery are not known.

METHODS: MRI of the brain was performed 6 to 14 days after cardiac surgery in 105 neo-nates and infants £ 6 months of age. Cardiac defects were classified as I: 2 ventricles/no aortic arch obstruction, II: 2 ventricles/aortic arch obstruction, III: 1 ventricle/no aortic arch obstruction, IV: 1 ventricle/aortic arch obstruction. Class W patients are those with Hypoplastic Left Heart Syndrome(HLHS) and variants.

RESULTS: The mean age at surgery was 24±42 days. Class I defects were present in 44 patients (42%), Class II in 11 (10%), Class III in 9 (9%), and Class IV in 41 (39%). MRI was performed at 7±3 days after surgery. PVL was present in 45/105 patients (43%). PVL tended to occur more frequently in Class IV patients (66%) and those in whom Deep Hypothermic Circulatory Arrest (DHCA) was utilized (53%). (Table) However, by multivariable analysis only younger age at surgery was significantly associated with PVL, p £ 0.02. PVL was found in 44/82 (54%) of neonates compared to only 1/23 (4%) of infants older than 30 days at surgery. Cardiac diagnosis, gesta-tional age, head circumference at birth, weight at surgery, duration of cardiopulmonary bypass, use and duration of DHCA, and hematocrit during cooling were not predictors of PVL.

	No PVL (n=60)	**PVL (n=45)**
Class 1: 2 Ventricles/No Arch Obstruction	33	11

Class II: 2 Ventricles/Arch Obstruction	7	4
Class III: 1 Ventricle/No Arch Obstruction	6	3
Class IV: 1 Ventricle/Arch Obstruction	14	27
Gestational Age (weeks)	38.6±2.2	38.0±2.3
Birth Head Circumference (%)	40±32	41±30
Age at Surgery (days)	36.9±48.9	8.7±20
Weight at Surgery (kg)	3.83±1.16	3.12±0.93
Duration of CPB(min)	72±40	80±50
Use of DHCA(%) Duration of DHCA(min)	30/60(50%)[36min±18]	34/45 (76%)[42 min ±21]

CONCLUSIONS: PVL is common early (1 to 2 weeks) after cardiac surgery in neonates. Patients with HLHS or variants may be at increased risk. Patients with PVL are likely at increased risk for adverse neurodevelopmental outcomes.

**By Invitation*

26. The Rise and Fall of Plasma Vascular Endothelial Growth Factor Levels during the Single Ventricle Surgical Pathway: One Step Closer to an "Anti-Hepatic Factor"?

Ali Dodge-Khatami*, Narayanswami Sreeram*, Igor I Tulevski*, Bas A De Mol*, J Francois Hitchcock*, Ger B Bennink*, Amsterdam, Netherlands; Utrecht, Netherlands.

Discussant: Pedro J Del Nido

OBJECTIVE: Chronic cyanosis and the cavopulmonary anastomosis (CPA) with exclusion of hepatic flow to the lungs are associated with pulmonary arterio-venous malformations (PAVM), in patients undergoing the single ventricle surgical pathway. An anti-hepatic factor may be implicated in this abnormal angiogenesis, and vascular endothelial growth factor (VEGF) may play a central role.

METHODS: Plasma VEGF levels were measured in 14 children without angiographically detectable PAVMs, during their staged surgical pathway leading to a completion total cavopulmonary connection (TCPC). Samples were taken immediately prior to and then months after CPA without antegrade pulsatile pulmonary flow (n=6), and immediately before TCPC and at least one month thereafter (n=9). Corresponding systemic arterial saturations were correlated with VEGF levels at each time frame.

RESULTS: Pre-CPA plasma VEGF levels rose from a mean of 24.4 +/- 28.3 pg/ml to 112.4 +/- 68.5 pg/ml ($p<0.03$) just prior to completion TCPC (during the CPA physiology interval; n=6), and normalized from 115.7 +/- 116.9 pg/ml to 48.9 +/- 27.1 pg/ml ($p<0.05$) after TCPC (n=9). In two patients there was no reduction in plasma VEGF levels after TCPC. VEGF levels were disproportionately elevated with regards to arterial saturations most notably after CPA (R2 = 0.0O2), suggesting an additional angiogenic stimulus besides cyanosis.

CONCLUSIONS: Plasma VEGF levels fluctuate in an almost predictable manner during the surgical stages of single ventricle physiology, with maximal levels after CPA, and regression to normal after completion TCPC in most instances. High VEGF levels are the most disproportionate to hypoxia after CPA, potentially incriminating the absence of hepatic flow to the lungs as an

abnormal angiogenic stimulus. The role of VEGF, an angiogenic anti-hepatic factor which is perhaps involved in increased PAVM development, may promote the future therapeutic use of anti-VEGF antibodies, and should revive the debate of one and a half ventricle repairs, with regards to PAVM prevention.

**By Invitation*

27. The Influence of Pulmonary Artery Morphology on Results of Surgery for Major Aortopulmonary Collateral Arteries and Complex Congenital Heart Defects

Massimo Griselli*, Simon P McGuirk*, David S Winlaw*, Oliver Stumper*,

Joseph V De Giovanni*, Paul Miller*, Kami Dhillon*, John G Wright*, David

J Barren*, William J Brawn*, Birmingham, UK.

Discussant: Gary K. Lofland

OBJECTIVE: Confluent intra-pulmonary pulmonary arteries can facilitate pulmonary artery reconstruction in patients with complex heart disease and major aortopulmonary collateral arteries (CHD/MAPCAs). The aim of this study was to determine the influence of anatomic variables, including distribution and size of pulmonary blood supply, on outcome.

METHODS: Between 1989 and 2002, 161 patients underwent surgery for CHD/MAPCAs, median age 10.2 months (range 1 day to 47.5 years) at initial operation. We analysed medical records and catheter data retrospectively. We identified three patterns of pulmonary blood flow. Group I: Intrapericardial pulmonary arteries (PAs) Group II: confluent intrapulmonary PAs, Group III: no PAs, MAPCAs only. Surgical procedures included one stage repair without any previous surgery (n=37, 23.0%) and multi stage repair (n=75, 46.6%). 28 (17.4%) await septation, 8 (5.0%) are unseptatable and 13 (8.0%) have died late. Follow up is 97% complete with median follow up of 5.6 years (range, 1 day to 23.4 years).

RESULTS: Early mortality following one stage or multi stage complete repair was 4.5% (n=5). Actuarial survival (±1 SEM) was 90±3% at 1 year, 88±3% at 5 years and 88±3% at 10 years post operation. Actuarial freedom from either reoperation or cardiological reintervention was 76±4% at 1 year, 56±6% at 5 years and 42±8% at 10 years. Surgical strategy (multi stage versus one stage repair) had no influence on early mortality or actuarial survival after completion, and did not affect the likelihood of reintervention and reoperation (p=.07). On multivariate analysis two factors significantly influenced survival: the morphology of the PAs (groups I to III), p=.03, and the calibre of the distal pulmonary vasculature (p=.03). Patients in group HI had the highest risk of death after septation (likelihood ratio 5. 1, p = .03). Notably, patients in group II fared considerably better than group III following initial operation (Actuarial survival at 5 years post operation was 91±6 % and 62±12% respectively, p < .05). Furthermore, a higher proportion of patients in group II achieved septation than those in group III 88% vs 53%,p = .01).

CONCLUSIONS: Contemporary surgical management of CHD/MAPCAs is associated with a good outcome. Current classification systems are based on the presence or absence of intrapericardial pulmonary arteries. We have identified a subgroup of those without intrapericardial PAs but with confluent intrapulmonary PAs and this group has a better outcome than those with MAPCAs only.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

**By Invitation*

3:40 p.m. SIMULTANEOUS SCIENTIFIC SESSION -CONGENITAL HEART DISEASE

Room 304, Hynes Convention Center

Moderators: Thomas L Spray
Ross M Ungerleider

28. Redefining the Impact of Oxygen and Hyperventilation after the Norwood Procedure

Scott M Bradley*, Andrew M Atz*, Janet M Simsic*, Charleston, SC.
Discussant: James S Tweddett

OBJECTIVE: Postoperative management following the Norwood procedure is aimed at optimizing systemic oxygen delivery and mixed venous oxygen saturation. High levels of inspired oxygen (FiO2), and hyperventilation, are commonly believed to cause pulmonary overcirculation, low systemic cardiac output, and decreased systemic oxygen delivery. The aim of this study was to determine the effects of these 2 interventions in postoperative neonates.

METHODS: We prospectively studied the effects of 100% FiO2, and hyperventilation, in 14 neonates (mean age 11 ± 7 days), 1-3 days after a Norwood procedure, while sedated, paralyzed, and mechanically ventilated. After establishing baseline conditions (mean FiO2 = 29 ± 7%, normal ventilation), patients were exposed to each of the 2 interventions in random order, followed by a return to baseline. Hyperventilation was achieved by increasing the ventilator rate. Mixed venous O2 saturation was measured via a transthoracic line in the superior vena cava. Assuming O2 consumption to remain constant, arteriovenous O2 saturation difference was used as an indicator of systemic cardiac output, and Omega (_= Systemic O2 saturation / Systemic - Mixed venous O2 saturation) as an indicator of systemic O2 delivery.

RESULTS: 100% FiO2 produced significant increases in mixed venous O2 saturation and Omega, with no change in arteriovenous saturation difference (Table). The increase in mixed venous O2 saturation occurred in 14/14 patients, and ranged from 4% to 17%. Hyperventilation produced no changes in mixed venous O2 saturation, Omega, or arteriovenous saturation difference.

	Baseline	100% FiO2	Hyperventilation
Mixed Venous O2 Saturation (%)	**43 ± 9**	**54 ± 11***	**43 ± 10**
Systemic O2 Saturation (%)	**79 ± 4**	**90 ± 3***	**80 ± 4**
Arteriovenous O2 Saturation Difference (%)	**36 ± 8**	**36 ± 9**	**37 ± 8**
Omega (Î©)	**2.3 ± 0.6**	**2.6 ± 0.7***	**2.2 ± 0.5**
PCO2 (torr)	**43 ± 6**	**47 ± 8**	**31 ± 4***
pH	**7.43 = .05**	**7.42 ± .04**	**7.56 ± .04***
Mean Arterial Pressure (mmHg)	**52 ± 4**	**52 ± 6**	**50 ± 4**
Common Atrial Pressure (mmHg)	**7** ± 1	**7 ± 2**	**6 ± 1***

FiO2 (%)	**29 ± 7**	**100 ± 0***	**29 ± 5**

Mean ± SD; *P<0.05 vs. Baseline

CONCLUSIONS: This prospective, patient-controlled study in neonates following a Norwood procedure demonstrates that neither high FiO2 nor hyperventilation impair systemic cardiac output, and that high FiO2 improves systemic oxygen delivery. Oxygen may act by increasing pulmonary venous oxygen saturation. Management strategies aimed at minimizing FiO2 and systemic oxygen saturation, and carefully controlling ventilation, do not appear to be warranted.

**By Invitation*

29. New Approach to the Surgical Management of Multiple Ventricular Septal Defects

Christian Olsson*, James L Wilkinson*, Christian P Brizard*, Uppsala, Sweden; Melbourne, Australia.

Discussant: Edward L Bove

OBJECTIVE: To evaluate a new approach to the management of multiple ventricular septa] defects (VSD). The approach uses 1) intra-operative localization of the VSD with epicardial echocardiography, 2) the signaling of the VSD before aortic cross clamp and 3) a surgically applied custom-made closure device.

METHODS: Retrospective study of nine consecutive patients operated on between January 2000 and August 2002. The multiple ventricular septal defects were exactly localized intra-operatively with epicardial echocardiography. The defects were then pinpointed with a guidewire traversing the defects from right to left inserted directly through the right ventricular anterior wall, with the heart beating and whilst on normothermic bypass. After cross clamp, the mid muscular or anterior muscular defects were approached through a standard right atriotomy. The apical defects were approached through a small apical right ventricular incision. The defects were closed using a custom-made, multilayered double patch device sandwiching the septum and covering one or several defects.

RESULTS: The median age and body weight at repair were 1 year 4 months (range, 1 week -8 years 3 months), and 9-8 kg (2.8 - 23.9 kg), respectively. Five patients had undergone at least one previous sternotomy and had pulmonary artery banding. The four others were neo-nates. Eight (89%, 95% CI 55 to 99%) had associated cardiac lesions : (transposition of the great arteries (TGA) n=2, TGA with left outflow obstruction n=1, truncus n=1, atrioventricular septal defect n=1, coarctation n=3). Three patients had a perimembranous VSD patch-closure. Three patients had 2 devices, 6 had one device and one had none. No left ventriculotomies were used. Closure of the multiple septal defects was successful in eight patients (89%, 55 to 99%). Due to failure of localizing all defects, one neonate patient had a pulmonary artery band. The residual anterior septal defect was closed with a percutaneous device six months later. No other patient required a reoperation for residual shunting. With 70 patient-months follow-up (median 6 months, range 1-19), there was no early or late mortality, all patients are free of cardiac medications, and no significant residual left-to-right shunts have been demonstrated in any patient with echocardiographic studies at follow up (n = 8) or with catheter study (n=1).

CONCLUSIONS: The reported management of multiple ventricular septal defects appears safe, simple, and effective, including in complex neonatal patients. There was no residual left-to-right shunting requiring re-intervention or medication and no morbidity from the technique.

**By Invitation*

30. Cerebral Oxygenation Decreases, and Somatic Oxygenation Increases, Following Cardiopulmonary Bypass with Continuous Cerebral Perfusion for Stage 1 Palliation of Hypoplastic Left Heart Syndrome.

George M Hoffman*, Eckehard A Stuth*, Robert D Jaquiss, Susan R Staudt*,

Todd J Troshynski*, Patrick L Vanderwal*, James S Tweddell, Milwaukee,

WI.

Discussant: Frank A Pigula

OBJECTIVE: Stage 1 palliation (SIP) of hypoplastic left heart syndrome requires interruption of whole body perfusion. Delayed reflow in the cerebral circulation occurs in neonates following deep hypothermic circulatory arrest. We examined relative changes in cerebral and somatic Oxygenation with a modified perfusion strategy which allowed continuous cerebral perfusion.

METHODS: Nine neonates undergoing S1P for HLHS had tissue Oxygenation continuously measured by frontal cerebral and T10-L2 posterior somatic (renal) reflectance oximetry probes (rSO2, INVOS, Somanetics). Surgery was accomplished using cardiopulmonary bypass (CPB) with whole-body cooling to 15-20 degrees C, and selective cerebral perfusion (SCP) via the innominate artery at flow rate guided by rSO2 for reconstruction of die aortic arch. Data was logged at 1 minute intervals and analyzed using repeated-measures ANOVA.

RESULTS: 5205 minutes of data were available for analysis. rSO2 pre-CPB was 69.5±0.5 vs 63.3±0.5 (cerebral vs. renal, $p<0.001$); during SCP 79.6±2.5 vs. 44.3±2.4 ($p<0.0001$), and post-cpb 54.5±0.3 vs. 75.8±0.3 ($p<0.0001$).

CONCLUSIONS: Cerebral Oxygenation was maintained during SCP. Post-CPB, cerebral oxygen-ation was lower compared to pre-CPB or somatic values. These results suggest that cerebral resistance is increased following hypothermic CPB even with continuous perfusion techniques.

**By Invitation*

31. Favorable Outcome with Heart Transplantation Following Failed Fontan Operation or Cavopubnonary Shunt

Max B Mitchell*, David N Campbell, Mark M Boucek*, Henry M Sondheimer*, David D Ivy*, Biagio Pietra*, Bibhuti Das*, Denver, CO.

Discussant: Carl L Backer

OBJECTIVE: Delayed development of pulmonary vascular disease may impair longterm palliation in patients surviving Fontan operation or cavopulmonary shunt (CPS). Data for outcomes of heart transplantation after failed Fontan or prior CPS are few, and 30-day mortality rates are high (25-45%). Our objectives were to assess the intermediate outcomes of failed Fontan/CPS patients transplanted in our center, and to determine if elevated pulmonary vascular resistance may have contributed to Fontan/CPS failure.

METHODS: Of 219 pediatric heart transplants performed in our center, all patients with failed Fontan/CPS were identified. Outcomes and post-transplant pulmonary vascular resistances (PVR - measured at initial routine surveillance catheterization) were determined. Graft survival was estimated by Kaplan-Meier method (endpoints: death, re-transplantation).

RESULTS: Nineteen patients (14 Fontan, 1 Kawashima, 1 Classic CPS, 1 bi-directional CPS, 2 bilateral bi-directional CPS) underwent heart transplantation. UNOS status was 1A in 5, IB in 7, and status 2 in 7. Four patients had protein losing enteropathy (PIE), and 3 had severe pulmonary arterio-venous malformations (PAVM). Mean prior cardiac operations was 3.3. Mean age at transplant was 11.2 yr. Mean interval from Fontan/CPS to transplant was 4.8 yr. Thirty-day survival was 100%. Hospital survival was 95% (18/19). The 1 hospital death occurred in a patient with PIE on day 179 following subsequent liver transplant. Median ICU and hospital stays were 7 and 10 days. Complications occurred in 10 of 19 patients (53%). PAVM and PLE resolved following heart transplant in all affected survivors. Mean follow-up was 56 months. Two patients were re-transplanted at 54 and 83 months, and there were 2 late deaths at 76 and 94 months. All 4 late endpoints were due to rejection and 3/4 were associated with adolescent noncompliance. Graft survival was 94%, 86%, and 59% at 3, 5, and 7 years respectively. All surviving patients remain in NYHA class I. Median time to initial post-transplant hemodynamic evaluation was 13.6 months. Post-transplant PVR was elevated in 12 of 15 measured patients, and mean PVR was 3.75 Woods Units x m^2 (normal £ 2 Woods Units x m^2).

CONCLUSIONS: Operative mortality and intermediate survival following heart transplantation for failed Fontan/CPS was good, and functional outcomes were excellent. Operative morbidity in these patients is significant. Graft rejection is the major factor limiting long-term outcome. Pulmonary vascular disease is a likely etiologic factor in Fontan/CPS failure.

5:00 p.m. ADJOURN

**By Invitation*

MONDAY AFTERNOON, MAY 5, 2003

1:45 p.m. CARDIAC SURGERY FORUM SESSION

(5 minutes presentation, 7 minutes discussion)

Room 311, Hynes Convention Center

Moderators: Irving L Kron
W. Randolph Chitwood

F1. Ethyl Pynivate Preserves Cardiac Function and Attenuates Infarct Size Following Prolonged Myocardial Ischemia

Y Joseph Woo*, Matthew D Taylor*, Jeffrey E Cohen*, Vasant Jayasankar*, Lawrence T Bish*, Jeffrey Burdick*, Timothy J Pirolli*, Mark F Berry*, H Lee Sweeney*, Timothy J Gardner, Philadelphia, PA.

OBJECTIVE: Myocardial injury and dysfunction following ischemia are mediated in part by reactive oxygen species. Pyruvate, a key intermediate glucose metabolite, is an effective free radical scavenger, however, its use as an antioxidant is limited by instability in solution, Ethyl pyruvate, a lipophilic derivative, is stable in solution and more capable of entering cells. It should function as an antioxidant and may provide substrate for lactate dehydrogenase, generating oxidized NAD for subsequent anaerobic ATP generation by phosphoglycerate kinase. This study sought to evaluate ethyl pyruvate administration as a novel myocardial protection strategy in a rat model of ischemia/reperfusion injury.

METHODS: Wistar rats underwent 30 minute ischemic occlusion of the left anterior descending coronary artery followed by 30 minutes of reperfusion. Immediately prior to both initiation of ischemia and reperfusion, animals received either an intravenous bolus of Ethyl pyruvate in Ringer's solution (n=10) or Ringer's solution as a control (n=8). Cardiac function was analyzed using a left ventricular intracavitary pressure/volume conductance microcatheter. To determine oxidative stress and ethyl pyruvate's antioxidant capacity, myocardial lipid peroxidation was measured in ischemic and non-ischemic regions. Left ventricular infarct size was then assessed by digital planimetry of triphenyltetrazolium chloride staining and reported as a percentage of the entire left ventricle.

RESULTS: See Table 1. Ethyl pyruvate administration significandy preserved myocardial contractility compared to controls. In non-ischemic myocardium, lipid peroxidation was equivalent between groups (Control 40.5±6.4nmol/g vs. Ethyl pyruvate 43.8±6.0nmol/g, p=0.73). Lipid peroxidation was elevated in ischemic myocardium but significandy less so with ethyl pyruvate administration. Ethyl pyruvate also significandy decreased myocardial infarct size.

Table 1	**Control**	**Ethyl Pyruvate**	**p-value**
Ejection Fraction %	20.4 ± 1.6	31.8 ± 2.1	0.004
Maximum LV Pressure, mmHg	69.1 ± 1.7	96.5 ± 4.6	0.0002
Maximum dP/dt, mmHg/sec	2940 ± 192	4695 ± 557	0.039
Ischemic Lipid Peroxidation, nmol/g	92.3 ± 3.1	62.5 ± 4.0	0.001
LV Infarct Size %	25.6 ± 1.5	8.5 ± 0.5	0.0001

CONCLUSIONS: In this novel, preliminary study, ethyl pyruvate, a non-toxic, inexpensive, intravenously administered antioxidant and potential glycolytic substrate significantly preserved cardiac function after prolonged myocardial ischemia. Ethyl pyruvate also effectively attenuated myocardial oxidative injury and infarct size.

**By Invitation*

F2. Immediate Ischemic Preconditioning Based on Somatosensory Evoked Potentials Prevents Spinal Cord Injury Following Descending Thoracic Aorta Cross-Clamping

Ivan Salvador B Contreras*, Luiz Felipe P Moreira*, Gerson Ballester*,

Bernardo A De Monaco*, Carmen Lucia P Lancellotti*, Sergio A Oliveira, Sao

Paulo, Brazil.

OBJECTIVE: Delayed ischemic preconditioning has demonstrated neuroprotective effects in spinal cord ischemia. We investigated the effects of immediate ischemic preconditioning based on somatosensory evoked potentials monitoring in a model of spinal cord injury due to descending thoracic aorta occlusion in dogs.

METHODS: Twenty-one dogs were submitted to spinal cord ischemia, which was induced for 45 minutes by combined cross-clamping of the thoracic aorta immediately after the left subclavian artery and nearby the diaphragm. They were divided in three groups: control group underwent only the aortic cross-clamping (n=7), group A underwent one period of ischemic preconditioning (n=7) and group B three equal periods of ischemic preconditioning (n=7) immediately before the aortic cross-clamping. Ischemic preconditioning periods were determined by somatosensory evoked potentials monitoring. During ischemic preconditioning, aortic occlusion was maintained until the decrease of somatosensory evoked potentials amplitude at less than 60% of its original value and it was followed by a recovery period that was sustained until the return of somatosensory evoked potentials amplitude above that level. Neurologic evaluation was performed by an independent observer according to the Tarlov score at 24,48 and 72 hours of follow-up. The animals were then sacrificed and the spinal cord harvested for histopathology.

RESULTS: Aortic pressures before and after the occluded segment were similar between the groups. Ischemic preconditioning periods corresponded to a mean ischemic time of 3 ± 1 minutes and a mean recovery time of 7 ± 2 minutes. Severe paraplegia was observed in 3 animals in control group, in 4 in group A and in none in group B. Mean Tarlov score of group B was significantly better in comparison to the control group at 72 hours of follow-up (4 vs 2.1, p=0.036). Histopathological examination showed severe neuronal necrosis in the thoracic and lombar gray matter in animals who presented severe paraplegia.

CONCLUSIONS: Immediate repetitive ischemic preconditioning based on somatosensory evoked potentials monitoring protects spinal cord during descending aorta cross-clamping, reducing paraplegia incidence.

**By Invitation*

F3. Regional Low Flow Perfiision Improves Neurological Outcome Following Deep Hypothermia Circulatory Arrest In Neonatal Piglets

Richard J Myung*, Matus Petko*, Alexander R Judkins*, Gregory Schears*,

Richard F Ittenbach*, Robert J Waibel*, William M Decampli*, Philadelphia,

PA.

OBJECTIVE: Neurological injury has been attributed to deep hypothermic circulatory arrest (DHCA). Regional low flow perfusion (RLFP) is used as an alternative to DHCA, but whether RLFP improves neurological outcome following DHCA in neonates remains unknown.

METHODS: Sixteen neonatal piglets underwent cardiopulmonary bypass (CPB). Hematocrit was maintained between 24-28% prior to arrest. Alpha-stat blood gas management was used. Animals were randomized to 90 minutes of DHCA or RLFP at 18°C. RLFP was conducted at 10 cc/kg/min via carotid cannulation. After modified ultrafiltration, animals were recovered then sacrificed at one week. Standardized behavior scores were obtained on postoperative days 1, 3, and 7 by a trained observer (0 = no deficit to 90 = brain death). Histopathologic scores were determined by a neuropathologist based on the percentage of injured neurons in the neocortex and hippocampus by hematoxylin & eosin staining (0 = no injury to 4 = diffuse injury). Both investigators were blinded to treatment group assignment.

RESULTS: There were no significant differences between groups during CPB. Following recovery, neuro-behavior scores were abnormal in 25% of RLFP animals (2/8) vs. 88% of control animals (7/8). RLFP animals significantly outperformed control animals on postoperative day 1. Deficits resolved by postoperative day 7 in all animals except one control. Neuronal injury consisted of clusters of necrotic or apoptotic neurons scattered throughout the cortex and hippocampus consistent with prior studies. Although injury was present in all animals on histopathological examination, there was a trend for less severe injury in the RLFP group.

	Neuro-behavior Score			**Histopathologic Score**
	POD1	**POD 3**	**POD 7**	
RLFP	1.25 ± 2.31	0.0	0.0	1.88 ± 0.99
DHCA	15.25 ± 16.23	7.50 ± 17.32	1.88 ± 5.30	3.00 ± 1.20
	(p<0.008)			(p=0.09)

CONCLUSIONS: RLFP decreases neuronal injury and improves early postoperative neurological function following DHCA in neonatal piglets. Further investigation is required to determine the optimum conditions for RLFP and long-term neuro-behavioral function.

**By Invitation*

F4. Cardiac Xenotransplantation: Continuing Progress in the Laboratory

Christopher G A McGregor M.D.*, Sumeet S Teotia*, Johannes M Schirmer*,

Robert P Frantz*, Henry D Tazelaar*, Randall C Walker*, Guerard W Byrne*,

John S Logan*, Rochester, MN; Princeton, NJ.

OBJECTIVE: Our long-term objective is to achieve preclinical outcomes in the pig-to-baboon heart transplant model that will allow clinical application of cardiac Xenotransplantation (xenotx).

METHODS: A model of heterotopic pig (transgenic for the human complement regulating protein CD46) to baboon cardiac xenotx was used (n=10 transplants). Immunosuppression consisted of FK506, Rapamycin, anti-CD20 monoclonal antibody, corticosteroids and Nex1285(an _-gal PEG polymer). Treatment of putative rejection consisted of intravenous corticosteroids and antithymocyte globulin.

RESULTS: The median duration of functioning xenograft hearts in this study was 76 days (95% CI, 56-92 days). These results represent a major advance in outcomes compared to published data of a median duration of 26 days. Outcomes would have been better had 6 of the 10 recipients not

died from baboon cytomegalovirus (BCMV) infections with functioning xenograft hearts. PCR assays for BCMV confirmed greater quantities of virus in these animals compared to others in the group. Pathology of explanted hearts showed variable degrees of delayed xenograft rejection with excellent preservation of gross cardiac appearance and histology in individual cases at nearly four months. Cellular infiltration of the grafts was notably absent.

CONCLUSIONS: These results represent a major improvement in outcomes following cardiac Xenotransplantation. More extended survival will be possible with better antiviral therapy in this baboon model. Ongoing improvements in outcomes can be anticipated with soon-to-be-available cloned pig donors with the inactivated galactosyl transferase gene.

**By Invitation*

F5. Pretreatment with Phenoxybenzamine Attenuates the Radial Artery's Vasoconstrictor Response to Alpha Adrenergic Stimuli

Joel S Corvera*, Cullen D Morris*, Jason M Budde*, Daniel A Velez*, John D Puskas*, Omar A Lattouf, William A Cooper*, Robert A Guyton, Jakob Vinten-Johansen*, Atlanta, GA.

OBJECTIVE: Although the radial artery (RA) bypass conduit has excellent long-term patency, it exhibits a proclivity to vasospasm. We tested the hypothesis that brief pretreatment of a RA graft with the irreversible adrenergic antagonist phenoxybenzamine (PBZ), attenuates the vasoconstrictor response to the vasopressors phenylephrine (PE) and norepinephrine (NE) compared to currently used papaverine/lidocaine.

METHODS: Segments of human radial artery grafts were obtained after a 30-minute intraoperative pretreatment with a solution containing 20mL heparinized blood, 0.4mL papaverine (30mg/mL), and 1.6mL lidocaine (1 %). The segments were transported to the laboratory and placed into a bath containing Krebs-Henseleit solution and 10,100 or 1000_M PBZ orvehicle. The segments were tested in organ chambers for contractile responses to increasing concentrations of PE and NE (0.5-15_M).

RESULTS: Contractile responses to 15_M PE in Control RA segments averaged 44.2±9.1% of the maximal contractile response to 30mM KCl. Papaverine/lidocaine modesdy attenuated contraction to 15_M PE (32.1±5.9%, $p=0.22$), but 1000_M PBZ completely abolished RA contraction (-7.2±4.4%, $p<0.001$). The effect of 10 and 100_M PBZ on attenuating vasocontraction was intermediate between 1000_M PBZ and papaverine/lidocaine. Responses to 15_M NE in Control RA segments averaged 54.7±7.5% of maximal contraction to 30mM KC1. Papaverine/ lidocaine modestly attenuated contraction response of RA segments (35.6±5.1%, $p=0.04$). In contrast, 1000_M PBZ showed the greatest attenuation of NE-induced contraction (-10.5±2.0%, $p<0.001$).

CONCLUSIONS: A brief pretreatment of the human RA bypass conduit with 1000_M PBZ completely attenuates the vasoconstrictor responses to the widely used vasopressors norepinephrine and phenylephrine. Papaverine/lidocaine alone did not block vasoconstriction to these alpha adrenergic agonists.

**By Invitation*

F6. Higher Bypass Temperature Correlates with Increased White Cell Activation in the Cerebral Microcirculation

Vesa Anttila*, Ikuo Hagino*, David Zurakowski*, Hart G. W. Lidov*, Richard A. Jonas; Boston, MA

OBJECTIVE: Normothermic and tepid (34°) bypass have been used with increased frequency despite concern regarding greater risk of cerebral injury. We hypothesized that microcirculatory disturbances would be greater with higher bypass temperatures particularly with reduced flow rates.

METHODS: Twenty-six piglets (mean weight 12.9+-1.1 kg) had a cranial window placed over parietal cerebral cortex for direct examination of the microcirculation by intravital microscopy. Animals were cooled over 40 minutes to 15°C, 2 5°C, or 34°C on CPB (pH-stat, Hct 20% or 30%, pump flow 100ml/kg/min), followed by 60 minutes of reduced flow (10, 25, or 50ml/kg/ min). Rhodamine-stained leukocytes were observed in post-capillary venules with analysis for adhesion and rolling. Plasma was labeled with Fluorescein-Isothiocyanate-Dextran for assessment of microvascular diameter.

RESULTS: Higher temperature was correlated with a greater number of adherent and rolling leukocytes during the full 60 minutes of reduced flow bypass (Spearman rho: 0.68-0.78, $P<0.001$). Poisson log-linear regression modeling detected significantly more adherent leukocytes at a temperature of 34°C compared to 15°C and at a flow rate of 10ml/kg/min compared to 50ml/kg/min. A significant inverse correlation was found between flow rate and the number of leukocytes at 45 min (rho = -0.47, $P=0.02$) and 60 min (rho = -0.50, $P<0.01$) of a reduced flow period. Arterial vasodilatation was seen throughout the reduced flow period compared to baseline in all groups ($P<0.01$) including the deep hypothermic (15°C) group. Multiple regression analysis indicated that higher temperature and lower flow rate were multi-variable predictors of higher venous lactate levels throughout the reduced flow and rewarming periods ($P<0.01$).

CONCLUSIONS: Higher temperature (34°) as well as low flow rate (10ml/kg/min) are independently associated with increased adherent and rolling leukocytes in postcapillary venules. Microcirculatory disturbances caused by white cell activation may be part of the causative mechanism of neurological injury observed with normothermic bypass.

**By Invitation*

F7. Reduction of Systolic and Diastolic Dysfunction by Retrograde Coronary Sinus Perfusion During Off Pump Coronary Surgery

Manuel Castella*, Gerald D Buckberg, Los Angeles, CA.

OBJECTIVES: To offset potential systolic and diastolic dysfunction (myocardial stunning) following temporary regional ischemia during OPCAB, we evaluated protective effects of retrograde coronary sinus perfusion.

METHODS: Nineteen Yorkshire-Duroc pigs underwent 15 minutes of mid-LAD ischemia in working beating hearts. Seven had no protective measures and twelve underwent aorta-coronary sinus shunting (conventional cannulae) for retrograde perfusion during ischemia. In six, the retrograde shunt was occlusive to avoid backflow right atrial drainage. Systolic dysfunction

(impaired regional shortening), diastolic dysfunction (contraction extending into early diastole) and coronary sinus (CS) nitric oxide and endothelin-1 levels were recorded.

RESULTS: Before ischemia, contraction did not extend into the diastolic interval. During ischemia, paradoxical bulging occurred in all hearts except in the occlusive CS shunt group (16±6% of baseline, $p<0.01$). Sixty minutes after ischemia, systolic segment shortening recovered 36±24% in the un-perfused group vs. 56±20% and 61±14% in the CS shunt groups ($p<0.05$). Diastolic dysfunction (Figure 1), as percentage of diastolic time in contraction, was 38±16% in the non-treated group vs. 22±22% and 9±9% ($p<0.05$) after shunting and occlusive-shunting respectively (figure 1), correlating with 4mmHg LVEDP rise in the ischemic group vs. unchanged in retrograde perfusion groups. Nitric oxide fell 15% without shunting and rose 8% after occlusive CS shunting ($p<0.05$).

CONCLUSIONS: Retrograde coronary sinus perfusion during simulated off-pump coronary revascularization diminishes systolic and diastolic dysfunction. An aortic-coronary sinus shunt is a rapid, recognized approach that can improve myocardial muscle and endothelial safety during off-pump CABG.

3:10 p.m. BREAK

**By Invitation*

3:45 p.m. CARDIAC SURGERY FORUM SESSION

Room 311, Hynes Convention Center

Moderators: Steven F Bolling

Andrew S Wechsler

F8. Pharmacologic Inhibition of Intracellular Caspases Following Ischemia-Reperfusion Attenuates Left Ventricular Remodeling: A Potentially Novel Pathway

William M Yarbrough*, Rupak Mukherjee*, G Patricia Escobar*, Jeffrey A Sample*, Julie E McLean*, Kathryn B Dowdy*, Jennifer W Hendrick*, William C Gibson*, Amy E Hardin*, Joseph T Mingoia*, Patrick C White*, Ann Stiko*, Robert C Armstrong*, Fred A Crawford Jr., Francis G Spinale*, Charleston, SC; San Diego, CA.

OBJECTIVE: Apoptosis occurs with myocardial ischemia-reperfusion (IR) injury and is mediated by proteases called caspases. Recent *in-vitro* studies suggest caspase activation within myocytes causes contractile protein degradation without inducing apoptosis. Thus, caspase activation post-IR may lead to left ventricular (LV) remodeling through two independent processes. The role of caspase activation with respect to changes in LV geometry post-IR remains unclear. Thus, this project applied pharmacologic pan-caspase inhibition (CASPI) to a chronic porcine model of IR.

METHODS: A circumflex snare and sonomicrometry crystals within the area-at-risk were placed in pigs (n=22, 34kg). Geometric measurements at end-diastole (ED) and systole (ES), including LV area (Area) by echocardiography and segmental chord length (Length) by sonomicrometry were

obtained at baseline. Coronary occlusion was instituted for 60min followed by reperfusion and repeated geometric measurements at 7 days. At reperfusion, pigs were randomized to IR only (n=12) or CASPI (n=10, IDN6734, 2mg/kg IV, then 2mg/kg/hr for 24hr) at a dose that achieved desired plasma concentrations (790± 142ng/mL) as predicted by prior pharmacokinetic studies.

RESULTS: Infarct size and 24hr troponin-I values were not significantly different between IR only and CASPI groups (51±8 vs 42±6% and 189±20 vs 152±26ng/mL, respectively, $p>0.10$). At 7 days, ED and ES areas increased from baseline in the IR only group (27±1 vs. 22±1 and 16±1 vs. 10±1cm^2, respectively, $p<0.05$). ED and ES lengths between remote and infarct regions were also increased from baseline (30±2 vs. 28±1 and 26±2 vs. 23±1mm, respectively, $p<0.05$). The effects of CASPI on regional and global LV geometry as a change from baseline are provided in the Figure.

CONCLUSIONS: CASPI reduced LV dilation at 7 days post-IR. Thus, caspase activation may alter LV geometry independent of a commitment to apoptosis and may prove useful in myocardial IR.

**By Invitation*

F9. Pulmonary Arterial Expression of the Hepatocyte Growth Factor Receptor c-Met Switches From Medium to Endothelium After Cavopulmonary Anastomsosis.

Akio Ikai*, R Kirk Riemer*, Xiaoyuan Ma*, Olaf Reinhartz, Frank L Hanley,

V Mohan Reddy, Stanford, CA.

OBJECTIVE: Pulmonary arteriovenous malformation (PAVM) occurs in up to 60% of patients after cavopulmonary anastomosis (CPA). To study the abnormal pulmonary vascular remodeling after the exclusion of inferior vena caval blood independent of reduced pulmonary blood flow, we compared the effects of CPA and pulmonary artery banding (PAB) on lung gene expression. We previously demonstrated by contrast echocardiography in an ovine model that PAVMs develop by 6 weeks after CPA but not after PAB. Hepatocyte growth factor (HGF), a pleiotropic factor with morphogenic, angiogenic, and anti-apoptotic activities, signals via its specific receptor c-Met, to induce the anti-apoptotic factor Bcl-2. We examined pulmonary artery (PA) expression of these genes for their potential role in PA remodeling.

METHODS: Eighteen lambs aged 35 to 45 days were placed into 3 groups: CPA, PAB, and control (n=6/group). In CPA the superior vena cava was anastomosed to the right PA in end to end fashion. PAB was performed to reduce left pulmonary blood flow by 80% of control. Control was a simple right PA clamp for 20 min. Lung was harvested for Western blot, RTPCR, and immunostaining at 2 and 5 weeks after surgery (n=3/group).

RESULTS: Expression of c-Met mRNA after CPA was increased more than 2-fold compared to control or PAB 2 weeks after surgery. Total lung expression of c-Met by Western blot was unchanged. Localization revealed that c-Met, which is localized to subendothelium but not observed in the endothelium of control animals, is expressed only in the endothelial layer after CPA. Localization of Bcl-2 followed the same pattern as c-Met. Local HGF synthesis in the lung was confirmed by RTPCR. Western blot with affinity-purified polyclonal antibodies to HGF_ subunit revealed that sheep lung expresses an unusual ca.10 kDa isoform of HGF, a novel finding we are currently verifying. Paradoxically, the overall level of HGF protein in lung declined by 37%

(mean relative density units: 38;60;60, respectively, n=3) 2 weeks after surgery in CPA compared with control or PAB, but was the same level in all groups by 5 weeks.

CONCLUSIONS: After CPA, PA expression of the HGF receptor c-Met and one of its downstream effectors, Bcl-2, increases in endothelial cells and decreases in the subendothelium. These results indicate that HGF is actively supporting endothelial cell survival through c-Met-stimulated Bcl-2 expression. The promotion of increased endothelial cell survival by HGF is consistent with a role in PA remodeling following CPA, which is not evident in control or PAB lungs. The reduced level of lung HGF protein following CPA may indicate the existence of a circulating factor that regulates local HGF expression.

**By Invitation*

F10. Myocardial Apoptosis Forty-Eight Hours After Brain Death: Implications for Donor Heart Dysfunction and Management

Thomas Yeh Jr.*, Mark McDonald*, Mark J Schroeder*, Stephanie J Webb*,

Steven C Koenig*, Mary Anne Hauck*, Constantine lonan*, Louisville, KY;

Columbia, SC; Fargo, ND.

OBJECTIVE: Brain death injures donor hearts. Severe myocardial injury precludes cardiac donation in the face of a critical organ shortage. Even when hearts are deemed acceptable, lesser degrees of injury impact negatively on donor hearts. We sought to correlate the hemodynamic deterioration after brain death with the presence of apoptosis. A 48 hour time window was employed to mirror the time typically required to coordinate clinical organ donation.

METHODS: An intracranial balloon expansion model of brain death was employed in rabbits. Survivors were maintained for 48 hours. Four out of fifteen rabbits died before 48 hours elapsed (27% mortality). LV and aortic pressures were used to derive heart rate, systolic and diastolic blood pressures, and maximum and minimum LV pressure first derivatives (+dP/dt and -dP/ dt). Cardiectomy was performed to assay for the presence of apoptosis manifested as DNA fragmentation using the TUNEL (terminal d-UTP nick end labeling) assay, and DNA laddering.

RESULTS: As a group, heart rate, LV dP/dt, and blood pressure declined (late deterioration). A brief period of hemodynamic recovery was ultimately not sustained. A percentage of cardiac nuclei from brain dead rabbits were consistent with apoptosis in that they were shrunken and TUNEL positive: right ventricle (RV) 5%, left ventricle (LV) 5%, and septum 7%. PCR-amplification of genomic DNA confirmed the presence of apoptosis (DNA laddering) in these specimens. A higher percentage of nuclei were TUNEL positive, but were morphologically larger and much less dense that the typical pyknotic nuclei associated with apoptosis: LV 25%, RV 27%, septum 30%. Electron microscopy revealed that some of these nuclei were distinctly abnormal: enlarged, with chromatin condensation and grossly deformed nuclear membranes (crenellation).

CONCLUSIONS: Brain death is associated with hemodynamic instability and a marked increase in the number of TUNEL positive nuclei. The subset that are shrunken are consistent with apoptosis. The subset that are larger, with open nuclear architecture have been hypothesized in other models to represent a form of "interrupted apoptosis." Both subsets of nuclei may be important prognostic indicators of donor heart quality, but the larger nuclei may represent an unrecognized therapeutic target that precedes the final commitment phase of apoptosis.

*By Invitation

F11. Pharmacological Platelet Anesthesia by GPIIb/IIIa Antagonist and Argatroban during In Vitro Extracorporeal Circulation

Shinji Kanemitsu*, Isao Yada, Tsu, Japan.

OBJECTIVE: Contact between blood and synthetic surfaces of a cardiopulmonary bypass (CPB) circuit leads to platelet activation, and resultant platelet dysfunction contributes to postoperative bleeding. Temporary pharmacological inhibition of platelets (platelet anesthesia) during CPB is the potential strategy for preserving platelet number and function.

METHODS: A fresh human blood was recirculated in an in vitro CPB model circuit. We tested effects on platelet consumption and functions during in vitro heparinized CPB circulation of various platelet inhibitors including FK633 (a peptide-mimetic GPIIb/IIIa antagonist), abciximab (Fab fragment of antibody against GPIIb/IIIa), AJvW-2 (an anti-von Willebrand factor (vWF) monoclonal antibody), aurin tricarboxylic acid (ATA, an inhibitor of platelet GPIb) and prostaglandin (PGE1). We measured various platelet activation markers including expressions of PAC-1 and P-selectin, annexin V binding and microparticle formations by using whole blood flow cytometry. We tested argatroban, direct thrombin inhibitor, as a substitute for heparin.

RESULTS: Two types of GPIIb/IIIa antagonists; FK633 and abciximab, and prostaglandin El significantly prevented platelet loss and the increase in binding of PAC-1, an antibody specific for fibrinogen receptor on activated platelets during extracorporeal circulation of heparinized blood. AJvW-2 and ATA had no effect on platelet activation during simulated CPB circulation. These data suggest that inhibition of fibrinogen binding GPIIb/IIIa is partly effective to attenuate platelet activation in a heparinized CPB model circuit. The direct thrombin inhibitor argatroban significantly prevented platelet loss and expression of P-selectin than did heparin. A combination of FK633 with argatroban, as a substitute for heparin, further prevented platelet loss and platelet secretion during simulated CPB circulation, although the inhibition of microparticle formation was less.

CONCLUSIONS: The inhibition of both platelet adhesion and thrombin may be effective to preserve platelet number and function during CPB circulation.

*By Invitation

F12. The Influence of pH Strategy on Cerebral and Collateral Circulation During Hypothermic Cardiopulmonary Bypass in Cyanotic Cardiac Patients - Results of a Randomized Trial and Real-time Monitoring -

Takahiko Sakamoto*, Hiromi Kurosawa, Toshiharu Shin'oka*, Mitsuru Aoki*,

Masayoshi Nagatsu*, Yukihisa Isomatsu*, Tokyo, Japan.

OBJECTIVE: The optimal pH strategy during hypothermic Cardiopulmonary bypass (CPB) remains controversial. Systemic-pulmonary collateral circulation (SPCC) may develop in patients

with cyanotic anomalies. The purpose of this study was to evaluate the effect of pH strategies on cerebral oxygenation and SPCC during hypothermic CPB in cyanotic cardiac patients.

METHODS: Forty cyanotic cardiac patients, older than 1-year were prospectively randomized into 2 groups. Gpl (n=19, 14.3+/-1.5kg) underwent hypothermic CPB with alpha-stat strategy and Gp2 (n=21, 12.5+/-0.9kg) with pH-stat. CPB was established using pump-assisted drainages. Cerebral oxygenation was assessed by near-infrared spectroscopy (NIRS) and the SPCC was calculated by pump flows (%SPCC = (perfusion flow - drainage flow) / perfusion flow x 100). Lactate was measured as an index of systemic anaerobic metabolism.

RESULTS: There were no significant differences in preoperative hematocrit, oxygen saturation, Qp/Qs, CPB duration, minimum temperatures, perfusion flow and pressure, urine output and depth of anesthesia between the groups. Oxyhemoglobin signal and Tissue Oxygenation Index of NIRS monitoring were significantly lower in Gpl, suggesting inadequate cerebral oxygenation with alpha-stat (ANOV A, $p < .0001$). The %SPCC was significantly lower in Gp2 compared with Gp 1, suggesting a reduced pulmonary collateral circulation with pH-stat (ANOVA, $p < .0001$, average; Gpl; 20.1+/-1.2%, Gp2; 7.7+/-0.7%). Serum lactate was significantly lower in Gp2 (ANOVA, $p < .0001$).

CONCLUSIONS: The pH-stat strategy results in an improved environment, including a sufficient cerebral oxygenation, a decrease in SPCC and a lower lactate level. Future studies should investigate the long-term outcome.

**By Invitation*

F13. Regression of Postobstructive Vasculopathy after Revascularization of Chronically Obstructed Pulmonary Artery

Elie Fadel*, Rene Michel*, Saadia Eddahibi*, Renee Bernatchez*, Michel Mazmanian*, Bruno Baudet*, Phih'ppe Dartevelle, Philippe Herve*, Le Plessis Robinson, France; Montreal, Canada.

OBJECTIVE: Pulmonary vascular resistance (PVR) dramatically decreases after pulmonary thromboendarterectomy and further improves during the first months. Initial decrease in PVR is caused by the reestablishment of pulmonary circulation in a previously obstructed vascular bed, whereas long-term progressive improvement may reflect the slow regression of post obstructive vasculopathy (POV). POV include small pulmonary artery (PA) remodeling, precapillary bronchopulmonary anastomoses, and dysfunction of PA endothelium. We tested the hypothesis that POV may regress after lung reperfusion in a piglet model of chronic (5 weeks) left PA obstruction.

METHODS: Chronically ligated left PA was reanastomosed laterally into the pulmonary artery trunk to reperfuse left lung. Left pulmonary blood flow and PA pressure were measured 2 days and five weeks after reperfusion. Left PA smooth muscle hyperplasia, PA endothelium dependent (calcium ionophore and acetylcholine) relaxation, left lung endothelial nitric oxide synthase (eNOS) activity and expression, were assessed after 5 weeks left PA ligation (n=10), and after 5 weeks left lung reperfusion (n=10). In addition, pulmonary angiography and non-selective thoracic aortography assessed patency of the anastomoses and systemic blood supply to the left lung, respectively. Ten animals were studied 5 weeks after left PA dissection without ligation (sham group).

RESULTS: PA anastomoses were patent in all animals as demonstrated by angiography. In addition left pulmonary blood flow values were similar in 5 weeks reperfused and sham animals. Systemic blood supply to the left lung decreased after 5 weeks reperfusion on aortography. Left PVR decreased by 50% at five weeks vs 2 days reperfusion (P=0.0013). The left PA (>_ 600 diameter) percent medial muscle thickness increased after 5 weeks ischemia and regressed to sham values after 5 weeks reperfusion (P=0.001). Endothelium-dependent relaxation was only partially restored after 5 weeks reperfusion, whereas left lung eNOS expressions and activities returned to sham values.

CONCLUSIONS: This study shows for the first time that POV regress after reperfusion in a model of chronic lung ischemia. This may explain the progressive improvement in pulmonary hemodynamics after pulmonary thomboendarterectomy.

**By Invitation*

F14. Efforts toward "Bio-CABG" for diffuse or "inoperative" coronary artery disease

Koji Ueyama*, Gao Bing*, Makoto Ozeki*, Yasuhiko Tabata*, Kazuhiko Doi

, Kazunobu Nishimura, Hisayoshi Suma, Masashi Komeda, Kyoto, Japan.

OBJECTIVES: In the era of drug-elluting stent, coronary artery surgery (CABG) may find a new way by treating many small vessels which catheters never reach and to which angiogenesis alone cannot supply enough blood. We are developing biological CABG (Bio-CABG) to make the way feasible.

METHODS: In 35 Japanese white rabbits, acute myocardial infarction (MI) was created by ligating the major branch of circumflex artery. These ischemic rabbits were divided in four groups: non treated group (Group N, n=7), omentum including the arch structure of a gastro-epiploic artery (GEA) was used to wrap the infarction area of the heart at the time of coronary ligation (Group G, n=10), the gelatin hydrogel sheet incorporating 100 μg of basic fibroblast growth factor (bFGF) was attached to the epicardium of infarction area (Group F, n=8), the gelatin hydrogel sheet incorporating of bFGF was attached to MI area, followed by the omental warpping (Group FG, n=10). Echocardiography was performed preoperatively, and every 2 weeks. Postmortem angiography via GEA was done 4 weeks after operation in group G and FG. MI size (% of whole LV) was calculated by quantitative histomorphometric methods and arte-riolar density was evaluated as the number in the border zone of infarction (by _ smooth muscle actin staining).

RESULTS: Group FG and F showed better ejection fraction and fractional shortening than Group G and N at 2 and 4 weeks ($P<0.05$). Angiography revealed GEA bypassed to coronary artery via rich neovascularization in all 7 animals of group FG (Figure), while some communication was recognized with poor collaterals in only 2/7 cases of Group G. MI size was reduced in group FG than GroupF, G and N (10±3, 16±5, 19±7, 23±2%, $p<0.01$) and Group FG had smaller MI area than Group F. Arteriolar number was significantly increased in Group FG than GroupF, G and N. (23±5, 14±3, 10±1, 4±2 /mm^2, $p<0.01$).

CONCLUSIONS: This study showed that GEA can be bypassed to coronary arteries, without surgical anastomosis, by applying slow release of bFGF in acute MI model. Its effectiveness for salvaging ischemic myocardium was higher than bFGF alone. This new concept

revascularization, *"Bio-CABG",* either alone or as combined with conventional CABG, could potentially revascularize many tiny coronary vessels who have been difficult to treat by conventional surgery or catheter intervention.

**By Invitation*

F15. Inflammatory Response to Cardiac Bypass in Ewe Fetuses: Effects of Steroid Administration or Continuous Hemodiafiltration

Adriano Carotti*, Francesco Emma*, Stefano Picca*, Enrico lannace*, Sonia B Albanese*, Mauro Grigioni*, Francesco Meo*, Mario Sciarra*, Roberto M Di Donate*, Roma, Italy.

OBJECTIVE: To investigate the effectiveness of glucocorticoid administration or continuous hemodiafiltration on Endothelin and Corticotropin Releasing Factor (CRF) release or clearance during prolonged fetal cardiac bypass (CB).

METHODS: Circulating Endothelin 1, 2, and 3 (ET-1, ET-2, ET-3) and CRF levels were measured in fetal ewes during a 60 minutes CB, using a in-line axial flow pump. Blood samples were collected before, during and 90 minutes after CB. Animals were divided in four groups. Groupl (n=6): maternal treatment with 12 mg of betamethasone, one and two days prior to the experiment. Group2 (n=5): fetal treatment with 40 mg of intravenous methyl-prednisolone at the beginning of CB. Groups (n=4): continuous hemodiafiltration with a 0.3 m2 polysulfone filter during the entire CB. Group4 (n=4): control group.

RESULTS: When compared to control animals, maternal steroid pre-treatment failed to decrease Endothelin or CRF production. Fetal treatment with methyl-prednisolone produced a moderate but significant decrease of ET-2 production during CB ($p<0.05$) and of ET-1 production at the end of the experiment ($p<0.05$). Continuous hemodiafiltration showed a strong and highly significant decrease of all Endothelin and CRF levels during CB (ET-1:$p<0.02$, ET-2:$p<0.007$, ET-3:$p<0.001$, CRF<0.01), that was maintained 90 minutes after CB (ET-l:$p<0.03$, ET-2:$p<0.04$, ET-3:$p<0.01$, CRF<0.04).

CONCLUSIONS: Hemodiafiltration represents a highly performant procedure to reduce Endomelin levels during fetal CB. This technique may help, possibly in association with fetal steroid treatment, to contain the inflammatory response leading to post-bypass placental dysfunction, which has been shown to be crucial to allow future feasibility of open fetal cardiac surgery.

5:20 p.m. ADJOURN

**By Invitation*

TUESDAY MORNING, MAY 6, 2003

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

(7 minutes presentation, 8 minutes discussion)

Ballroom, Hynes Convention Center

Moderators: Ralph J Damiano, Jr.

G Alexander Patterson

L1. Gene Delivery to Aortocoronary Saphenous Vein Grafts in a Large-animal Model of Intiraal Hyperplasia

Jason A Petrofski*, Jonathan A Hata*, Thomas R Gerhig*, Steven I Hanish*, Matthew L Williams*, Richard B Thompson*, Cyrus J Parsa*, Walter J Koch*, Carmelo A Milano*, Durham, NC.

INTRODUCTION: Over 50% of aortocoronary saphenous vein grafts (SVGs) are occluded 10 years after surgery. Vascular intimal hyperplasia (IH) is an initial, critical step in the progression toward stenosis. Gene therapy has been used to inhibit IH in previous studies of non-coronary vein grafts. Similar therapies may be useful in preventing aortocoronary SVG stenosis. To date, no large animal models of aortocoronary SVG IH have been described. The two objectives of this study were (1) to characterize a canine model of aortocoronary SVG IH and (2) to demonstrate that gene delivery is possible in these grafts.

METHODS: Nine mongrel dogs (27-32 kg) underwent off-pump aortocoronary bypass utilizing a SVG to the ligated left anterior descending artery via a median sternotomy. A subset (n=5) of dogs had arteriograms on post-op day (POD) 30, 60, and 90. SVGs were also imaged using a Jomed 6F digital intravascular ultrasound probe (IVUS) to monitor wall thickness. At POD 90, dogs were sacrificed and histologically stained SVGs compared to non-grafted saphenous (CON) veins. For each SVG, intimal area, medial area, maximal wall thickness, and minimal wall thickness were determined in 3 representative sections using Image Tool v.3.0 and mean values calculated. Similar measurements were performed for CON veins. Four dogs underwent the same procedure, but SVGs were treated with adenoviruses containing either _-galactosidase (_-gal, n=2) or the _ARKct transgene (a G protein inhibitor) (n=2). Adenoviral-mediated gene delivery ($5x10^{11}$ particles in 3 ml warm PBS) to the SVG was performed by distension of the vein for 20 min immediately prior to anastomosis. These dogs were sacrificed on POD 7 for biochemical analysis of the SVGs.

RESULTS: All SVGs were patent by arteriogram on POD 90. Mean intimal area of SVGs was significantly increased when compared to CON veins (2.79 mm^2 vs. 0.09 mm^2, $p<0.02$). Medial area (3.95 mm^2 vs. 1.58 mm^2, $p=0.01$), intimal/medial ratio (0.68 mm^2 vs. 0.06 mm^2, $p<0.003$), maximal wall thickness (2.21 mm^2 vs. 0.72 mm^2, $p<0.02$), and minimal wall thickness (0.69 mm^2 vs. 0.35 mm^2, $p<0.005$) were also significantly increased (paired t-test). Intimal thickening determined by IVUS was found to be consistent with angiographic appearance and final histologic analysis. In the adenovirus-treated SVGs, positive transgene expression was noted in all samples either by X-gal staining for _-gal treated SVGs or by Northern blotting of _ARKct treated SVGs.

CONCLUSIONS: This study characterizes a large-animal model of aortocoronary SVG vascular IH. Moreover, it demonstrates that adenoviral transgenes may be successfully delivered to veins prior to grafting. Thus, this model may be used in future gene therapy studies to manipulate molecular targets critical in aortocoronary SVG failure due to IH.

**By Invitation*

L2. Preoperative Viral Gene Transfer of Interferon-beta Prevents Recurrence and Improves Survival in Advanced Thoracic Malignancies

Sunil Singhal*, Robert Kruklitis*, Jacob Greenberg*, Steven M Albelda*,

Larry R Kaiser, Philadelphia, PA.

OBJECTIVE: Interferon-beta (IFN-_) can cure thoracic malignancies in murine models with low tumor burden. For large tumors (> 800 mm^3), this benefit is lost. We hypothesize this is due to inadequate T-cell:tumor ratio and to production of immunosuppressive cytokines. Our first goal is to determine the effect of preoperative IFN-_ and surgical debulking for treatment of large tumors. Our second goal is to determine the mechanism of IFN-_ immunotherapy as this is the basis for an ongoing human clinical trial.

METHODS: BALB/c mice underwent subcutaneous injection with NSCLC (L1C2, LLC1, TCI) or mesothelioma (AB12, AC29) cell lines. Tumor bearing mice were treated 72 hours prior to surgical debulking with adenoviral IFN-_ (Ad.IFN-_) (n=49) or control Ad.LacZ (n=43). Mice were assessed for tumor free survival and the growth of distant metastatic foci. Tumor infiltration by T lymphocytes was quantitated using immunohistochemistry. Splenocytes or purified CD8+ T lymphocytes from mice treated with Ad.IFN-_ or Ad.LacZ were simultaneously injected with tumor cells into naive mice (Winn assay).

RESULTS: Treatment with Ad.IFN-_ versus Ad.LacZ prior to surgical debulking resulted in a delay of local tumor recurrence (mean recurrence time: 19 days vs. 8 days, $p<0.01$), an increase in long-term tumor free survival (58% vs.l 1%, $p<0.01$), and a 6-fold smaller foci of implanted tumor cells (64 mm^3 vs. 365 mm^3, $p<0.01$). Splenocytes from mice treated with Ad.IFN-_ versus Ad.LacZ were able to prevent tumor growth in 87% and 20% ($p<0.01$) of the naive mice, respectively. Tumor development in mice treated with CD8+ T lymphocytes isolated from Ad.ffN-_ treated mice was significantly less than in those treated with Ad.LacZ (12% vs. 80%, $p<0.01$).

CONCLUSIONS: These data demonstrate that combining preoperative Ad.IFN-_ with surgical debulking significantly reduces tumor recurrence and improves long-term tumor free survival. This benefit results from Ad.IFN-_ stimulating an effective antitumor immune response. IFN-_ increases the number of CD8+ T lymphocytes and enhances their ability to infiltrate residual tumors. Neoadjuvant immunogenetherapy offers a novel strategy to improve survival in patients with surgically resectable disease and to qualify unresectable patients for surgical intervention.

**By Invitation*

L3. Blockade of the Mitogen-Activated Protein Kinase/Extracellular Signal Regulated Kinase Pathway by U0126 Attenuates Neuronal Damage Following Circulatory Arrest in Neonatal Piglets: A Novel Therapeutic Approach

Matthew R Mulloy*, Deog-Gon Cho*, Paul A Chang*, Mahlon D Johnson*,

Alon S Aharon, Trevor A Robison*, Tamara Buckles*, Davis C Drinkwater Jr.,

Nashville, TN; St. Louis, MO.

OBJECTIVE: Mitogen-activated protein kinases have been implicated in the neuronal and endothelial dysfunction witnessed after cerebral ischemia reperfusion injury. Extracellular signal-regulated kinases 1 and 2 (ERK1/2) represent one subfamily of Mitogen-activated protein kinase (MAPK). ERK1/2 is activated by MAPK/ERK kinase1/2 (MEK1/2) and may be directly involved in the post ischemic cellular injury and endothelial dysfunction seen following cerebral ischemia. Therefore, inhibition of ERK1/2 activation may prevent the neuronal damage associated with circulatory arrest (CA). This study evaluated the ability of a MEK1/2 specific inhibitor - U0126 - to block ERK 1/2 activation and mitigate ischemic neuronal damage in neonatal piglets subjected to CA. Additionally, immunohistochemistry was utilized in an attempt to correlate the presence of ERK 1/2 activation in ischemic cerebral endothelium with the degree of neuronal damage present following CA.

METHODS: Neonatal piglets were subjected to normal flow cardiopulmonary bypass (control, n=4), CA (n=6) and CA with U0126 (CA+U, n=5) at 20°C for a period of 60 minutes. The CA+U group was given 200 (µg/kg of U0126 45 minutes prior to initiation of bypass followed by 100 (µg/kg at the time of reperfusion. Following 24 hours of post cardiopulmonary bypass recovery, brains were harvested for analysis. Hippocampus, basal ganglia, thalamus, cerebellum, mesencephalon, pons, medulla and watershed zones (WSZ) of cerebral cortex were evaluated for neuronal damage using hematoxylin and eosin staining. A section of ischemic cortex was further evaluated by immunohistochemistry with rabbit polyclonal antibody against phosphorylated ERK1/2. All specimens were evaluated in a blinded fashion by a single neuropathologist using a modified grading system to quantify neuronal damage.

RESULTS: In comparison to controls, piglets subjected to CA and CA with U0126 showed diffuse ischemic changes. However, the CA+U group possessed significantly lower neuronal damage scores in the basal ganglia, thalamus, and right frontal WSZ ($p<0.05$) and an overall trend toward decreased neuronal damage versus the CA group. The neuroprotection observed in the CA+U group was accompanied by a near complete blockade of ERK 1/2 expression in endothelia of leptomeningeal and cortical blood vessels.

CONCLUSIONS: Our results demonstrate the ability of U0126 to block ERK 1/2 activation and provide a significant neuroprotective effect in a neonatal piglet model of CA. These results support the targeting of MAPK signaling pathways for inhibition as a novel therapeutic approach to significantly decrease the cerebral injury associated with CA.

**By Invitation*

L4. C-Reactive Protein is a Novel Mediator of Endothelial Activation and Vascular Remodeling in Saphenous Veins: A New Target For Restenosis

Subodh Verma*, Shu-Hong Li*, Renke Li*, Paul Fedak*, Richard D Weisel,

Toronto, ON, Canada.

OBJECTIVE: Restenosis following CABG is a formidable problem, which results from a cascade of events eventually leading to progressive endothelial dysfunction and intimal hyperplasia. Importantly, the inflammatory marker CRP has emerged as one of the most powerful predictors of this process, yet the mechanistic basis of this link remains elusive. We herein report, characterization of CRPs effects on endothelial activation and vascular remodeling which underscore the importance of CRP as a novel target for vein graft restenosis.

METHODS: Study 1: The effects of human recombinant CRP on endothelial activation, angiogenesis, and apoptosis. Human saphenous vein endothelial cells (HSVECs) (n = 20/group) were incubated with CRP (1-100 ug/ml) & ET-1, IL-6, NO, cGMP, VCAM-1, ICAM-1 and MCP-1 quantified. eNOS protein, transcript, and mRNA stability were evaluated, in addition to AT1 and AT2 receptor density. Angiogenesis was evaluated using wound cell migration & tube formation assays. NF-kB activation was determined by confocal microscopy assessing the nuclear localization, while degradation of IkB-a and IkB-b were assessed by Western Blotting. Study 2: Modified Boyden chamber and cell migration assays were performed to evaluate the effects of CRP on saphenous vein smooth muscle cell migration and proliferation. Study 3: Carotid-vein graft interposition experiments were conducted in the presence of locally influsd CRP, and intimal thickening studied.

RESULTS: Incubation of HSVECs resulted in a significant increase in ET-1 & IL-6 production ($p<0.001$), while dose-dependently inhibiting NO and cGMP production, and destabilizing eNOS mRNA ($p<0.001$). CRP markedly stimulated endothelial production of ICAM-1, VCAM-1, and MCP-1, in addition to upregulating ATI receptor density ($p<0.002$). CRP facilitated the degradation of IkB-a and increased NF-kB nuclear localization ($p<0.01$). CRP caused a marked reduction in both basal & VEGF-stimulated angiogenesis ($p<0.01$) and promoted endodielial cell apoptosis. Lastly, CRP stimulated smooth muscle cell migration in-vitro ($p<0.002$) and augmented neointimal formation in-vivo ($P<0.05$).

CONCLUSIONS: CRP directly promotes endothelial activation and vascular remodeling in saphenous veins in-vitro and in-vivo. CRP is a novel target for strategies aimed at reducing vein graft restenosis following CABG.

**By Invitation*

L5. Tumor Necrosis Factor-alpha From Resident Alveolar Macrophages is a Key Initiating Factor in Pulmonary Ischemia Reperfusion Injury

Thomas S Maxey*, Allan Doctor*, Richard Enelow*, Victor E Laubach*,

Irving L Kron, Charlottesville, VA.

OBJECTIVE: The central role of alveolar macrophages in initiating lung ischemia-reperfusion (IR) injury is emerging. Tumor Necrosis Factor alpha (TNF-_) is a pro-inflammatory cytokine secreted by macrophages under various conditions. By utilizing a murine, non-blood perfused, isolated organ system, we modeled pulmonary IR. Transgenic mice were used to explore the role of resident macrophage secretion of TNF-_ in the injury cascade of pulmonary IR injury.

METHODS: Pulmonary IR injury was modeled in our murine isolated lung system for both wild-type (WT) and TNF-_ (TNF -/-) deficient mice. A 1-hour period of ischemia and hypoxic ventilation was followed by 1 hour of constant flow reperfusion under normoxia. Measurements of lung compliance (LC), airway resistance (AW), mean pulmonary artery pressure (mPAP), vascular reactivity (VR), and wet lung weights index (LWI) were obtained and statistically compared using repeated measures ANOVA.

RESULTS: TNF -/- lungs suffered significantly less injury in all physiologic parameters throughout the entire 60-minute reperfusion period compared to WT lungs ($p<0.001$). The most notable effects were observed in (mPAP) and (AR). VR (acute vasoconstrictive episodes per 60 minutes) was also blunted in the TNF -/- group compared to WT (5.8 responses/hr vs. 1.2). LWI

showed a significant increase in edema in the WT compared to TNF -/- group (14.2±0.8 vs. 11,9± 1 gm/kg body weight p <0.05).

Physiologic Parameters Following Reperfusion

Group	0 min	10 min	20 min	30 min	40 min	50 min	60 min
TNF -/- LC	13.3±0.9	13.1±1.2	13.8 ± 1.2	13.6 ± 1.3	14.2 ± 1.2	14.3±1.1	13.5±3.0
WT LC	10.8 ± 1.8	11.8±1.9	10.4±2.3	10.0 ± 2.2	9.8±1.8	9.9±1.8	8.4±1.6
TNF -/- mPAP	8±1.7	7.7 ± 1.8	7.1±1.3	7.1±1.4	7.2±1.4	10.6±3.2	13.3*2.4
WT mPAP	28.6±1.7	22.6 ± 2.6	24.2±6.9	29.8 ± 3.0	32.6±4.9	30.8±2.8	28.9*3.8
TNF-/- AR	0.78±0.04	0.72±0.06	0.69±0.03	0.69±0.04	0.67±0.03	0.68±0.04	0.58*0.02
WT AR	0.9±0.2	0.89±0.2	0.87±0.2	1.7±0.9	1.6±0.8	1.7±0.8	3.7±0.9

CONCLUSIONS: These results demonstrate the profound early effects of TNF-_ from the donor macrophages on ischemia reperfusion injury. Therapies targeted toward diminishing TNF-_ production may be promising in diminishing reperfusion injury.

**By Invitation*

L6. Neonatal Vulnerability to Apoptosis-Related Mitochondrial Dysfunction after Cardioplegic Arrest

Mohsen Karimi*, Li Xing Wang*, James M Hammel*, Mohamed Abdulhamid*, Elesa W Earner*, Wei Gen Li*, Thomas D Scholz*, Jeffrey L Segar*, Christopher A Caldarone*, Iowa City, IA.

OBJECTIVE: Neonatal cardiac surgery is frequently followed by significant postoperative myocardial dysfunction. Our previous reports demonstrated neonatal vulnerability to apoptosis after cardiac surgery. Because apoptosis is intimately associated with mitochondrial dysfunction, we hypothesize that apoptosis-related mitochondrial dysfunction (ARMD) is greater in neonatal than mature lambs six hours after cardioplegic arrest and reperfusion.

METHODS: Newborn lambs and mature lambs (n=5, each group) underwent cardiopulmonary bypass and cardioplegic arrest with cold crystalloid cardioplegia. Cardioplegia was delivered at 20-minute intervals for 60 minutes followed by a six-hour recovery time. Hearts were excised and myocardium examined by cell fractionation, Western blotting, in-vitro kinase assays, and immunohistochemistry.

RESULTS: The Bax/Bcl-2 ratio, an indicator of the pro-apoptotic state, was higher in the newborn group ($p = 0.036$). Apoptosis signal-regulating kinase 1 (ASK-1), an oxidative stress-sensitive protease, and cleaved caspase-3, an apoptosis-effector, were higher in the newborn group ($p = 0.023$ and 0.0085). Mitochondrial release of cytochrome c normalized to citrate synthase activity level was higher in the newborn group versus mature group ($p = 0.0087$). Immunohistochemistry

demonstrated a diffuse cytochrome c pattern in the newborn group supporting mitochondrial release of cytochrome c.

CONCLUSIONS: These results demonstrate that neonatal heart is more vulnerable to apoptosis-related mitochondrial dysfunction (ARMD) than mature hearts. The increased Bax/Bcl-2 ratio and/or greater sensitivity of neonatal hearts to oxidative stress through ASK-1 mediated mechanisms may be responsible for greater release of mitochondrial cytochrome c. Redesign of presently available myoprotective strategies may allow prevention of ARMD in the postoperative neonatal heart.

**By Invitation*

L7. Expression Profiling of Non-Small Cell Lung Carcinoma Identifies Metastatic Genotypes Based on Lymph Node Tumor Burden

Chuong D Hoang*, Jonathan D'Cunha*, Sherif H Tawflc*, Robert A Kratzke*,

Michael A Maddaus, Minneapolis, MN.

OBJECTIVE: This study hypothesized that non-small cell lung carcinoma (NSCLC) cells isolated by laser capture microdissection exhibit a metastatic expression profile that may correlate with the presence and extent of metastatic cells in lymph nodes.

METHODS: Lymph node (LN) metastatic cell burden was defined by two methods hematoxylin and eosin staining (H&E) positive/negative (+/-) and carcinoembryonic antigen-based real time reverse transcriptase polymerase chain reaction (QRT-PCR) +/-.15 NSCLC tumors were then divided into 3 groups after testing of matched LNs (4-7 nodes per tumor): 5 H&E-/-QRT-PCR-, 5 H&E-/QRT-PCR+ (micrometastatic), and 5 H&E+. Total RNA was extracted from 3000-5000 LCM-isolated NSCLC cells and amplified. Each tumor sample was hybridized to a microarray with pooled normal adult lung as control. 3-fold overexpression and 30% underexpression were significant. K groups analysis identified genes differentially expressed between tumor types for use in hierarchical clustering.

RESULTS: The 15 primary tumors clustered into three distinct groups: cluster 1 predominated in tumors with H&E+ LN, cluster 2 had more tumors with LN micrometastases, and cluster 3 had only tumors without LN metastases (Table). Tumors with LN micrometastases were distributed across clusters 1 and 2, suggesting molecular sub-groups. The clusters are comprised of 75 genes differentially expressed between tumor groups. The gastrin-releasing peptide gene, regulating an autocrine growth factor, was notably overexpressed (11.1-fold average) in highly metastatic cluster 1 tumors and may contribute to metastatic aggressiveness.

Cluster Analysis of NSCLC Tumors

	Stage I		Stage III	**% of tumors with LN metastases**
	H&E-/QRT-PCR-	H&E-/QRT-PCR+	H&E+	
Cluster 1	1	2	4	86% (6/7)
Cluster 2	1	3	1	80% (4/5)
Cluster 3	3	0	0	0%(0/3)

CONCLUSIONS: LCM combined with microarray analysis is a powerful methods to characterize the molecular profile of tumor cells. The gene expression profiles of clusters 1 and 2 may define

genotypes prone to metastasize. The three groups of LN tumor burden trended towards separate gene clusters suggesting that this novel approach may identify graded metastatic propensity. Further, individual genes defined in the clusters may suggest metastatic mechanisms and represent new therapeutic targets.

**By Invitation*

L8. Late Outcomes in Patients with Unconnected Mild-Moderate Mitral Regurgitation at The Time of Isolated Coronary Artery Bypass Grafting.

Hari R Mallidi*, Marc P PeUetier*, Nimesh D Desai*, Jeri Sever*, George T Christakis, Gideon Cohen*, Bernard S Goldman, Stephen E Fremes, Toronto, ON, Canada.

OBJECTIVE: Asymptomatic mitral regurgitation (MR) is often present in patients undergoing coronary artery bypass surgery. The long-term outcome in these patients follows an uncertain course. The purpose of this study is to examine the late outcomes in patients with mild to moderate MR at the time of isolated CABG.

METHODS: Patients with mild-moderate MR at the time of isolated CABG were identified from the prospectively collected cardiovascular database at Sunnybrook and Women's HSC. These patients were matched 1:2 by gender, age, LVEF, NYHA class, vascular disease, diabetes, extent of coronary disease, and year of surgery, with patients who had isolated CABG without MR. Eleven patients in the MR group and 17 patients in the NoMR group were determined to be alive by utilizing provincial registry data. There was 99% complete follow-up. Actuarial survival and event-free (death, MI, stroke, cardiac hospitalization, and cardiac re-intervention) survival were compared by log-rank methods. Cox regression was utilized to assess the effect of the presence of MR on late survival and event-free survival. All statistics were performed utilizing the SAS system (v8.0). Preliminary post-operative follow-up echocardiography was available on 32/163 patients.

RESULTS: There were 489 patients in the matched-cohort study. 163 patients had MR and 326 patients had no MR. The average length of follow-up was 3.37 +/- 2.04 years. There was no difference in actuarial survival at 6 years post-operatively (MR 0.810 vs. 0.847 NoMR, p=0.9185). Event-free survival was worse for patients with MR (MR 0.457 vs. 0.647 NoMR, p=0.0258) at 6 years. They also had worse functional status (NYHA Class III-IV: MR 20% (30/150) vs. 8.1% (25/307) NoMR, p=0.0046). After controlling for the matched variables, the hazard ratio associated with the presence of MR by Cox regression was 0.958 (p=0.7626) for survival and 1.198 (p=0.0333) for event-free survival. The only other significant predictor of late survival was preoperative IABP insertion (HR 2.484, p=0.0365). Thirty-one percent of patients (10/32) had progression of MR to at least moderate-severe degree on post-operative echocardiography at an average of 6 months post-operatively.

CONCLUSIONS: Overall late survival was not affected by the presence of mild-moderate degrees of MR in patients undergoing CABG. However, the presence of such MR was associated with poorer event-free survival and worse late functional status. The course of MR post-operatively was variable and nearly a third of patients had worsening MR. These observations suggest that incidental mild-moderate MR may have important intermediate implications for patients requiring CABG.

9:00 a.m. 50th Anniversary of the First Successful Gibbon Bypass

Denton A Cooley

Ballroom, Hynes Convention Center

9:05 a.m. BASIC SCIENCE LECTURE

Advanced Imaging: Aiding the "Mind's Eye" of the Cardiothoracic Surgeon

Richard White, Head, Section of Cardiovascular Imaging, Cleveland Clinic, Cleveland, OH

Introduced By: Fred A Crawford, Jr., Charleston, South Carolina

**By Invitation*

9:35 a.m. SCIENTIFIC SESSION

(8 minute presentation, 12 minutes discussion)

Ballroom, Hynes Convention Center

Moderators: Fred A Crawford, Jr.

Tirone E David

32. Tricuspid Regurgitation or Dilatation: Which Should Be the Criteria for Surgical Repair?

Gilles D Dreyfus, Toufan Bahrami*, K M John Chan*, London, UK.

Discussant: David H Adams

OBJECTIVE: Secondary tricuspid dilatation (TD) is rarely assessed. It may not be accompanied by tricuspid regurgitation (TR). TD can be objectively measured whereas TR can vary according to the preload, afterload and right ventricular function. The purpose of this prospective study was to determine whether surgical repair of the tricuspid valve based on TD rather than TR could lead to potential benefits.

METHOD: Between 1989 and 2002, 311 patients underwent mitral valve repair. The tricuspid valve was examined in each patient. Tricuspid annuloplasty was performed only if the tricuspid annular diameter was greater than twice normal (_ 70mm), regardless of the grade of regurgitation. Patients in Group I (163 patients; 52.4%) received mitral valve repair alone while patients in Group II (148 patients; 47.6%) received mitral valve repair plus tricuspid annuloplasty. Both groups were similar with respect to age, sex, etiology, NYHA class, M.R. grading, L.V.E.S.D. and L.V.E.D.D.

RESULTS: Mean follow up was 4.9 +/- 3 years (0.2-12.5 years). Although not significant, there was a difference in hospital mortality (Gpl: 1.8%, GpII: 0.7%) and also in actuarial survival (Kaplan Meir - GpI: 97.3%, 96.2%, 85.5% and GpII: 98.5%, 98.5%, 90.3% at 3,5 and 10 years respectively). NYHA functional class was significantly better in GpII (GpI: 1.59 +/- 0.84; GpII: 1.11 +/- 0.31; $p < 0.001$). TR grading increased by more than two grades in 48% of patients in GpI and in only 2% in GpII ($p < 0.001$).

CONCLUSIONS: Surgical repair of the tricuspid valve based on TD improves functional status irrespective of the grade of regurgitation. TD and TR were not correlated. TD is an ongoing disease process which will, with time, lead to severe TR.

**By Invitation*

33. Risk of Subsequent Primary Neoplasms Developing in Lung Cancer Patients with Prior Malignancies

Malcolm V Brock*, Anthony Alberg*, Craig Hooker*,

Ann Kammera*, Carmen Roig*, Stephen Yang*, Baltimore, MD.

Discussant: Joe B Putnam, Jr.

OBJECTIVE: Patients presenting with lung cancer often have a history of other primary neoplasms. They also have a higher risk of developing subsequent primary malignancies. Few descriptive studies exist about these patients, and little is known about the risk of three or more independent malignancies arising in the same individual.

METHODS: We examined a cohort of 881 lung cancer patients who had at least one previous primary malignancy in a site other than the lung, and who presented to a single academic institution from 1975-2002. Patients with previous basal and squamous cell skin cancers were excluded. The cohort consisted of 85% (752/881) primary non-small cell lung cancer patients, 12% (103/881) with small cell, and 3% (26/881) with other lung cancer histology. Median age of the cohort was 66 years with 56% female (495/881), 76% Caucasian (668/ 881), and 86% smokers (660/768). Eight hundred nine patients (Group I) had multiple primary cancer diagnoses of which lung cancer was the last malignancy diagnosed while 72 patients (Group II) with lung cancer had a prior history of non-lung primary malignancies, and developed a new primary neoplasm subsequent to their lung cancer.

RESULTS: The cumulative probability of a subsequent malignancy developing synchronously or metachronously in Group II patients was 44% (32/72) at 12 months and 57% (41/72) by 24 months. After 5 years, Group II patients maintained a 25% (18/72) lifelong risk of developing a subsequent malignancy. Smokers were twice as likely as non-smokers to develop subsequent neoplasms. The risk of subsequent cancer development was higher in patients with early stage lung cancer 11% (30/284) vs. 6% (29/507) in patients with advanced disease ($p=0.013$). Age, gender, race, marital status and occupational exposure were not strong determinants of risk in developing a subsequent primary. Group I patients also had a significantly lower 1-year and 5-year survival than Group II patients by Kaplan Meier analysis (48% vs. 59% and 17% vs. 29%, respectively; $p=0.008$). In both groups, the 5-year survival data for stage 1 patients (45% and 48%, respectively) were lower than published norms for patients presenting with lung cancer alone.

CONCLUSIONS: In patients with lung cancer and a history of other previous primary malignancies, the first 2 years following the lung cancer diagnosis is the period of highest risk for developing a subsequent neoplasm. Even after surviving 5 years, there is a moderate risk of developing another primary malignancy. This suggests that given prolonged survival, lung cancer patients with prior malignancies and early stage lesions have a considerable, sustained risk of developing subsequent primary cancers.

10:15 a.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall

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

PRESENTATION

Ballroom, Hynes Convention Center

**By Invitation*

11:05 a.m. SCIENTIFIC SESSION

Moderators: Fred A Crawford, Jr.

Tirone E David

34. Determinants of Mortality and Type of Repair in Neonates with Pulmonary Atresia-Intact Ventricular Septum

David A Ashburn*, Eugene H Blackstone, Winfield J Wells, Richard A Jonas, Frank A Pigula*, Peter B Manning*, Gary K Lofland*, William G Williams, Brian W McCrindle*, And Members*, Toronto, ON, Canada; Cleveland, OH; Los Angeles, CA; Boston, MA; Pittsburgh, PA; Cincinnati, OH; Kansas City, MO; Toronto, ON.

Discussant: Roger B B Mee

OBJECTIVE: To define the prevalence of definitive end-states and their determinants in children diagnosed with pulmonary atresia-intact ventricular septum (PATVS) during the neonatal period.

METHODS: n=408 neonates with PATVS were entered into a prospective study by 33 institutions (n=346; Jan 1987 to Oct 1993; n=62: Jan 1995 to May 1997). Morphologic and hemo-dynamic data were obtained from preoperative echo and catheterization data. Median follow-up beyond initial hospital admission date for surviving children is 10.3 years (<1 month to 15.6 years). Time-related outcomes were analyzed by multivariable modeling of the hazard function. Competing risks methodology was used to demonstrate the prevalence of defined end-states (2-ventricle repair, n=110; 1.5-ventricle repair, n=21; 1-ventricle repair, n=75; heart transplant, n=9; death before achieving definitive repair, n=l49; and alive without definitive repair, n=44).

RESULTS: Overall survival is 77% at 1 month, 70% at 6 months, 60% at 5 years, and 58% at 15 years. The prevalence of end-states at 10 years beyond initial hospital admission include: 2-ventricle repair, 31%; 1.5-ventricle repair, 6%; 1-ventricle repair, 20%; heart transplant, 2%; and death before definitive repair, 37%. The remaining 4% were alive without definitive repair at the time of last follow-up. Incremental risk factors for death occurring before definitive repair include low birth weight (P<0.01), non-cardiac anomaly (P=0.02), reduced RV:LV systolic pressure ratio (P=0.02), enlarged right ventricle (P=0.02), severe tricuspid regurgitation (P<0.01), extremes of tricuspid valve diameter (P=0.01), RV dependent coronary circulation (P=0.01), and Ebsteins malformation (P=0.03). Later date of enrollment was protective from mortality (P=0.06). Factors discriminating between 1, 1.5, and 2-ventricle repairs include tricuspid valve size and measures of RV size and function. Larger right ventricle and tricuspid valve increase the chance of achieving a 2-ventricle repair. Extreme values (both large and small) of tricuspid valve dimension, right ventricle size, and RV:LV systolic pressure ratio (both high and low) favor a 1-ventricle repair.

CONCLUSIONS: Based on these results, we infer that the impact of poorly developed right heart structures on early death in neonates with PAIVS has been partly mitigated by increasing knowledge and experience during the course of this study. Adequacy of right heart structures predicts the type of definitive repair achieved with approximately one-third of neonates expected to undergo a 2-ventricle repair.

**By Invitation*

35. LITA-Radial Artery Composite Grafts as the Technique of Choice for Myocardial Revascularization in the Elderly: A Prospective Randomized Evaluation

Claudio Muneretto, Alberto Negri*, Gianluigi Bisleri*, Jacopo Manfredi*, Marco Metra*, Savina Nodari*, Livio Dei Cas*, Brescia, Italy.

Discussant: Brian Buxton

OBJECTIVE: To evaluate in the elderly the advantages offered by the use of LITA-radial artery composite grafts in comparison to conventional CABG.

METHODS: We prospectively enrolled 160 patients older than 70 years of age undergoing myocardial revascularization. Patients were assigned at random to Group 1 (G1)=80 pts, receiving total arterial revascularization (LITA on LAD plus radial artery) or Group 2 (G2)=80 pts, receiving standard CABG (LITA on LAD plus saphenous vein grafts). The Radial Artery was used in all cases as a composite Y graft. We compared the two groups in terms of early post-operative complications and late cardiac events as angina/MI recurrence, graft occlusion and death.

RESULTS: Pre-operative characteristics and risk factors were comparable between the groups (Euroscore:Gl=7.9 vs G2=8.1). We did not observe differences in terms of mean number of grafted coronary vessels (G1=2.4 vs G2=2.5), mean aortic cross-clamping time (G1=37±7 vs G2=38±7 min), mechanical ventilation time (G1=22±12 vs G2=23±11 hrs), ICU stay (Gl=39±10 vs G2=40±9 hrs), and hospital mortality (Gl=3 pts, 3.75% vs G2=4 pts, 5%). Cerebrovascular accidents occurred in 0 pts in G1 vs 4 pts(5%) in G2 (transient damage in 2 pts). In G2 leg wound infections were observed in 7 pts(8.75%). At a mean follow-up of 16±3 months incidence of angina recurrence was2,5% (2pts.) inGlvs 12,5% (10pz.) in G2. (p=0.03) Angiography showed 100% and 98.7% (1/75) patency rate for LITAs in Gl and G2 respectively, 98.7% radial artery patency in G1 (1/76 radial arteries) and 89% saphenous vein graft patency in G2 (11/100 saphenous vein grafts). (p=0.02) Multivariate logistic regression and Cox regression model identified saphenous vein grafts as indipendent predictor for graft occlusion and angina recurrence.

CONCLUSIONS: The use of LITA-radial artery composite grafts in the elderly proved to be safe and associated with a significant lower incidence of early cerebrovascular accidents. Arterial myocardial revascularization also reduced the incidence of late cardiac events in elderly when compared with conventional CABG.

11:45 a.m. ADDRESS BY HONORED SPEAKER

The Challenges of Human Space Flight

Frank Culbertson, Captain, USN/Ret, Astronaut

Ballroom, Hynes Convention Center

Introduced By: Fred A Crawford, Jr.

Charleston, South Carolina

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

Exhibit Hall, 2nd Floor, Hynes Convention Center

CARDIOTHORACIC RESIDENTS' LUNCHEON

Room 200, Hynes Convention Center

**By Invitation*

TUESDAY AFTERNOON, MAY 6, 2003

1:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION -ADULT CARDIAC SURGERY

(8 minutes presentation, 12 minutes discussion

Ballroom, Hynes Convention Center

Moderators: Bruce W Lytle
Hartzell V Schaff

36. Late Incidence and Predictors of Persistent or Recurrent Heart Failure in Patients with Prosthetic Heart Valves

Marc Ruel*, Roy G Masters*, Eraser D Rubens*, Andrew L Pipe*, Thierry G

Mesana*, Ottawa, ON, Canada.

Discussant: A Mark Gillinov

OBJECTIVE: To examine the causes of persistent or recurrent CHF after aortic and mitral valve replacement and account for selection bias, confounding, and sample overfilling in determining the impact of prosthesis size.

METHODS: Patients (N=2634) who underwent left-heart valve replacement with contemporary prostheses between 1976 and 2001 were followed-up with annual clinical assessment and echocardiography. The impact of demographic, comorbid and valve-related variables on postoperative CHF (NYHA class 3-4 or CHF death) was evaluated with stratified log-rank tests and Cox proportional hazard models. Predictors of CHF prevalence at 1, 5 and 10 years were also determined with logistic regression. All models were bootstrapped 1000 times (Intercooled Stata, College Station, TX).

RESULTS: Total follow-up was 10952 years (median 4.1 years, range 60 days to 19.9 years). Freedom from CHF at 1, 5, 10 and 15 years was 98.2±0.3%, 88.5±1.0%, 73.8±2.2% and 48.4±6.8%, respectively, for aortic valves, and 96.6±0.6%,83.1±1.5%,66.7±0.03, and 38.0±6.5% for mitral valves (hazard ratio: 1.45±0.16 versus aortic; P=0.001). For aortic valves, age, preoperative NYHA class, LV grade, elevated diastolic pulmonary artery pressures, atrial fibrillation, coronary disease, tricuspid regurgitation and redo status predicted a higher incidence and prevalence of CHF postoperatively (all $P<0.05$). Larger prosthesis size, both absolute and adjusted for body surface area, was independently and robustly associated with freedom from CHF

(bias-corrected bootstrapped hazard ratio: 0.8010.11 per size increase; 95% CI: 0.55,0.98; P=0.007). For mitral valves, advanced preoperative NYHA, coronary disease and previous CVA independently predicted postoperative CHF, while prosthesis size had no effect.

CONCLUSIONS: These analyses identify independent predictors of CHF and CHF death after valve replacement and indicate that aortic prosthesis size has a marked causal impact.

**By Invitation*

37. Twenty-Year Experience with the St Jude Mechanical Valve Prosthesis

John S Ikonomidis*, John M Kratz, Arthur J Crumbley III*, Martha R Stroud*,

Scott M Bradley*, Robert M Sade, Fred A Crawford Jr., Charleston, SC.

Discussant: Alfredo Trento

OBJECTIVE: We have prospectively followed all St. Jude mechanical valve recipients at the Medical University of South Carolina since the initial implant in January 1979 and now present our 20-year experience in adults.

METHODS: 837 aortic (AVR; n=478) or mitral (MVR; n=359) valve recipients from January 1979 to December 2000 were followed prospectively at 12-month intervals.

RESULTS: Ages ranged from 19 to 84 years. Follow-up averaged (mean±S.D.) 6.9±5.4 years (98% complete). Patients were in NYHA class III or IV in 77% (AVR) and 89% (MVR) preoperatively. A 19 mm valve was implanted in 15.5% of AVR patients. Coronary bypass (CABG) was required in 31% (AVR) and 20% (MVR). Thirty day mortality was 17/478 (3.6%) (AVR) and 19/359 (5.3%) (MVR), and multivariable predictors (p value; odds ratio;95% C.I.) of 30 day mortality were 19 mm valve size (0.011;3.81;1.36,10.65), ‰¥ 3 CABG grafts (0.041;3.55;1.06,11.94) and NYHA IV (0.039;2.86;1.05,7.79) for AVR and NYHA IV (0.022;3.48;1.19,10.14) and age (0.039;1.05;1.00,1.09) for MVR. Actuarial survivorship at 10 and 20 years was 56.8±2.8% and 26.3±4.7% (AVR) and60.7±3.1%and38.5±4.1% (MVR). Multivariable predictors of late death were African-American ethnicity (0.0005;2.49; 1.73,3.60), NYHA III or IV (0.001;2.25;1.42,3.55), CABG (0.002;1.69;1.22,2.33) and age (0.0005;1.04;1.03,1.05) for AVR and NYHA III or IV (0.015;3.04;1.24,7.46), CABG (0.047;1.60;1.01,2.55) and age (0.002;1.03;1.01,1.04) for MVR. For AVR, effective orifice area (EGA) was univariately (p=0.002) but not multivariately (p=0.378) predictive of late death. Structural valve deterioration was not observed. For AVR, actuarial freedom (at 10 and 20 years) from reoperation (REOP) was 93.2±1.3% and 89.9±2.3%, thromboembolism (TE) 81.6±2.7% and 67.5±7.5%, hemorrhage (HEM) 76.7±2.6% and 65.5±6.3% and prosthetic valve endocarditis (PVE) 94.1±1.2% and 94.1±1.2%. For MVR, actuarial freedom (at 10 and 20 years) from REOP was 96.4±1.2% and 90.0±2.9%, TE 76.8±3.1% and 58.8±7.2%, HEM 85.6±2.4% and 64.8±7.6% and PVE 97.7±1.0% and 96.1±1.8%.

CONCLUSIONS: These results compare favorably with other mechanical prostheses. After two decades of observation with close follow-up, the St. Jude mechanical valve continues to be a reliable mechanical prosthesis with stable rates of valve-related complications.

**By Invitation*

38. Mid Term Results of the Edge-to-Edge Mitral Valve Repair without Annuloplasty

Francesco Maisano*, Alessandro Caldarola*, Andrea Blasio*, Michele De Bonis*, Eustachio Agricola*, Michele Oppizzi*, Giovanni La Canna*, Ottavio Alfleri*, Milano, Italy.

Discussant: W Randolph Chitwood, Jr.

OBJECTIVE: The edge-to-edge technique is an effective method of mitral valve repair, usually performed in association with an annuloplasty (to increase the stability of the repair) with some rare exemptions. We retrospectively analyzed the results of the patients who underwent isolated edge-to-edge repair, particularly in view of potential minimally invasive and percutaneus applications.

METHODS: From Dec 1993 to Dec 2001, 91 patients (median age 65 yrs) underwent mitral valve repair by isolated edge-to-edge procedure, without the association of annuloplasty. Ethiology of mitral regurgitation was: degenerative (62 pts), ischemic (13 pts), rheumatic (7), endocarditis (6), other (3). Preoperative EF was 56±9.8%. There were 3 pts in NYHA class I, 41 pts in class II, 45 in class III and 2 in class IV. Type I lesions were present in 8 pts, type II in 68 and type III in 15 (in 9 combined with type II). A double orifice valve was created in 77 pts, while paracommissural repair was done in the remaining 14 pts. Decision not to perform annuloplasty was taken because of: small preoperative annular dimensions and risk for postoperative stenosis (53 pts), diffuse and severe annular calcifications (33 pts) and other reasons (5 pts).

RESULTS: There where 2 hospital and 3 late deaths for a 5 yrs survival of 96±2,1%. At a mean follow-up of 2.5 yrs, 48 pts were in NYHA class I, 30 pts in class II and 9 pts in class III. Nine pts required reoperation for recurrent mitral regurgitation. Overall freedom from reoperation was 97±1.9% and 82±7.4% at 1 and 5 yrs. Annular calcification was associated with higher reoperation rate (freedom from reoperation at 5 yrs was 56±23.4% vs 92±3.7% in the pts without calcified annulus, p=0.1). Among the subgroups, no patients with ischemic or endocarditis ethiology had failure of repair, freedom from reoperation at 5 yrs was 76+12.3% and 71±17.0% in the degenerative and rheumatic group, respectively.

CONCLUSIONS: Our data confirm suboptimal results of the edge-to-edge technique when annuloplasty is not added to the repair. The isolated edge-to-edge repair in presence of annular calcification is associated with high reoperation rate, and should not be considered as first option in this setting, although it can be carried out with very low operative risk. Although our experience is still limited, the edge-to-edge without ring annuloplasty is a viable option to treat ischemic mitral regurgitation in selected patients.

**By Invitation*

39. Redo Mitral Surgery via Thoracotomy: Warm Beating Heart is Superior to Fibrillatory Arrest

Matthew A Romano*, Iva M Smolens*, Francis D Pagani*, Richard L Prager,

G Michael Deeb, Steven F Boiling, Ann Arbor, MI.

Discussant: Hartzell V Schaff

OBJECTIVE: Right thoracotomy employing ventricular fibrillatory arrest with systemic cooling (VF) has been used for redo mitral valve surgery after previous sternotomy. This approach avoids the high mortality (up to 20%) and the known complications of redo sternotomy such as injury to or embolism from prior grafts, sternal dehiscence, phrenic nerve injury, and excessive hemorrhage. Recently we have enhanced the redo right thoracotomy approach, and utilized a warm beating heart (BH) technique, which offers significant benefits over VF redo right thoracotomy.

METHODS: To determine the advantages of beating heart redo mitral valve surgery, we reviewed and analyzed 181 patients who underwent redo mitral surgery via a right thoracotomy. From 1996-1999, 134 patients underwent redo mitral surgery with VF, and more recently from 2000 to 2002, 47 patients underwent BH surgery. The age, risk factors, NYHA class, and preoperative LVEF were not significantly different. Core temperature on cardiopulmonary bypass (CPB) for BH was 32.6°C vs. 26.2°C for VF ($p<0.001$). The vast majority of patients were completely cannulated via the chest, without femoral cannulation.

RESULTS: The 30-day hospital mortality was significandy lower for BH patients at 4.7%, than for VF patients at 7.4%.0ther results are shown in the table. Significant complications were uncommon. Reoperation was required in less than 2%, only 1 BH patient required reexploration for bleeding. Post operative hospital stay was the same in both populations at 7 days. The CNS event rate was not significantly different at 2%.

	BH	**VF**
CPB Time (min)	84±4*	113±36
PRBC (units)	2.8±2*	3.8±5
FFP (units)	0.7±1 1.3*	1.8±4
Platelets (packs)	0.5±4*	1.5±17
Blood Loss (ml)	446±206*	589±485
Time to Extubation (hrs)	13±24*	34±101

*$p<0.001$ vs. VF

CONCLUSIONS: As the rate of patients being referred for reoperative mitral valve surgery increases, new approaches must be adopted to improve outcome in this complex operative population. Performing redo mitral valve surgery on the warm beating heart offers superior results with shorter bypass times, lower transfusion requirements, decreased blood loss, earlier extubation and most importantly, decreased mortality compared to VF.

**By Invitation*

40. Hypercholesterolemia is an Important Risk Factor for Bioprosthetic Heart Valve Calcification, Failure and Reoperation

Robert S Farivar*, Lawrence H Cohn, Boston, MA.

Discussant: D. Craig Miller

OBJECTIVE: Risk factors for bioprosthetic valve (BPV) calcification include younger age, renal failure and hyperparathyroidism. Since cholesterol and lipids have been found in explanted BPV, we hypothesized that hypercholesterolemia per se may be linked to BPV calcification and may be a risk factor for late failure.

METHODS: We performed *cohort* and *case-control* analysis to test our hypothesis. For *cohort analysis,* we identified 144 pts (82 male, 55 y (23-79y)) with a BPV removed at our institution from 1992-2002, who had serum cholesterol measured and calcification quantified by radiography and histology. For the *case-control* study, we matched 66 patients with failed BPV to 66 with intact BPV for age at implant (mean 58y), BPV survival (mean 7.3y) and position, and determined mean cholesterol and odds ratio for valve failure.

RESULTS: In *cohort* analysis, hypercholesterolemia (FigA, p=0.01), younger age and CAD (p=0.02) was linked to calcification. Valves lasted a mean of 10.9y. In *case-control* analysis, patients with intact BPV had lower cholesterol than those with failed BPV (FigB). The mean cholesterol of patients requiring reoperation was 189 mg/dl, vs 163 for those free of reop (p<0.0001). Statin use was higher at 18% (n=12) in the intact valve group vs. those needing reop 6% (n=3;p=0.02). The odds ratio for failure was 3-9 (CI:1.7-8.9) for patients with cholesterol above 200 mg/dl vs those below. Hypertension, diabetes, sex, smoking, valve type or position was not linked to calcification or failure in either study.

CONCLUSIONS: We have shown for the first time that hypercholesterolemia is a risk factor for BPV calcification, late valve failure and reoperation. Studies to test if statins will prolong valve durability are warranted.

3:25 p.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

**By Invitation*

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

Ballroom, Hynes Convention Center

Moderators: Bruce W Lytle
Hartzell V Schaff

41. Is Repair of Aortic Valve Regurgitation a Safe Alternative to Valve Replacement?

Kenji Minakata*, Hartzell V Schaff, Kenton J Zehr*, Joseph A Dearani, Richard C Daly, Thomas A Orszulak, Francisco J Puga, Gordon K Danielson, Rochester, MN.

Discussant: Lawrence H Cohn

OBJECTIVE: The aim of this study is to assess outcome of valve repair in patients with aortic valve regurgitation (AR) particularly as regards incidence and risk of reoperation.

METHODS: We retrospectively reviewed 160 consecutive patients (127 males) who underwent aortic valve repair between 1986 and 2001. This study focused on patients with primary valve

pathology and does not include patients who developed AR associated with aortic dissection, ventricular septal defect or annuloaortic ectasia. Ages ranged from 14 to 84 years (mean 56±17 years). Patients were categorized according to the main etiology of AR; 64 patients (40%) had annular dilatation leading to central leakage, 54 (33%) had bicuspid valves, 33 (21%) with tricuspid valves had prolapse of one or more cusps, and 9 (6%) were found to have cusp perforation. Repair methods included use of commissural plication (n= 153, 96%), partial cusp resection with plication (n=47, 29%), resuspension or cusp shortening (n=44, 28%), patch closure (n=10, 6%), and cusp shaving (n=6, 4%). Concomitant procedures were performed in 98 patients; mitral valve repair or replacement in 46, CABG in 29, aneurysm repair in 27, septal myectomy in 9, and Maze procedure in 4.

RESULTS: There were one early death (0.6%) and 13 late deaths; myocardial infarction (n=2), sudden death (n=2), stroke (n=1), descending thoracic aneurysm rupture (n= 1), abdominal aortic aneurysm rupture (n=l), congestive heart failure (n=l), unknown (n=l), and other non-cardiac (n=4). Survival from cardiac death and any cause of death were 97% and 92% at 5 years, respectively. The only risk factor identified for all late death was the history of previous CABG ($p<0.01$). Overall, 16 of 159 (10%) hospital survivors had reoperation for recurrent AR; two patients required re-repair of aortic valve during initial hospitalization. Later aortic valve reoperation was necessary in 14 patients (mean interval from initial operation; 2.9 years); aortic valve replacement in 12, composite aortic root replacement in 2. There were no deaths related to reoperation. Freedom from reoperation for AR were 98% at 1 year, 90% at 3 years, and 87% at 5 years. There were no risk factors identified for reoperation for AR in this study including specific etiology or repair technique.

CONCLUSIONS: Aortic valve repair can be performed with excellent freedom from morbidity and mortality. The freedom from late reoperation is low, thus aortic valve repair should be considered in the selected patients with aortic valve regurgitation.

**By Invitation*

42. Tricuspid Valve Repair: Durability and Risk Factors for Failure

Sunil K Bhudia*, Patrick M McCarthy, Jeevanantham Rajeswaran*, Katherine J Hoercher*, Bruce W Lytle, Delos M Cosgrove, Eugene H Blackstone, Cleveland, OH.

Discussant: Steven F. Boiling

OBJECTIVE: This study was undertaken to compare the durability of different tricuspid valve annuloplasty (TVA) techniques and establish risk factors for failure of tricuspid valve repair.

METHODS: From 1990 to 1999, 790 patients (mean age 65±12 years, 64% female, New York Heart Association functional class III or IV in 51% and right ventricular systolic pressure 56±18mmHg) underwent TVA for *functional* regurgitation using 4 techniques: Carpentier rigid ring, Cosgrove flexible band, De Vega procedure, and customized Peri-Guard ring. 89% of the patients had a concomitant mitral valve procedure. 2247 transthoracic echocardiograms were retrieved during follow-up, and tricuspid regurgitation (TR) was analyzed using ordinal longitudinal methods. Multivariable analysis was done to identify risk factors for repair failure.

RESULTS: After TVA, TR at 1 week was still 3+ or 4+ in 14% of patients (Table). Severity of TR was stable across time with the Carpentier rigid ring (P=.7), increased slowly with the Cosgrove flexible band (P=.05), and more rapidly with both the De Vega procedure (P=.002) and Peri-Guard

ring (P=.0009) (Table). Risk factors for tricuspid valve repair failure included the following: higher preoperative TR grade, poor left ventricular function, and repair type other than the Carpentier rigid ring annuloplasty. Tricuspid valve repair failure did *not* correlate with right ventricular systolic pressure, ring size, preoperative New York Heart Association functional class, or need for concomitant surgery.

	3+ and 4+ tricupid valve regurgitation (%)						
Technique	**Preoperative**	**1 week**	**1 month**	**6 months**	**1 year**	**5 years**	**8 years**
Carpentier rigid ring (n=139)	94	15	15	15	15	17	17
Cosgrove flexible band (n=291)	86	15	15	17	18	18	15
De Vega procedure (n=116)	76	13	14	15	18	28	33
Peri-Guard (n=243)	93	15	15	18	22	32	37
Overall (n=789, 1 missing)	88	14	15	19	20	24	31

CONCLUSIONS: Tricuspid valve annuloplasty did not consistently eliminate severe functional regurgitation, and across time TR increased in Peri-Guard and De Vega annuloplasties as opposed to ring techniques. In an effort to improve early and late outcomes tricuspid valve repair techniques should be modified.

**By Invitation*

43. Preoperative Detection and Management with Iloprost of Patients with Immune Heparin-Induced Thrombocytopenia Undergoing Open Heart Surgery

George M Palatianos, Christoforos N Foroulis*, Maria I Vassili*, Phaedra Matsouka*, George M Astras*, George Kantidakis*, Evi Iliopoulou*, Efthimia Melissari*, Athens, Greece.

Discussant: Michael A Acker

OBJECTIVE: Detection and management of patients with immune heparin-induced thrombocytopenia (HIT) who undergo open-heart surgery are not well defined. We established a protocol for these patients based on the prostacyclin analog, Iloprost.

METHODS: Among 1518 patients who underwent open-heart surgery by our Department between 6¥1998 and 5¥2001, 32 (2.1%) patients had previous repeated or prolonged intravenous (>3 days) heparin exposure and/or thrombocytopenia (platelets<150,000¥mm^3). These 32 patients were evaluated with ELBA assay for antibodies against heparin-platelet factor-4 complex (H-PF4). Platelets of patients with detected antibodies were tested with Iloprost for inhibition of heparin aggregation and determination of the inhibiting concentration and the corresponding intravenous infusion rate of Iloprost. Iloprost was infused at the predetermined rate and complete inhibition of platelet aggregation was confirmed prior to heparinization of the patient. Iloprost infusion was gradually discontinued 60 minutes after protamine. Hypotension was prevented or treated with intravenous noradrenaline. Ten additional patients with similar preoperative characteristics, no previous extended exposure to heparin and with normal platelet counts (217000±52637 ¥mm^3) served as controls (group C).

RESULTS: Of the 32 patients, 10 (31.25%) had positive liters of antibodies against H-PF4 and were operated receiving Iloprost infusion at 6-24 ng¥kg¥min according to our protocol (group A). The patients in either group underwent coronary artery bypass (n=7) or valve surgery (n=3). Prior to CPB, mean systolic blood pressure was reduced by 36.0±.9-9 mmHg in group A versus

16.4±14.3 mmHg in controls (p=0.03). In group A, blood pressure was kept >85 mmHg with infusion of norepinephrine at 1-4_g¥kg¥min. There were no adverse effects during CPB. Operative mortality was zero. There were no thrombotic complications or bleeding requiring exploration. One patient from group A bled >1000 ml¥6 hours but did not need exploration. In the remaining patients, chest tube drainage and transfusions of red blood cells and blood products did not differ between groups (p>0.05). There was no significant difference in postoperative morbidity between groups. Platelet counts were reduced by 12.5±8.7% and 38.1±15.2% in groups A and C respectively (p<0.001) one hour after operation and reached preoperative values in both groups by the fifth postoperative day.

CONCLUSION: HIT patients can be easily detected by history and ELISA preoperatively in patients with previous prolonged exposure to heparin and/or thrombocytopenia. Open-heart surgery can be done safely using Iloprost to prevent heparin-induced platelet aggregation.

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

Ballroom, Hynes Convention Center

**By Invitation*

TUESDAY AFTERNOON, MAY 6, 2003

1:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION -GENERAL THORACIC SURGERY

(8 minutes presentation, 12 minutes discussion)

Room 302, Hynes Convention Center

Moderators: Carolyn E. Reed

David J Sugarbaker

44. Pulmonary Metastasectomy for Renal Cell Carcinoma: Determinants of Long-term Survival

Sudish C Murthy*, Kwhanmien Kim*, Thomas VC Rice, Jeevanantham

Rajeswaran*, Ronald M Bukowski*, Malcolm M Decamp, Eugene H

Blackstone, Cleveland, OH.

Discussant: Scott J Swanson

OBJECTIVE: To 1) determine safety of pulmonary metastasectomy for renal cell carcinoma and 2) identify patient and tumor-specific predictors of long-term survival.

METHODS: Between January 1984 and July 2001, 417 patients were diagnosed with pulmonary metastases from renal cell carcinoma. Of these, 92 (mean age 59±9 yr) underwent pulmonary metastasectomy, complete in 63 (68%). ECOG status at diagnosis was 0 in 61 (66%) and 1 in 25 (27%). Preoperatively, number of nodules per patient ranged from 0 to 20, with 50 patients (54%) having unilateral nodules. Median nodule size was 15 mm. Surgical approach was thoracotomy in 56 (60%), median sternotomy in 19 (21%), VATS in 12 (13%), and other combinations in 5. 31 underwent anatomic resection and 61 wedge resection(s). 16% had mediastinal lymph nodes

sampled or resected. 20 (22%) had preoperative immunotherapy. Multivariable hazard function analysis was used to identify patient and tumor-specific predictors of survival.

RESULTS: Operative mortality was 0%. Only 11 (11%) experienced a postoperative morbidity, including 2 neurologic, 1 pulmonary, 1 renal, 1 sepsis. 5-year survival was 42% for completely resected patients and 8% for incompletely resected patients (Figure). The only patient-specific predictor of late mortality was lower preoperative FEV1 (P=.01). Tumor-specific risk factors were presence of lymphatic metastasis (P=.004), increasing number of nodules (P=.0001), and larger nodules (P=.0003). Survival was not improved in patients who underwent preoperative immunotherapy.

CONCLUSIONS: Pulmonary metastasectomy for renal cell carcinoma is safe and well tolerated. Long-term survival depends on the ability to achieve complete resection. Operative planning should include thorough assessment of resectability, and this may require mediastinoscopy to exclude lymph node metastases.

**By Invitation*

45. Long Term Results After Lung Volume Reduction Surgery In Patients With Alpha-1 Antitrypsin Deficiency

Michaela Tutic*, Konrad Bloch*, Didier Lardinois*, Thomas Brack*, Erich Russi*, Walter Weder, Zurich, Switzerland.

Discussant: Malcolm M Decamp, Jr.

OBJECTIVE: The favorable effect of LVRS on dyspnea and lung function in selected patients with emphysema has been demonstrated. However, outcome data on patients with Alpha-1 Antitrypsin Deficiency (A-1 ATD) are scarce.

METHODS: We prospectively studied pulmonary function, dyspnea and 6 min. walking distance in 23 patients (Pi ZZ: 20, Pi SZ: 3,11 females, median age: 56 years, range: 38-74) for up to 3 and a half years after thoracoscopic LVRS.

RESULTS: LVRS improved mean dyspnea score for up to 3 years and mean vital capacity for up to 2 years in patients with advanced emphysema due to A-1ATD. Four patients were long-term responders with an improved pulmonary function for up to three and a half years.

Results

	preop	**3 months**	**6 months**	**12 months**	**24 months**	**36 months**	**42 months**
n	**23**	**18**	**17**	**15**	**14**	**9**	**5**
CD		**2**	**2**	**3**	**4**	**4**	**5**
#TPL		**1**	**1**	**1**	**1**	**2**	**3**
FEV1	0.80±0.1	1.04±0.1*	0.96±0.1*	0.95±0.1	0.84- 0.1	0.75±0.1	0.82±0.2
FEV1 %	28±1.9	38±3.3*	34±3.6*	34±3.5	30 ± 3.4	28±2.5	31 ±5.6
IVC	2.89±0.1	3.48±0.2*	3.49±0.2*	3.55±0.2*	3.12±0.2*	2.63±0.2	2.81±0.3
IVC%	79±4.4	98±4.8*	98±5.9*	98±5.7*	89±6.7*	80±6.1	83±8.3

RV/TLC	0.66±0.1	0.51±0.3*	0.53±0.1*	0.54±0.1*	0.57-0.1	0.60±0.1	0.67±0.1
MRC	3.7±0.1	1.4±0.2*	1.6±0.2*	1.7±0.2*	2.2±0.3*	2.57±0.3*	1.83±0.5
6min WD(m)	262 22	365±20*	399±20*	406-26	367±26	343 ±32	368 ±53
ˆ†FEV1^{3}50 ml		10/18	5/17	7/15	4/14	3/9	2/7
ˆ†RV/TLC‰¤-0.05		15/18	13/17	12/15	7/14	6/9	3/7

(mean; SE)*: p < 0.05 compared to pre-LVRS; ˆ† FEV1 ,ˆ† RV/TLV: compared to baseline, CD: cumulative deaths; # TPL: Patients underwent lung transplantation, MRC: dyspnea score (0-4); 6 min WD: 6 min walking

CONCLUSIONS: The benefits of LVRS are inferior with respect to magnitude and duration as compared to the effect of LVRS in patients with pure smokers emphysema (Bloch, KE et al.; J Thorac Cardiovasc Surg; 2002; 123:845-854). However, patients with A-1 ATD should not be generally excluded from LVRS, since some are long-term responder.

**By Invitation*

46. Preoperative Pulmonary Artery Pressure Impacts Survival After Single Lung Transplantation for COPD

Peter S Dahlberg*, Soon J Park, Brian C Grubbs*, Kay Savik*, Jordan M

Dunitz*, Marshall I Hertz*, Minneapolis, MN.

Discussant: Michael S Mulligan

OBJECTIVE: This study was conducted to determine whether preoperative pulmonary artery (PA) pressure impacts long-term survival following single lung transplantation for chronic obstructive pulmonary disease (COPD).

METHODS: We surveyed charts from 111 patients with COPD that had single lung transplants over a 9-year period. Several covariates known to influence survival were analyzed along with mean PA pressure.

RESULTS: The mean PA pressure of patients, obtained by cardiac cadieterization, was 35±10 mm Hg; the mean FEV1 was 0.65± 0.23. A 1 mm increase in mean PA pressure was found to independently increase the relative risk of death by 1.06 times (95% CI 1.02 to 1.08, p=0.02). Hypothetical survival curves based on this regression analysis are shown in the figure.

CONCLUSIONS: Increasing pretransplant mean PA pressure is associated with decreased survival after single lung transplantation for COPD. Bilateral single lung transplantation may be a better operation for COPD patients with mean PA pressures greater than 55 mm Hg.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

**By Invitation*

3:20 p.m. SIMULTANEOUS SCIENTIFIC SESSION -GENERAL THORACIC SURGERY

Room 302, Hynes Convention Center

Moderators: Carolyn E. Reed
David J Sugarbaker

47. Surgical Benefits of Resection for Metachronous Lung Cancer

[1]Richard J Battafarano*, Seth Force*, Bryan F Meyers*, Sara F Hicks*, Joel D Cooper, G Alexander Patterson, St. Louis, MO.
Discussant: Richard I Whyte

OBJECTIVE: The benefits of resection for metachronous lung cancer are not well described. The objective of this study is to evaluate the safety and efficacy of surgical resection for metachronous lung cancers.

METHODS: We reviewed charts of all patients who underwent a second resection for a metachronous lung cancer from 1988 to 2002. Type of resection, operative morbidity, mortality and survival by stage were analyzed. Survival was determined by the Kaplan-Meier survival method. All patients were pathologically staged using the 1997 AJCC standards.

RESULTS: Pulmonary resections were preformed in 74 patients who had undergone a previous resection. The mean interval between the first and second resection was 2.4±2.5 years. Seventy-two percent of patients presented with stage I cancers, 11% with stage II cancers, and 17% with stage III cancers. Lobectomy and wedge resection were performed equally (40% each) for the metachronous cancers. Twenty-one wedge resections were performed for T1 lesions and three were performed for T2 lesions. Postoperative morbidity (34%) was nearly twice that for the initial resection. Operative mortality for the second resection was 5%. The mean follow up after the second resection was 3-2 years. One, two and five year survival for stage I (n=53) was 85%, 77% and 42%, for stage II (n=8) 88%, 70% and 23%, and for stage III (n=13) 62%, 35% and 23%.

CONCLUSIONS: Despite an increase in morbidity compared to initial lung resections, surgery for metachronous cancers provided stage-adjusted survival that approximated historical survival for lung cancer. Surgery should be considered as a safe and effective treatment for resectable metachronous lung cancers in patients with adequate physiologic pulmonary reserve.

[1]2000-03 Research Scholar

**By Invitation*

48. Prevention, Early Detection and Management of Complications Following 328 Consecutive Extrapleural Pneumonectomies

David J Sugarbaker, William Richards*, Michael Jaklitsch*,

Jeanne Lukanich*, Steven Mentzer*, 'Yolonda Colson*, Michael Chang*,

Philip Linden*, Raphael Bueno*, Boston, MA.

Discussant: Daniel L Miller

OBJECTIVE: EPP for therapy of mesothelioma, thymoma, and lung cancer, has been associated with perioperative mortality and morbidity. Postoperative complications require unique management. We have developed treatment algorhythms for the most common complications and have reduced mortality and hospital stay. We examined complications following EPP in order to elucidate means of avoidance, early detection and treatment.

METHODS: Complications following 328 consecutive EPPs between 1988 and 2000 we examined utilizing a prospective clinical database.

RESULTS: Median age was 58 years (28-77), with 10-day (4-101) median LOS, 198/328 (60.4%) minor and major complications, and 11:328 (3.4%) mortality. Complications included: atrial fibrillation (AF) (145; 44.2%), prolonged intubation (26; 7.9%),vocal cord paralysis (22; 6.7%), deep vein thrombosis (DVT) (21; 6.4%), technical complications (path dehiscence and/or hemorrhage) (20; 6.1%), tamponade (12; 3.6%), ARDS (12; 3.6%), cardiac arrest (10; 3%), constrictive pericarditis (9; 2.7%), aspiration (9; 2.7%), renal failure (9; 2.7%), empyema (8; 2.4%), pulmonary failure (6; 1.8%), myocardial infarction 5; 1.5%), pulmonary embolus (5; 1.5%), and bronchopleural fistula (2; 0.6%). Clinical data demonstrated the following: 1. AF prophylaxis is recommended. 2. Early ambulation, aspiration precautions, endoscopic assessment of vocal cords, and avoidance of fluid overload are crucial. 3. Perioperative diagnosis and aggressive management of DVT are important. 4. Immediate reoperation and open cardiac massage are essential for relief of cardiac herniation and tamponade from cardiac patch dysfunction. 5. Diaphragmatic patch dehiscence and/or hemorrhage require immediate reoperation. 6. Early signs of infection may indicate BPF or empyema and should be treated with thoracoscopic or open drainage and staged removal of patch material. 7. Excessive perioperative mediastinal shift is treated with a catheter placed intraoperatively.

CONCLUSIONS: Complications following EPP require unique management and mortality can be minimized by early detection and aggressive management.

[1]2002-04 Research Scholar

**By Invitation*

49. Combined Bronchoscopy, Mediastinoscopy, and Thoracotomy for Lung Cancer: Who Benefits?

Malcolm M Decamp, Thomas W Rice, Sudish C Murthy*, Kwhanmien Kim*, Daniel P Karchmer*, Christopher D Pierce*, Lisa Rybicki*, Eugene H Blackstone, Cleveland, OH.

Discussant: Bryan Fitch Meyers

OBJECTIVE: Operative staging and resection of lung cancer may be done separately or combined. The purposes of this study were to compare outcomes and costs of the two strategies.

METHODS: Between 1998 and 2001, 343 patients underwent bronchoscopy, mediastinoscopy and thoracotomy (BM-T) for bronchogenic carcinoma. 286 underwent combined BM-T and 57 separate procedures. Separate BM-T patients had higher clinical stage (P<.001): I, 33 (58%) vs. 240 (84%); II, 9 (16%) vs. 26 (9%); III, 12 (21%) vs. 13 (4.5%); and IV, 3 (5.3%) vs. 7 (2.4%). To obtain comparable groups, two propensity matches were performed. The first used 17 variables, including clinical stage; the second added surgeon as a variable.

RESULTS: Prior to matching, clinical outcomes were similar: pulmonary complications, 4/57 (7.0%) vs. 26/286 (9.1%) in separate vs. combined BM-T (P=.9); cardiac complications, 11/57 (19%) vs. 51/286 (18%) (P=.8); and hospital mortality, 1/57 (1.8%) vs. 6/286 (2.1%) (P=.9). Anesthesia time for combined BM-T was longer (5.5±1.0 h) than the thoracotomy portion of separate BM-T (4.7±1.1 h), P<.001, although cumulative operative time was longer for separate BM-T (6.9±1.4 h). Costs were 24% higher for separate BM-T (P<.001). In propensity-matched comparisons, pulmonary complications were possibly higher with combined BM-T, and cardiac complications were similar (Table). Cost-saving was demonstrated when patients were not matched for surgeon. When surgeon was included in matching, costs of both strategies were similar and higher.

	Surgeon Match (n=44)			**No Surgeon Match (n=54)**		
Pathology	**Separate**	**Combined**	**P**	**Separate**	**Combined**	**P**
Pulmonary	2 (4.5%)	6(14%)	.14	3 (5.6%)	8(15%)	.11
Cardiac	7(16%)	7(16%)	1.0	10(18%)	9(17%)	.8
Total cost*	1.08	1.0	.9	1.28	1.0	<.001
Total cost**	1.23	1.14	--	1.28	1	--.

Relative to combined approach within each match. ** Relative to least expensive approach.

CONCLUSIONS: Cost-saving for staging and resection of lung cancer can be realized by combining BM-T under a single anesthetic. In the current cost-containment atmosphere, combined BM-T may benefit the hospital without appreciably changing patient outcome.

**By Invitation*

50. Detection of Micrometastases in Mediastinal Lymph Nodes in Non-Small Cell Lung Cancer Patients by EUS-FNA and Real-Tune rtPCR

Michael B Wallace*, Mark I Block, William E Gillanders*, Loretta Hoover*, James Ravenel*, Valerie Durkalski*, BrendaJ Hoffman*, Robert H Hawes*, Gerard A Silvestri*, Carolyn E Reed, Mostafa M Fraig*, David J Cole*, Michael Mitas*, Charleston, SC.

Discussant: Michael A Maddaus

OBJECTIVE: Despite complete resection, more than half of patients with stage I (NO) NSCLC have recurrence of their disease. We hypothesized that metastatic cancer cells, below the limit of detection of standard pathological techniques (micrometastases), are present in die lymph nodes of NSCLC patients. The aim of our study was to develop minimally invasive (BUS FNA) methods to detect lymphatic micrometastases using real time rtPCR for a panel of cancer associated genes.

METHODS: Patients with proven NSCLC underwent standard staging with helical CT, and PET. Patients without proven metastatic disease underwent BUS FNA of mediastinal lymph nodes in ATS levels 2, 4, 5, 7, 8 and 9. Normal control specimens were obtained from patients with no suspected cancer who were undergoing EUS for benign diseases, and whole cervical lymph nodes were obtained from patients without cancer, undergoing cardiovascular surgery. Each lymph node was examined by standard histopathology. In addition, mRNA was extracted from each lymph node and examined by real time rtPCR for expression of 5 molecular biomarkers of lung cancer (CEA,

CK19, KS1/4, lunx, Mucl). Levels of each biomarker were compared in known positive (path positive), known negative (normal controls), and path negative lymph nodes from NSCLC patients.

RESULTS: Sixty seven patients with 114 lymph nodes and 41 (30 whole LN and 11 EUS FNA LN) normal controls were evaluated. Among the 67 patients, 85/114 (75%) had evaluable mRNA. Nearly all RNA failures occurred early in the study. With modified techniques more than 90% of lymph nodes were evaluable. Using a cutoff of 2 S.D. above the mean of normal control lymph nodes, 14/16 (88%) path positive, 13/69 (19%) path negative, and 1/41 (2%, $p<0.02$) normal control lymph nodes overexpressed at least one of the makers. There was no apparent difference in expression profiles according to tumor histology (squamous vs. adenocarcinoma, data not shown).

CONCLUSIONS: These results suggest that almost one out of five pathologically negative lymph nodes procured by minimally invasive staging techniques overexpress cancer associated biomarkers, which are surrogates for the presence of metastatic disease. These can be detected with minimally invasive staging techniques. The presence of micrometastases may explain the recurrence of lung cancer in patients with stage I/II disease after complete resection. Future studies should focus on the clinical outcome and therapeutic management of rtPCR positive patients.

**By Invitation*

51. Optimal Management When Unsuspected N2 Nodal Disease Is Identified During Thoracotomy for Lung Cancer. Cost-Effectiveness Analysis

[1]Mark K Ferguson, Chicago, IL.

Discussant: Douglas J Mathisen

BACKGROUND: Whether to proceed with lung resection when N2 nodal disease is identified at the time of thoracotomy is controversial because of poor long-term survival with surgery alone, the lack of proven utility of postoperative adjuvant therapy, and the demonstrated benefit of neoadjuvant therapy for this stage of disease.

METHODS: Reports published 1990-2002 were reviewed for outcomes of therapy after the diagnosis of N2 disease. A meta-analysis evaluated patients who were treated by initial resection (clinical N0-N1, pathological N2; 907 pts) or with resection after neoadjuvant therapy (clinical or pathological N2; 264 pts). Cost data for resection were derived from 100 patients from our institution operated 1998-2000. Cost data for radiation therapy and chemotherapy were derived from recently published reports. The decision model compared initial resection ± adjuvant therapy (RES) to thoracotomy without resection followed by neoadjuvant therapy± subsequent resection (RxRES) from the perspective of the medical center. Survival (life years; LY), hospital costs, and quality-adjusted LY (QALY) were used as the payoffs, and cost-effectiveness was assessed.

RESULTS: Overall survival was better but costs were higher for RxRES than for RES (table). The model was sensitive to survival after RES, survival after RxRES, the likelihood of receiving postoperative adjuvant therapy, and the likelihood of undergoing resection after neoadjuvant therapy. The incremental cost-effectiveness ratios (ICER) for RxRES were $23,700/LY and $35,500/QALY.

Treatment	LY	QALY	Cost	Cost/LY	Cost/QALY
RES	1.69	1.56	$16,600	$9,900	$10,900
RxRES	2.14	1.86	$27,200	$14,700	$18,100

CONCLUSIONS: When pathologic N2 nodal disease is discovered during thoracotomy, the approach of delaying resection until after neoadjuvant therapy provides the best overall survival at a modest incremental cost. This is likely due to the beneficial effects of neoadjuvant therapy and the loss of patients with more aggressive disease from the surgical candidate pool during neoadjuvant therapy.

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

Ballroom, Hynes Convention Center

[1]*1986-88 Research Scholar*

**By Invitation*

TUESDAY AFTERNOON, MAY 6, 2003

1:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION -CONGENITAL HEART DISEASE
(8 minutes presentation, 12 minutes discussion)

Room 304, Hynes Convention Center

Moderators: Richard A Jonas

Constantine Mavroudis

52. Modified Single Patch Repair of Complete Atrioventricular Canal Defect

Bassem N Mora*, Gerald R Marx*, Stephen J Roth*, Peter Lang*, Richard A Jonas, Boston, MA.

Discussant: Erle H Austin

OBJECTIVE: We have recently adopted the modification of the classic single patch repair of complete atrioventricular canal as described by Wilcox and Nunn in which the AV valve (AW) leaflets are sutured directly to the crest of the ventricular septum, thereby closing the VSD and obviating the need for leaflet division.

METHODS: Retrospective chart review of 34 consecutive pts with CAVC repaired between 12/97 and 10/02 using the modified single patch technique.

RESULTS: Average weight 5.6 kg; age 0.7 yr (median: 0.3 yr). A single papillary muscle was present in 5. The canal was unbalanced and favored the LV in 3 and RV in 6. AW regurgitation was trivial to none in 5 (15%), mild in 23 (68%), moderate in 5 (15%), severe in 1 (3%). The VSD was small in 6, moderate in 9, large in 19. Three pts had a prior PA band placed elsewhere. 31 pts were repaired using the modified single patch technique; 3 pts had a combination of the classic and modified single patch repairs, 2 of whom had the superior common leaflet divided and resuspended. The mitral valve cleft was closed in 31 pts (91 %). One pt had moderate intraop AW regurgitation, requiring takedown and conversion to the classic single patch method. There were no perioperative deaths. No pt developed LV outflow tract obstruction (LVOTO) or required reoperation for AW regurgitation. Two pts developed atrial arrhythmias; 1 pt with heterotaxy developed heart block requiring pacemaker implantation. One pt required ECMO then was weaned successfully. Postop echos were performed in 30 pts (88%). Mitral regurgitation was trivial to none in 4 (13%), mild in 19 (63%), moderate in 7 (23%), severe in none. Residual VSD was trivial or none in 15, small in 14, moderate in 1. Postop mitral stenosis was trivial or none in 28, mild in 2. LV function was

normal in 25, mildly depressed in 2, moderately depressed in 3. Follow-up echos were performed in 14 pts (41%) following hospital discharge. Mitral regurgitation was trivial to none in 1 (7%), mild in 11 (79%), moderate in 2 (14%), severe in none. Residual VSD was none in 9, small in 3, moderate in 2. Two pts had mild mitral stenosis.

CONCLUSIONS: Repair of CAVC using the modified single patch technique preserves AVV leaflet integrity without producing LVOTO or compromising AVV repair, even in the presence of large VSDs or unbalanced CAVC with a single papillary muscle.

**By Invitation*

53. Valved Bovine Jugular Vein Conduits for Right Ventricular Outflow Tract Reconstruction: An Attractive and Less Expensive Alternative to Pulmonary Homografts

John W Brown, Robert K Darragh*, Indianapolis, IN.

Discussant: Kirk H Kanter

OBJECTIVE: Pulmonary homografts (PH) have been the preferred valved conduits for RVOT reconstruction in the US since the mid-1980s. Although PHs have worked well for Ross patients, the extracardiac conduits used for congenital heart surgery suffer from degeneration and develop regurgitation and obstruction within months after implantation and require replacement within 4-6 years. Recently a valve-containing bovine jugular vein (Contegra Medtronic, Inc. Minneapolis, MN) has been introduced in clinical trials for a variety of patients requiring RVOT reconstruction.

METHODS: The early clinical and echocardiographic results of RVOT reconstruction utilizing the bovine jugular venous valved conduits were retrospectively analyzed in 27 patients. This series consisted of 8 newborns with truncus arteriosus, 18 patients with failed PHs, and 3 patients undergoing a Ross procedure. The patients ages ranged from 2 weeks-37 years and weights were from 2-75kg. The bovine jugular conduits varied in diameter between 12-22mm.

RESULTS: The clinical outcomes have been excellent with the Contegra group. There has been no conduit related morbidity or mortality. There was 1 early death in a complex truncus infant with DiGeorge Syndrome who died several weeks postoperatively of a fungal pneumonia and septicemia. A second non-cardiac death occurred in a 4 month old child who had undergone truncus repair in the newborn period and developed viral meningitis. No conduit has required replacement; however one child developed a significant obstruction at the distal anastomosis approximately 18 months after insertion which did not yield to balloon angioplasty. That distal anastomosis underwent surgical patch arterioplasty. The peak gradients across the RVOT conduits have been acceptable with no patient having a gradient ‰¥ than 30mm Hg. Mild regurgitation across the conduit has been observed in 14 patients. There has been no evidence of shrinkage of the jugular vein conduits as was seen with pulmonary homografts.

CONCLUSIONS: The bovine jugular venous valved conduit offers a promising alternative for RVOT. Early homodynamic performance compares favorably with pulmonary homografts. Clinical advantages are greater shelf availability and natural continuity between the valve and conduit that allows proximal infundibular shaping without additional materials. The price of the bovine jugular venous valve is projected to be approximately one-half that of a pulmonary homograft. Long term durability must be determined for this new conduit. The bovine jugular

venous valved conduit (Contegra) is currently our conduit of choice for right ventricular outflow tract reconstruction in infants, children and young adults.

**By Invitation*

54. Primary Closure for Post-operative Mediastinitis in Children

Richard G Ohye*, Robert Manlker*, Eric J Devaney*, Edward L Bove, Ann

Arbor, MI.

Discussant: John J Lamberti

OBJECTIVES: Mediastinitis is a significant complication of median sternotomy, affecting 0.1-5% of pediatric patients following open-heart surgery. Standard treatment of this infection involves re-sternotomy and debridement of infected bone and tissue. Following debridement, either several days to weeks of continuous irrigation and open drainage with delayed closure, or muscle flap closure is undertaken. Despite these strategies, reported mortality remains significant at up to 25% in pediatric patients. We hypothesized that our policy of simple primary closure at the time of initial debridement would be an effective, less traumatic treatment for mediastinitis in the pediatric population.

METHODS: From January 1986 to July 2002, 6705 sternotomies were performed at the C.S. Mott Children's Hospital, resulting in 57 cases of mediastinitis (0.85%). The cases were divided into two groups based on management, with 42 cases treated by primary closure and 12 cases treated by delayed or muscle flap closure. Treatment method was at the discretion of the surgeon, with only the most severe cases with copious purulence surrounding the heart or hemodynamic compromise selected for delayed closure. The 42 cases of primary closure comprised the primary study group of this institutional IRB-approved, retrospective analysis. All patients received 6 weeks of parenteral antibiotics from the time of diagnosis of mediastinitis. Patient demographics, diagnosis, procedure, operative times, cultures and outcomes were evaluated.

RESULTS: In 93% of these cases (39/42), debridement and primary closure was considered a treatment success. There were three treatment failures, including one death (1/42,3%). Treatment failures were considered any case requiring re-exploration, regardless of whether or not recurrent infection was found. Of the three re-explorations, only one patient had recurrent mediastinitis for an overall rate of infection eradication of 97% (40/41). The remaining two patients with sepsis of unclear etiology were re-explored without the finding of clinical or culture evidence of recurrent infection. One of these patients ultimately succumbed to multisystem organ failure without active mediastinitis. No statistically significant differences could be detected between the treatment successes and failures in this small cohort of patients.

CONCLUSIONS: Debridement and primary closure is an effective means to treat post-operative mediastinitis in children. Results compare favorably with other more lengthy or debilitating treatments. Primary closure avoids the physical and psychological trauma of open treatment and the sternal instability of muscle flaps.

**By Invitation*

55. Extended Septal Myectomy for Hypertrophic Obstructive Cardiomyopathy with Anomalous Mitral Papillary Muscles or Chordae

Joseph A Dearani, Kenji Minakata*, Rick A Nishimura*, Gordon K Danielson,

Rochester, MN.

Discussant: William G Williams

OBJECTIVE: Transaortic left ventricular septal myectomy gives excellent outcome for symptomatic patients with severe hypertrophic obstructive cardiomyopathy (HOCM) unresponsive to medical therapy. Mitral valve replacement (MVR) has been recommended in patients with anomalous mitral subvalvular apparatus because incomplete or only temporary relief of obstruction had been obtained from myectomy alone.

METHODS: 292 patients underwent septal myectomy from 1975 to July 2002 on the authors' surgical services. Early mortality was 0% for 200 patients undergoing isolated myectomy and 1.0% for the entire series. 56 (33 females) patients were found to have anomalous mitral subvalvular apparatus. This included anomalous papillary muscles (n=45; 13 with direct insertion into anterior mitral leaflet, 31 with fusion to septum, 12 with fusion to left ventricular free wall), anomalous chordal structures (n=28), and direct fusion of mitral leaflet into ventricular septum (n=3). Ages ranged from 2 to 77 years (mean 42±20). NYHA functional class 3 or 4 was present in 45 patients (81%) preoperatively.

RESULTS: An extended septal myectomy performed in all patients included a deep trough that was made much wider at the apex than the base, relief of papillary muscle fusion to septum or free wall, and resection of anomalous chordal connections to the outflow tract. Concomitant cardiac procedures were performed in 9 patients including 2 mitral valve repairs. There were no early deaths and no patient required MVR. Mean intraoperative pressure gradients decreased from 70±28 to 5.0±9.0 mmHg. Intraoperative pre-bypass transesophageal echocardiography (TEE) demonstrated moderate mitral regurgitation (MR) in 18 patients (32%) and moderately-severe to severe MR in 19 patients (34%). Post-bypass TEE demonstrated mild or no MR in 51 patients (91%), and moderate MR in 5 (8.9%). Mean follow-up was 2.8 years (maximum 13 years, 96% complete). Two late reoperations were required in children at 3 and 10 years postoperatively for recurrent left ventricular outflow tract obstruction (LVOTO); concomitant mitral valve repair was performed in 1. The majority of patients (98%) were in NYHA functional class 1 or 2 at follow-up.

CONCLUSIONS: Anomalous mitral papillary muscles or chordae associated with HOCM can be successfully treated with the extended septal myectomy. This procedure can be performed with low early mortality, with significant decrease in LVOTO and MR, and without the need for MVR.

3:05 p.m. INTERMISSION - VISIT EXHIBITS

Exhibit Hall, 2nd Floor, Hynes Convention Center

**By Invitation*

3:40 SIMULTANEOUS SCIENTIFIC SESSION - CONGENITAL HEART DISEASE

Room 304, Hynes Convention Center

Moderators: Richard A Jonas

Constantine Mavroudis

56. Intermediate Results and Impact of Age on Outcome in Repair of Ebstein's Anomaly

Jonathan M Chen*, Ralph S Mosca, Karen Altmann*, Beth F Printz*, Kimara Targoff*, Pamela A Mazzeo*, Jan M Quaegebeur, New York, NY.

Discussant: Christopher J Knott-Craig

OBJECTIVE: Several techniques have been promoted as alternatives to valve replacement for repair of Ebstein's anomaly, all involving atrial plication and tricuspid valve restoration. However, few have described preliminary outcomes, and thus we sought to evaluate our results with repair of Ebstein's anomaly.

METHODS: Records were reviewed of patients who underwent repair of Ebstein's anomaly from Sept. 1990 -Sept. 2002 at the Columbia Presbyterian Medical Center. Functional and demographic parameters were evaluated, and preoperative echocardiograms were analyzed with regard to biventricular function, severity of tricuspid regurgitation, and degree of anterior tricuspid leaflet mobility. Postoperative echocardiograms were then reviewed with emphasis on biventricular function, degree of tricuspid regurgitation (TR) and level of displacement of the tricuspid apparatus. Postoperative functional status was correlated with surgical outcome, as were atrial arrhythmias, heart block, the need for adjunctive procedures or tricuspid valve replacement, and death. Repair involved detachment of the valve leaflets, plication of the atrialized ventricle, and leaflet reattachment after clockwise rotation. Follow-up was defined as functional status at last clinic visit.

RESULTS: 25 patients (19 children, 6 adults) 14.7±16 years (range 2 months-47 years) underwent repair during the 12 year period. Indications for operation included diminished exercise capacity, cyanosis, and/or evidence of progressive heart failure. Preoperatively, 11 (44%) demonstrated severe TR; most had normal biventricular function, a large, noncontrac-tile atrialized chamber and significant valve tethering. Associated procedures included ASD/ PFO closure 15 (60%), VSD closure 4 (16%) and radiofrequency ablation 4 (16%). Postop-eratively, 10/19 (53%) children and 2/6 (33%) adults demonstrated moderate to severe (although significantly reduced) TR with good biventricular function. Despite this, 84% of patients had significant improvement in their functional exercise capacity at 4.9±3.2 years follow-up. 2(11%) children required permanent pacemakers for heart block, and one child required early (POD3) reoperation for cavopulmonary shunt. 2/6 (33%) adults required ultimate tricuspid valve replacement 4 and 8 years after repair. There were no operative or perioperative mortalities, however 2 (11%) children died late (1 and 5 years) after repair.

CONCLUSIONS: Repair of Ebstein's anomaly has good functional outcomes in children despite residual, but reduced, echocardiographic TR, likely owing both to the reduced volume load on the right ventricle and to relative annular and ventricular plasticity. Adult patients did not enjoy the same durability of valve repair and not infrequently required later valve replacement.

**By Invitation*

57. Age-Related Gene Expression in Children Undergoing Cardiac Surgery for Right Heart Obstructive Lesions

Igor E Konstantinov*, John G Coles, Cathy Boscarino*, Mark Takahashi*,

Jason Goncalves*, Julia Ritter*, Glen Van Arsdell, Toronto, ON, Canada.

Discussant: Peter J Gruber

BACKGROUND: The global myocardial stress response during cardiac surgery has not been systematically studied, nor is it known whether the response of neonatal myocardium is intrinsically different from that of older children.

METHODS: We studied gene expression profiles in 24 patients during surgery for tetralogy of Fallot, stratified into in Group I (7 pts, aged from 5 to 66 days, mean 30 days) and Group II (17 pts, aged from 4 months to 180 months, mean 33.5 months). Biopsies from the right ventricular outflow tract were taken following aortic occlusion and archived in liquid nitrogen. RNA isolation, fluorescence-labeling of cDNA, hybridization to spotted arrays containing 19,008 characterized or unknown human cDNAs, and quantitative fluoresence scanning of gene expression intensity, were performed at the University of Toronto Health Network Microarray Centre (www.microarray.ca).

DATA ANALYSIS: Data were analyzed with the Significance Analysis for Microarrays program. Minimum Information about Microarray Experiments (MIAME)-compliant, log2 -normalized data sets were compared using repeated permutation to ascertain potential statistical differences in gene expression between patient groups.

RESULTS: There were no hospital deaths or major post-operative morbid events. We identified 50 transcripts differentially expressed in the neonatal group {predicted false discovery rate < 0.8 transcripts}. The pattern of gene expression in the neonatal group was consistent with activation of both cardioprotective and growth-regulatory programs {Table}. Several neonatally-expressed genes have not previously been reported in heart.

Genes with Reported Myocardial Expression

Transcript	Fold change	Expression in neonates
Atrial natriuretic peptide	51.4	increased
Myosin light chain 4	22.2	increased
Myosin light chain 2a	7.7	increased
Hepatoma-derived growth factor	7.5	increased
Syndecan binding protein	7.4	increased
Protein phosphatase 2a	5.7	increased
Fibroblast growth factor 1	7.9	decreased

CONCLUSIONS: Neonatal myocardium has a unique pattern of gene expression consistent with activation of an endogenous, stress-induced protective program. This includes both novel and precedented genes, which are predicted to have effects on both cardiac and pulmonary vascular remodeling. This study indicates that chronically stressed myocardium presents a highly adaptive, and therefore therapeutically relevant, molecular phenotype.

**By Invitation*

58. Blood Group Incompatibility and Accelerated Homograft Fibrocalcifications

Jan T Christenson*, Dominique Vala*, Jorge Sierra*, Maurice Beghetti*,

Afksendiyos Kalangos*, Geneva, Switzerland.

Discussant: John A Hawkins

OBJECTIVE: Cryopreserved valved homograft has become the conduit of choice for right ventricular outflow tract (RVOT) reconstruction in pediatric cardiac surgery. Aortic homografts have been frequently used in pulmonary position due to less important homograft degeneration, but accelerated aortic homograft fibrocalcification may occur. Blood group incompatibility between receiver and homograft donor may play a central role in this context.

METHODS: Between 1993 and 2000, 59 children (32 boys and 27 girls, aged 6.4±4.4 years, 1 month to 14 years) received a cryopreserved valved homograft for RVOT reconstruction, and were followed for 1 to 8 years clinically, with echocardiography and chest X-ray for detection of development of homograft calcifications. There were 59 aortic homografts and 9 pulmonary homografts all used in pulmonary position. 49% (29 patients) had the same blood group (ABO) as the homograft donor (iso-group), while 23 were blood group incompatible (non-iso-group).

RESULTS: No deaths occurred during follow up. During follow up 6 patients (10.2%) required homograft replacement because of severe fibrocalcifications, and another 3 patients showed moderate homograft calcifications (5.1%) at the latest examination. Freedom from moderate to severe homograft calcifications at 5 years (Kaplan-Meier) was 90.6% for the iso-group compared to 69.2% for the non-iso-group, $p<0.0001$. Freedom from calcifications at 5 years for aortic homografts in pulmonary position was 86.1%. Interesting to note is that 80% of detectable fibrocalcifications in the non-iso-group occurred early, within the first year of operation.

CONCLUSIONS: Blood group incompatibility (ABO) between receiver and homograft donor seems to play an important role in the development of accelerated fibrocalcifications in cryopreserved homografts. Aortic homografts may have less graft degeneration than pulmonary homografts in pulmonary position. Blood group compatibility should be respected to avoid accelerated homograft fibrocalcifications.

**By Invitation*

59. Improved Earty Outcome for End-stage Dilated Paediatric Cardiomyopadiy

Anne-Marie McMahon*, Carin Van Doom*, Michael Burch*, Pauline Whitmore*, Sophia Neligan*, Philip Rees*, Alan Goldman*, Victor Tsang*, Martin Elliott*, [1]Marc De Leval, London, UK.

Discussant: Charles B Huddleston

OBJECTIVE: To review the impact of changes in the management strategy of End-stage Dilated Paediatric Cardiomyopathy (EDPC) on early outcome.

METHODS: EDPC was defined as requiring inotropic support with or without mechanical support. Over the last two years EDPC management changed as follows: introduction of high priority listing for transplant, more aggressive approach towards mechanical support, and ABO incompatible transplant for infants. Patients were divided into: Group I: July 1998 - December 1999 (n=24, age 6.0 ± 5.8 years [mean ± SD]) and Group II: January 2000 - October 2002 (n=46, age 5.2 ± 5.7 years).

RESULTS: Treatment modalities and results are summarized in the table. Death refers to hospital mortality. Group II mortality was significantly less ($p<0.02$) than Group I. Priority listing (13

patients) reduced the waiting time for transplant to 6.5 ± 4.6 days compared to 19.8 ± 26.7 days for non-priority listing.

	Group 1		**Group II**	
Total	**n=24**	**Died 9(37%)**	**n=46**	**Died 6(1 3%)**
Medical Therapy	***17***	***7(41%)***	***30***	***4(13%)***
Medical treatment only	12	6(50%)	17	4(24%)
Progressed to transplant	5	1(20%)	13*	0
ECMO	***2***	***0***	***16***	***2(13%)***
Bridge to transplant	0	0	10	0
Bridge to recovery	2	0	4	0
No transplant/recovery	0	0	2	2(100%)
BIVAD	***5***	***2(40%)***	***0***	***0***
Bridge to transplant	3	0	0	0
No transplant/recovery	2	2(100%)	0	0

* includes 3 ABO incompatible t ransplants ECMO=Extracorportal Membrane Oxgenation
BIVAD=Biventricular Assist Device

CONCLUSIONS: Recent refinements in the management of EDPC has had a significant impact on early mortality. Timing of mechanical support, bridge to recovery versus bridge to transplantation and the development of mechanical devices for long term support of children remain areas for future research and development.

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

Ballroom, Hynes Convention Center

[1]*1973-74 Graham Fellow*

**By Invitation*

TUESDAY AFTERNOON, MAY 6, 2003

1:30 p.m. GENERAL THORACIC FORUM SESSION

(5 minutes presentation, 7 minutes discussion)

Room 311, Hynes Convention Center

Moderators: Mark S Allen
Douglas E Wood

F16. Selective Adenosine-A2A Agonist Improves Cardiac Dysfunction Following Pulmonary Ischemia-Reperfusion Injury

Thomas B Reece*, Curtis G Tribble, Thomas S Maxey*, Jonathan D Davis*,

Andrew M Schulman*, Victor E Laubach*, Joel M Linden*, John A Kern*,

Irving L Kron, Charlottesville, VA.

OBJECTIVE: Ischemia-reperfusion (IR) injury negatively impacts patient outcomes in lung transplantation. Clinically, we have observed that lung transplant patients with IR injury tend to have lower cardiac outputs. Previous studies have shown that ATL-146e, a selective adenosine-A_{2A} receptor agonist, reduces inflammation in various reperfusion injury models. We hypothesized that pulmonary IR injury causes secondary heart dysfunction due to inflammation, and secondly, that the anti-inflammatory actions of adenosine-A_{2A} activation will improve this dysfunction.

METHODS: We utilized a rabbit model of warm lung ischemia-reperfusion. The sham group (n=10) underwent 120 minutes of right lung ventilation. The IR group (n=10) and the A_{2A}group (n=10) underwent 90 minutes of right lung ischemia with 30 minutes of reperfusion. ATL-146e (0.04 _g/kg/min) was given intravenously to the A_{2A} group beginning 10 minutes prior to reperfusion and continuing throughout the reperfusion period. Hemodynamics, including cardiac output, and arterial blood gases were monitored during the procedure. Myeloperoxidase (MPO) activity, an indicator of neutrophil sequestration, was measured in lung tissue harvested after reperfusion.

RESULTS: All cardiac outputs (ml/min) were similar prior to lung reperfusion. Reperfusion immediately caused significant drops in cardiac output in both IR and A_{2A} groups compared to sham (92.4 ± 7.4 and 97.6 ± 7.2 vs 127.5 ± 10.0, respectively, $p < 0.03$). Within 5 minutes of reperfusion, the cardiac output in the A_{2A} group improved significantly over the IR group (111.7 ± 3.2 vs 86.4 ± 6.4, $p < 0.03$) and remained significant through the reperfusion period. Lung MPO (_OD/min/g) in A_{2A} and sham groups (40.0 ± 6.5, $p < 0.05$ and 42.2 ± 7.0, $p = 0.053$) were lower than IR group (83.5 ± 18.7). Although never hypoxemic, oxygenation was lower in IR (145 + 54.9, $p = 0.04$) and A_{2A} groups (180 ± 40.6, $p > 0.05$) compared to sham (307 ± 50.3). Central venous pressures and mean arterial pressures were similar among groups.

CONCLUSIONS: Pulmonary IR injury causes cardiac dysfunction independent of preload, afterload, and oxygenation. ATL-146e improves the left ventricular dysfunction caused by pulmonary IR injury, presumably by the anti-inflammatory effects of adenosine-A_{2A}activation on neutrophils.

**By Invitation*

F17. A Clinically Relevant Lung Cancer Prognostic Test From Gene Expression Profiling Data

Gavin J Gordon*, William G Richards*, David J Sugarbaker, Raphael Bueno*,

Boston, MA.

OBJECTIVE: The clinical applicability of bioinformatics tools applied to expression profiling data remains unfulfilled. We have developed a method to predict prognosis in cancer based on ratios of gene expression using data from expression profiling studies (Gordon et al., Cancer Res., 62:4963-4967). Here we have applied this technique to identify patients with Stage I lung cancer at risk for recurrence.

METHODS: Gene expression data composing the training set (Beer et al., Nat. Med. 8:816-24) consisted of Stage I lung adenocarcinoma (ADCA) tumors from patients considered to be treatment responders (i.e. good outcome, survival >60 months, n=21) and those considered to be treatments failures (i.e. poor outcome, survival <60 months, n=11). Gene expression data composing the test set (Bhattacharjee et al., Proc. Natl. Acad. Sci. 98:13790-5) consisted of 62 independent Stage I lung ADCA samples.

RESULTS: We found a total of 8 genes with inversely correlated average expression levels in the training set that matched our filtering criteria ($P<0.01$, at least a 2-fold difference in mean expression levels between both groups). We calculated 16 expression ratios per sample by dividing the expression value of each of the 4 genes expressed at relatively higher levels in good outcome samples by the expression value of each of the 4 genes expressed at relatively higher levels in poor outcome samples. Ratio values >1 and <1 were called good and poor, respectively. To use multiple genes, we calculated the geometric mean for the optimal 3 ratios and found that we could identify training samples with accuracy that exceeded that of any of the gene pair ratios when used alone: 95% (20/21) for good outcome samples, 82% (9/11) for poor outcome samples, and 91% (29/32) overall. We then calculated the geometric mean of these 3 ratios in every sample of the test set and assigned each to an outcome group. We subjected the two groups to Kaplan-Meier survival analysis which resulted in significantly different survival curves ($P=0.0289$, log-rank test, Fig.) with the good prognosis group having a substantially longer median survival (not reached) than the poor prognosis group (47 months).

CONCLUSIONS: Expression ratios are highly accurate in the prognosis of lung cancer and may provide a clinically useful means to predict response to therapy in early detected resectable lung ADCA. This diagnostic methods can be potentially applied to fine needle aspirates.

**By Invitation*

F18. Gene Transfer of Tumor Necrosis Factor Inhibitor Ameliorates Acute Rejection in Lung Allograft

Tsutomu Tagawa*, Benjamin D Kozower*, Samer A Kanaan*, Niccolo Daddi*, Jon H Ritter*, Masashi Muraoka*, Takeshi Nagayasu*, Shinji Akamine*, Tadayuki Oka*, G Alexander Patterson, St. Louis, MO; Nagasaki, Japan.

OBJECTIVE: Tumor necrosis factor (TNF) is an important mediator of lung transplant acute rejection. Soluble TNF receptor I (sTNF-RI) competitively inhibits TNF _ and _. The objectives of this study were to accomplish efficient in vivo sTNF-RI gene transfer and determine its effect on lung allograft acute rejection.

METHODS: Experiment 1: Time-dependent sTNF-RI expression in muscle, serum and lung. The right gastrocnemius muscle of Fischer 344 rats was injected with $1x10^{10}$ pfu adenovirus encoding human sTNF-RI (Ad.sTNF-RI-Ig). Animals were sacrificed 6, 12, 18 and 24 hours after transfection (n=3, each). Muscle, serum and lung transgene expression of sTNF-RI was evaluated using enzyme-linked immunosorbent assay. Experiment 2: The effect of sTNF-RI gene transfer on lung allograft acute rejection. Fischer 344 rats were divided into four groups. Three groups (n=6,

each) underwent recipient intramuscular transfection 24 hours prior to transplant with either saline, $1x10^{10}$ pfu control adenovirus encoding non-functional _-galactosidase, or $1x10^{10}$ pfu adenovirus encoding Ad.sTNF-RI-Ig. One group (n=6) underwent recipient intramuscular transfection at the time of transplant with $1x10^{10}$Ad.sTNF-RI-Ig. Brown Norway donor lung grafts were stored for 5 hours prior to orthotopic lung allotransplant. Graft function and rejection score were assessed 5 days after transplantation. Transgene expression in muscle, serum and lung graft was also evaluated.

RESULTS: Experiment 1: Time-dependent transgene expression was observed in muscle, serum and lung. Experiment 2: Recipient intramuscular transfection 24 hours prior to transplant with $Ix10^{10}$ pfu Ad.sTNF-RI-Ig significantly improved arterial oxygenation compared with the saline, _-galactosidase and transfection at the time of transplant (435.8 ± 106.6 vs. 142.3 ± 146.3, 177.4 ± 153.7 and 237.3 ± 185.2 mmHg, P=0.002, 0.005 and 0.046, respectively). Rejection score was lower in the 24 hours prior to transplant group but not significantly. Soluble TNF-RI was still expressed at the time of sacrifice in both sTNF-RI transfected groups.

CONCLUSIONS: Recipient intramuscular sTNF-RI gene transfer ameliorates experimental acute lung rejection and improves graft oxygenation. Maximum benefit was observed only when transfection occurred prior to transplant.

**By Invitation*

F19. Combined Histone Deacetylation and Proteosome Inhibition Enhances Apoptosis in Non-Small Cell Lung Cancer

Chadrick E Denlinger*, R Michael Broad*, Marty W Mayo*, David R Jones*,

Charlottesville, VA.

OBJECTIVE: Inhibitors of histone deacetylases (HDACs) are potent inducers of cell cycle arrest and apoptosis in certain tumors. We have previously shown that HDAC inhibitors activate the anti-apoptotic transcription factor NF-_B in non-small cell lung cancer (NSCLC). We hypothesize that inhibition of NF-_B activation using the proteosome inhibitors MG-132 or bortezomib (VELCADE TM), formerly known as PS-341, will sensitize NSCLC cells to HDAC inhibitor-mediated apoptosis.

METHODS: Tumorigenic NSCLC cells (A549, H358, H460) were treated with a proteosome inhibitor (MG-132 or bortezomib) followed by the HDAC inhibitor sodium butyrate (NaB). Following treatment, NF-_B transcriptional activity was measured by luciferase reporter assay, and apoptosis determined by DNA fragmentation. Western blot analysis for the cell-cycle regulatory protein p21 was also performed. Finally, cells were pretreated with the caspase inhibitor Boc-D, subjected to above experimental conditions, and apoptosis assessed. Experiments were performed in triplicate and the data analyzed by ANOVA.

RESULTS: NaB increased NF-_B transcriptional activity by 5-fold compared to controls ($p<0.05$) in all NSCLC cell lines. Treatment with MG-132 or bortezomib reduced NaB-induced NF-_B activation to control levels ($p<0.05$). Treatment with NaB alone resulted in little apoptosis, but combined HDAC and proteosome inhibition increased apoptosis by 3-4-fold ($p<0.01$). Pre-treatment with Boc-D rescued cell death following NaB/MG-132 treatment, suggesting a caspase-mediated process. The p21 protein was stabilized following treatment with NaB and MG-132 or bortezomib.

CONCLUSIONS: Molecular targeting of HDACs and proteosomes using combination therapy synergistically enhances apoptosis in NSCLC. NF-_B suppression through proteosome inhibition, followed by treatment with HDAC inhibitors, may be a novel treatment strategy for patients with NSCLC.

**By Invitation*

F20. Inhibition of Esophageal Cancer By Downregulation of Beta Catenin

N Veeramachaneni*, H Kubokura*, C Alexander Patterson, J Drebin*, [1]R

Battafarano*, St Louis, MO.

INTRODUCTION: Progression of esophageal cancer has been associated with mutations of _ catenin-dependent signaling pathways and increased _ catenin localization to the nucleus. _ catenin regulates the activity of TCP family transcription factors in the nucleus. _ catenin regulates several genes critical for the neoplastic growth of cancer cells, including cydin D1, myc, the multidrug resistance gene MDR-1, and matrix metalloprotease 7. Nuclear localization of _ catenin correlates with grade and stage of esophageal cancer, and parallels increased _ catenin dependent transcription. We hypothesize that downregulation of _ catenin will result in inhibition of esophageal cancer cell growth.

METHODS: TE10, an esophageal cancer cell line, was used for this study. _ catenin mRNA and protein levels were determined by RTPCR and Western blot. _ catenin was inhibited by antisense phosphorothioate oligonucleotide (ODN), using the lipid carrier, Effectene (EFT). Control treatments with nonspecific ODN sequence (scrambled; SC), and EFT alone, were performed. Cell viability was determined using a colorimetric thiazolyl blue reduction assay (MTT). Cell growth was assayed by counting the number of trypan blue-excluded viable cells.

RESULTS: The efficacy of _ catenin antisense (AS) treatment was confirmed by both RTPCR and Western blot (Fig 1). RTPCR showed 50% inhibition of _ catenin copy number (EFT 0.9 vs AS 0.4; normalized to GAPDH) 48 hours after treatment of cells. Exposure to _ catenin ODN resulted in >60% reduction of cell viability (EFT 267 OD vs 97 OD; MTT assay, $p<0.05$), and 85% reduction in cell growth (EFT 7733 thousand cells vs AS 966 thousand cells; $p<0.05$) 3 days after treatment.

CONCLUSIONS: Specific down-regulation of _ catenin using antisense ODN resulted in inhibition of esophageal tumor cell proliferation. These data demonstrate the important role of _ catenin in the pathogenesis of esophageal cancer and suggest a beneficial role of treatment targeted to _ catenin.

[1]2001-03 Research Scholar

**By Invitation*

F21. Upregulating the Expression of Class I Molecules by Established Murine Tumors Enhances Their Susceptibility to CD8+ T Lymphocyte-mediated Immunotherapy

Robert E Merritt*, Reiko E Yamada*, Ronald G Crystal*, Robert J Korst, New York, NY.

OBJECTIVE: Malignant lung tumors may escape killing by cytotoxic T lymphocytes (CTL) based on their ability to downregulate the expression of Class I molecules of the Major Histocompatibility Complex (MHC). With this background, the purpose of this study is to determine if (1) MHC class 1 expression by established tumors *in vivo* can be significantly enhanced, and (2) if this enhancement will render these tumors more susceptible to a CTL-mediated immunotherapy strategy.

METHODS: B16.F10 murine tumor cells were exposed to interferon-gamma (IFN_) in culture, and MHC class I expression was quantified using flow cytometry. B16.F10 tumor-bearing C57BI/6 mice were injected with a single subtherapeutic, intraperitoneal (i.p.) dose of IFN_, and tumors were harvested and stained immunohistochemically for MHC class I. Tumor-bearing mice were then treated with i.p. IFN_ as well as an established, CTL-mediated, immunotherapy strategy (intratumoral injection of an adenovirus vector encoding murine CD40 ligand; AdCD40L). Endpoints measured were tumor size and cure rate. Splenocytes from treated animals were evaluated for their ability to lyse B16.F10 target cells using a ^{5l}chromium release, cytotoxicity assay.

RESULTS: IFN_-treated tumor cells expressed significantly more MHC class I than untreated cells, both *in vitro* and *in vivo.* This upregulation persisted *in vivo* for at least 48 hr after dosing. Tumors from animals receiving both i.p. IFN_ and intratumoral AdCD40L were significantly smaller than those from animals receiving the vector alone ($p<0.02$). 80% (4/5) animals receiving both i.p. IFN_ and intratumoral AdCD40L were cured of their tumors, compared to 40% (2/5) of those receiving the vector alone. Splenocytes from AdCD40L-treated mice lysed IFN_-treated B16.F10 cells more efficiently than untreated B16.F10 cells ($p<0.01$).

CONCLUSIONS: Persistent *in vivo* upregulation of MHC class I occurs following one, subtherapeutic dose of i.p. IFN_ in the B16.F10/C57BL/6 syngeneic tumor model. Upregulating MHC class I, in this fashion, results in enhanced tumor cell killing by CTL induced by an established active immunotherapy strategy, resulting in a higher rate of tumor eradication. Such a strategy may be useful for tumors which express little MHC class I in the clinical setting.

**By Invitation*

F22. Replacement of a Tracheal Defect with a Tissue-Engineered Tracheal Prosthesis: Early Results in Animal Experiments

Jhingook Kim*, Soo Won Suh*, Hojoong Kim*, Ji Youn Shin*, Seoul, South Korea.

OBJECTIVE: Major problems in the development of tracheal prosthesis were anastomotic dehiscence and stenosis, which were attributed to the poor epithelization of the prosthetic graft. We had developed a novel tracheal prosthesis with viable mucosa, which was transplanted from oral cavity, and reported its excellent long-term results after thoracic tracheal replacements in dogs

(ASAIO J 47: 496-500, 2001). In the current study, we have used tissue-engineering technique to make lining mucosa of the prosthesis and evaluated its usefulness in tracheal replacement.

METHODS: The experiments were done in 8 adult mongrel dogs. The abdominal skin (5x10 cm) was harvested. And the epithelial cells were selected, in vitro cultured for 4 weeks, and seeded onto a porous poly-lactic glycolic acid scaffold (6x8 cm) to make a lining mucosa of the tracheal prosthesis. Then, it was mounted onto the prosthesis framework, which was made with polypropylene mesh reinforced with polypropylene rings. The mucosa- lined prosthesis was wrapped with greater omentum of the same dog and placed in the peritoneal cavity for 1 week. Complete surgical resection and replacement of thoracic tracheal segment (5 cm in length, just above the carina) was performed. The serial bronchoscopic examinations were done to evaluate the status of prosthesis.

RESULTS: The animals regained their full activity and survived well with normal activity.. The bronchoscope at 1 week, 1 and 2 month showed no stenosis at the anastomosis.

CONCLUSIONS: We concluded that this highly biocompatible tracheal prosthesis could be very useful for the reconstruction of large, circumferential tracheal defects.

3:10 p.m. BREAK

**By Invitation*

3:25 p.m. GENERAL THORACIC FORUM SESSION

Room 311, Hynes Convention Center

Moderators: Steven J Mentzer
David H Harpole, Jr.

F23. Liposomal Delivery of Heat Shock Protein 72 Into Lung Alveolar Macrophages Downregulates Inflammatory Cytokine Production: Potential Implications For Lung Preservation

Daniel R Meldrum*, Kirstan K Meldrum*, John Brown, Indianapolis, IN.

BACKGROUND: Proinflammatory cytokine production by alveolar macrophages contributes to lung injury following lung ischemia-reperfusion and during lung transplant rejection. Although endogenous heat shock protein 72 (HSP 72) is one of the most potent inhibitors of macrophage proinflammatory monokine production, this protective protein is not normally produced without the clinically unappealing circumstance of heat shock to 41.5 degrees C. Further, there is no known acceptable pharmacologic means of induction. The purpose of this study was to: 1) develop a liposomal delivery system for the transmembrane delivery of intact and functional HSP-72 into lung alveolar macrophage, and 2) determine whether liposomal delivery of HSP72 into lung alveolar macrophage decreases proinflammatory cytokine production (tumor necrosis factor, TNF and interleukin 1, IL-1).

METHODS: Lung macrophages were harvested by bronchoalveolar lavage (BAL; 106 macrophage/ml from normal Spraque-Dawley rats, n=7 animals/group) and treated under ex vivo tissue culture conditions with or without liposomally encapsulated recombinant HSP72 (10 mg/ml for 4 hours). Western blot analysis was used to confirm uptake of HSP72. To verify function of the protein, HSP72 was introduced prior to the induction of inflammatory cytokines (endotoxin; ETX,

50 ng/ml x 24 hrs). Supernatants were assayed for inflammatory cytokine production (tumor necrosis factora, TNFa; interleukin Ib, IL-lb) by enzyme linked immunosorbent assay (ELISA).

RESULTS: This liposomal transfer technique resulted in the successful delivery of HSP72 into lung alveolar macrophage. Liposomally delivered HSP72 also decreased lung macrophage TNFa and IL-lb production (Table). Data: means+SEM; n=7 rats/group; data was analyzed for significance (P<0.001) using two-way ANOVA with Tukey's post-hoc correction (Table: *P<0.001 vs. NS;**P<0.001vs ETX).

Alveolar Macrophage TNF and IL-1 production after liposomal HSP-72

	TNF	**IL-1**
NS	25+/-4	9.8+/-2
ETX	1272+/-79*	322+/-23*
HSP+ETX	23S+/-33**	58+/-10**

CONCLUSIONS: 1) Liposomal delivery of functioning heat shock protein 72 to lung alveolar macrophages is feasible; 2) the liposmal delivery of HSP72 decreases lung alveolar macrophage inflammatory cytokine production; and 3) liposomal delivery of liposomal HSP72 may reduce whole organ inflammation and injury during lung preservation.

**By Invitation*

F24. Transcript Profiling of Patients with NO Lung Adenocarcinomas to Predict Survival

Sunil Singhal*, John Kucharczuk*, Blair Marshall*, Steven M Albelda*, Larry R Kaiser, Philadelphia, PA.

OBJECTIVE: Genomic profiling was used to compare gene expression levels in node negative lung adenocarcinomas and pulmonary tissue in order to identify candidate genes for improved selection of prognostic markers and for targeted therapeutic strategies.

METHODS: RNA extracted from node negative lung adenocarcinomas (15 patients) and non-neoplastic pulmonary tissue (5 patients) was hybridized to oligonucleotide microarray filters containing 44,363 genes. Clinicopathological data including survival outcomes was accumulated. A combination of supervised machine learning algorithms and hierarchical and probabilistic clustering predicted 403 upregulated and 121 downregulated gene expression levels. Quantitative real-time PCR, serial analysis of NIH gene expression (SAGE) libraries, literature review and immunohistochemistry confirmed our results.

RESULTS: Genes consistently upregulated in lung adenocarcinomas function in (i) cell cycle control (ie. STAT1, CDC25), (ii) signaling pathways (ie. VACM-1), and (iii) cellular proliferation (ie. prothymosin, Arp 2/3, EGFR). Genes with loss of function in malignant transformation include pathways involving (i) cellular adhesion (ie. CD31, thrombospondin), (ii) apoptosis (ie. death associated proteins), and (iii) cellular differentiation (ie. tumor associated membrane proteins). Mean follow up was 65 months and multivariate analysis did not demonstrate any clinicopathological feature to predict patient outcome. However, alterations in subsets of genes that function in transcription regulation, signal transduction, and cell motility correlated ($r>0.7, p<0.05$)

with patient outcomes. Several unannotated genes (26 ESTs) with undescribed function were also discovered to have significant ($p<10^{-5}$) differential expression in lung adenocarcinomas.

CONCLUSIONS: These results demonstrate integration of gene expression profiling with clinical data can potentially predict outcomes in patients with node negative lung adenocarcinomas. Transcript profiling has yielded pathways that will have major significance in biological treatments.

**By Invitation*

F25. Effectiveness of a New Bioabsorbable Barrier in Preventing Pleural Adhesions: A Prospective, Randomized, Experimental Study.

Domenico Galetta*, Dominique Grunenwald*, Pierre Validire*, Nicolas Borenstein*, Paris, France.

OBJECTIVE: Postoperative pleural adhesions formation is a well-known phenomenon in thoracic surgery. In re-operated patients pleural adhesions may results in serious perioperative and postoperative complications. The purpose of this experimental study was to assess in an animal model the preventive effect of a new sodium hyaluronate/carboxymethylcellulose membrane/unmodified USP glycerol absorbable adhesion barrier (Sepraflm® II) on pleural adhesions formation. Materials and

METHODS: Eleven sheeps (weight, 60 to 77 kg) underwent thoracotomy followed by parietal pleurectomy. Two additional 5-cm incisions were realized on pulmonary parenchyma and closed by absorbable suture. The animals were randomized, receiving either adhesion barrier (group A, n=6), to cover the exposed lung parenchyma, or no adhesion barrier (group B, n=5). Thoracotomy was closed in the usual manner. The animals were sacrificed at 2 weeks after surgery. A re-thoracotomy was performed and the incidence and the severity of pleural adhesions were estimated. Sections of the lung near to the pulmonary incisions were histologically examined and graded for inflammation and fibrosis.

RESULTS: Ten animals survived until the end point. Group A (mean adhesion score, 0.2±0.44) had no adhesions or filmy avascular adhesions easily taken down with blunt dissection. In contrast, dense and sometimes vascular pleural adhesions were formed in the group B (mean adhesion score, 2.6±0.54), particularly on the thoracotomy way. The incidence and severity of the pleural adhesions between the two groups were statistically significant ($p<0.001$, and $p=0.0062$, respectively). No difference was observed in degree of inflammation and fibrosis.

CONCLUSIONS: In this model, the use of Seprafilm® II, effectively prevented the pleural adhesions formation after major lung and pleural irritation. This adhesion barrier should be investigated in humans to assess its effectiveness in reducing pleural adhesions formation and avoiding major complications in re-operated patients.

**By Invitation*

F26. Altering the Optical Properties of the Bronchial Tree Permits Illumination of an Entire Lung for Photodynamic Therapy

Joseph Friedberg*, Cynthia S Skema*, Jeffrey Burdick*, Arjun G Yodh*, Annamarie D Horan*, Shamus R Carr*, Joseph P Culver*, Philadelphia, PA; Durham, NC; Charlestown, MA.

OBJECTIVE: Photodynamic Therapy (PDT) is an effective cancer treatment, but currently central endobronchial tumors are the only lung cancers considered targets for PDT. Tumors such as diffuse bronchoalveolar carcinoma could potentially be treated with PDT if light could be delivered throughout the entire lung. Even if technically feasible, endobronchial and surface illumination of a lung would result in light delivery to only a small percentage of the total lung volume. The goal of this study was to determine if it is possible alter the optical properties of the bronchial tree to facilitate light delivery and achieve illumination of an entire lung.

METHODS: Ex vivo sheep lungs were inflated with substances with refractive indices (RI) higher (mineral oil), lower (air), and equal (saline) to tissue. A laser fiber with the tip positioned 2 cm above the carina was used to deliver 0.1 watts of 630 nm light. Photographs and quantitative charge coupled device (CCD) images were then obtained.

RESULTS: When the lung was inflated with air, light was not visible to the naked eye at the lung surface. With saline a small area of light was visible, centered about the tip of the laser fiber. With mineral oil, the entire lung illuminated (Figure 1). CCD images allowed quantification of light at the surface of the lung and revealed that mineral oil resulted in a 43 fold increase over saline and 123 times over air.

FIGURE 1 Large panels show 3 separate ex vivo sheep lungs (filled with (A)air, (B)saline, and (C)mineral oil) viewed in the dark and illuminated with 630 nm light introduced at the carinal level. Small insets show the same lungs as viewed with the lights on.

CONCLUSIONS: It is possible to manipulate the optical properties of an ex vivo lung by introducing a substance with a high refractive index into the bronchial tree. This arrangement favors internal reflection and results in a light piping phenomenon, similar to fiberoptic cables, and permits light delivery throughout an entire lung. This technique may provide a way to allow light to be delivered for PDT treatment of certain lung cancers for which effective treatments do not currently exist.

[6]1996-94 AATS Graham Fellow

**By Invitation*

F27. Simultaneous Progression of Oxidative Stress and Neoangiogenesis in Malignant Transformation of Barretts Esophagus

Eero I Sihvo*, Merja I Auvinen*, Aki Koivistoinen*, [1]Ari L Harjula, Jarmo A Salo*, Helsinki, Finland.

OBJECTIVE: Oxidative stress and angiogenesis are important elements in the pathogenesis of inflammatory diseases and cancer. Our aim was to evaluate the role of oxidative stress, antioxidant capacity, and angiogenesis in the metaplasia-dysplasia-adenocarcinoma sequence in Barretts epithelium.

METHODS: In mucosal specimens from 52 patients grouped as: symptomatic gastroesophageal reflux disease, Barretts epithelium, adenocarcinoma in the esophagus, and controls, we measured myeloperoxidase activity, superoxidase dismutase activity, glutathione content, and total aromatic DNA adducts. To evaluate blood vessel densities and angioarcitecture we used immunohistochemistry and a modified whole mount technique: sections stained with endothelium-specific markers PAL-E and EN4, vascular endothelial growth factor (VEGF) and its receptor (VEGFR), matrix metalloproteinases (MMPs), and smooth muscle cell actin.

RESULTS: The metaplasia-dysplasia-carcinoma sequence of Barrett's esophagus revealed progressively increased oxidative stress (increased myeloperoxidase activity), decreased antioxidant capacity (glutathione content), and simultaneous formation of DNA adducts. Pooled data showed negative correlation between glutathione content and DNA adducts (-0.28, $p<0.05$). This sequence was also characterized by increased intensity in microvessels, increasing percentage of immature blood vessels, and overexpression of VEGF-A, VEGFR-2, MMP-2, and MMP-9. In addition, whole mount technique showed 3-dimensional evidence that the rich new vascular bed is highly abnormal with repeated twists, bends or turns even in non-malignant Barretts.

CONCLUSIONS: Simultaneous formation of DNA adducts, increased oxidative stress, decreased antioxidant capacity, and negative correlation between glutathione content and DNA adducts indicate a link between oxidative stress and malignant transformation of Barretts epithelium. At the same time, this transformation acquires angiogenic capacity, strong neovascularization, and abnormal angioarchitecture.

[1]1986-87 Graham Fellow

**By Invitation*

F28. Impact of Retrograde Graft Preservation in Perfadex-based Pig Lung Transplantation

Thorsten Wittwer*, Ulrich F Franke*, Antonia Fehrenbach*, Tim Sandhaus*,

Felix Pfeifer*, Thomas Mueller*, Harald Schubert*, Joachim Richter*,

Thorsten Wahlers*, Jena, Germany; Goettingen, Germany.

OBJECTIVE: Currently, clinical lung preservation is mostly performed by single antegrade flush perfusion. An entirely novel approach is retrograde instillation of a preservation solution through the left atrium with the pulmonary artery used for outflow. However, no comparative studies exist concerning the preservation quality when the low-potassium dextran solution Perfadex is used for retrograde pulmonoplegia.

METHODS: Pig lungs (n=5/group) were preserved with Perfadex (PER) either antegradely (PERant) or retrogradely (PERret). After 28 hours of ischemia, left lung transplantation was performed. Following contralateral lung exclusion, relative oxygenation capacity index (ROCI), hemodynamics and dynamic compliance were monitored for 6 hours. Quantification of pulmonary edema formation was achieved stereologically. Leucocyte infiltration into parenchyma (PMN/alveolus) was evaluated histologically. All results were compared to sham-operated controls. Statistics comprised Kruskal-Wallis and Mann-Whitney test and ANOVA analysis with repeated measurements.

RESULTS: Survival was 100%. Flush perfusion time and oxygenation of retrogradely preserved lungs was superior as compared to antegrade preservation ($p<0.024$) and comparable to sham-operated controls. Dynamic lung compliance did not show any difference between groups. PMN-infiltration was highest in the antegrade group. Volume fraction of intraalveolar edema in PERant lungs was significantly higher as compared to sham-lungs (7.80%+/-5.42% vs. 1.32%+/ -0.21%), while edema formation in PERret organs was as low as in sham-controls.

CONCLUSIONS: Perfadex solution provides sufficient graft function after extended ischemia. However, retrograde application through the pulmonary venous system with simultaneous perfusion of the bronchial circulation can optimize postischemic lung function in terms of oxygenation and intraalveolar edema formation and should therefore be evaluated in larger clinical trials.

**By Invitation*

F29. Inhibition of PI3K/AKT-dependent Survival Pathways by LY294002 Enhances Taxol Cytotoxicity in Non-Small Cell Lung Cancer and Esophageal Cancer Cells.

Dao M Nguyen*, Wilson S Tsai*, G A Chen*, William D Schrump*, David S

Schrump, Bethesda, MD.

OBJECTIVE: An emerging body of literature has indicated the critical role of the PI3K/AKT-dependent survival pathway in the intrinsic resistance of cancer cells to chemotherapeutics. AKT phosphorylates its downstream targets IKK and IkB resulting in activation of the NF-kB pathway (with upregulation of BclXL and cIAP-2) as well as BAD to inactivate this proapoptotic protein, thus creating an antiapoptotic milieu in tumor cells. This study aimed to investigate the ability of LY294002 (LY), a potent PI3K inhibitor, to downregulate the activity of the PI3K/AKT-dependent survival pathways leading to potentiation of the cytotoxic effect of Taxol (Tx) in non-small cell lung cancer (NSCLC) or esophageal cancer (EsC) cells.

METHODS: Cultured NSCLC (H322, H226, H460) and EsC (TE3, TE12 SKGT5, HCE4) cells were treated with sequential Tx (90 minutes) and LY (72 hours) combinations. Cell viability and apoptosis were evaluated by MTT and by TUNEL-based ApoBrdU assays respectively. NF-kB activity was evaluated by functional assay using transient transfection of NF-kB-Luciferase reporter plasmid. Levels of total and phosphorylated AKT and BAD, and of clAP-2, BclXL, IkB were determined by western blots. Bay 11-7082 was used to achieve pharmacologic inhibition of NF-kB activity in cancer cells. Results were derived from 3 independent experiments.

RESULTS: Profound dose-dependent reduction of phosphorylated (activated) AKT was achieved with LY exposure (10 to 40 mM x 24 hours) which was coupled with increased total IkB expression and >50% reduction of NF-kB activity as well as significantly decreased expression of the antiapoptotic proteins BclXL, cAIP-2 and phosphorylated BAD. This was correlated with LY-mediated potentiation of Tx cytotoxicity as evidenced by 4- to 10-fold reduction of Tx IC50 values (concentrations of Tx that mediate 50% cytotoxicity). While either Tx (100-500 nM) or LY (40 mM) treatment caused <10% of cell death, Tx/LY combinations induced profound induction of apoptosis (40% to 60%) in H460, H322 NSCLC and SKGT5, TE3 EsC cells. Inhibition of NF-kB by BAY 11-7082 also profoundly sensitized tumor cells to Tx.

CONCLUSIONS: Inhibition of PI3K/AKT survival pathway by LY sensitizes NSCLC and EsC cells to Tx, which is mediated in part by inhibition of NF-kB pathway. PI3K may prove to be an important molecular target for chemosensitization in lung and esophageal cancers.

**By Invitation*

F30. The Problems of Bleeding and Air Leakage in Pulmonary Redosurgery. Comparison Between Fibrin Sealant and Standard Techniques

P P Brega Massone*, C Lequaglie*, B Magnani, B Conti*, I Cataldo*, Milan

and Pavia, Italy.

OBJECTIVE: Redosurgery represents a consistent problem in thoracic surgery because of the high rate of related expected complications. During reoperations strong adhesions are always present, and performing adhesiolisys it's easy to procure visceral pleura or pulmonary parenchyma lesions causing unavoidably consistent air leak and determining the risk of haemothorax. The aim of our work is to analyse the effectiveness of fibrin glue in preventing or reducing postoperative air leak and bleeding.

METHODS: In our Institute we performed a comparative study on 80 patients submitted to redo-surgery for metachronous lung diseases. Group 1 was composed of 40 subjects treated with fibrin glue apposition (on average 5 ml) on air-leaking zones or on parenchyma lacerations with spray or manual technique; the 40 patients belonging to group 2 have been treated with conventional techniques such as electrocauterization and suture of the widest air-leaking zones or pulmonary lacerations. Surgical approaches were the same in the two groups: rethoracothomy in 33 cases, and resternotomy in 7.

RESULTS: Performed operation were: lobar resection in 4 cases, wedge resections in 16, precision resections in 20. In group 1 we didn't observe any major postoperative complications, while in group 2 we had prolonged air leaks (> 10 days) in 7 cases, and haemothorax in 2. Air leak duration was 2.48±0.89 days, (median: 2, range: 1-4), while in group 2 it was 4.93±5.71 days (median: 3, range: 1-25) [p=0.008]; drain time was 4.67±1.30 days, (median: 4, range: 3-8) in group 1, and 6.87±5.37 days (median: 5, range: 2-26) in group 2 [p=0.013]; in-hospital stay was 6.67±1.30 days (median: 6, range: 5-10) in group 1, and 8.37±5.62 days (median: 8, range: 4-28) in group 2 [p=0.013].

CONCLUSIONS: In presence of wide air leaking and bleeding zones, which are very difficult to control with standard techniques, the apposition of fibrin glue determined an immediate reduction of complications. In our experience fibrin glue led to a significant reduction of air leak duration, drain time, and hospital stay in patients undergoing pulmonary redosurgery.

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

Ballroom, Hynes Convention Center

7:00 p.m. ATTENDEE RECEPTION -

A Return to Camelot at the Kennedy Library and Museum

John F Kennedy Library and Museum *(Separate Subscription)*

**By Invitation*

WEDNESDAY MORNING, MAY 7, 2003

7:00 a.m. EMERGING TECHNOLOGIES AND TECHNIQUES FORUM
(7 Minutes Presentation, 8 Minutes Discussion)

Ballroom, Hynes Convention Center

Moderators: David H Adams
Robert W Emery

T1. Intraoperative Device Closure of Perimembranous Ventricular Septal Defects without Cardiopulmonary Bypass: Preliminary Results of the Perventricular Approach

Zahid Amin*, David A Danford*, Stacey E Froemming*, Jon Lof*, Omaha, NE.

Discussant: Emile A Bacha

OBJECTIVE: In infants undergoing closure of perimembranous ventricular septal defects (PVSD), cardiopulmonary bypass (CPB) remains the single most important factor that prolongs hospital stay and morbidity. In order to prevent the deleterious effects of CPB, a new technique was used to close PVSD under echocardiographc guidance, without CPB. The objective of this study was to evaluate the feasibility of this technique in an animal model.

METHODS: Recenly, a new device designed for PVSD (AGA Medical Corporation, Golden Valey, MN) was introduced. The device has a double-disk design with a short connecting waist. The left ventriclular disk is eccentric in design, to prevent encroachment of the aortic valve leaflet. Eight micro Yucatan pigs with naturally occurring PVSD, underwent closure of the defects with the perventricular technique. The mean age was 4 months and the mean weight was 33 kg (range 17-53 kg). The PVSD size ranged from 3 to 11 mm (median 6 mm). Under general endotracheal anesthesia, median sternotomy was performed and the pericardium incised. Epicaridal echocardiogram was performed for the size and location of the PVSD. A purse string suture was placed on the free wall of the right ventricle (RV). The RV was punctured with an angio-catheter and a wire advanced through the catheter into the RV. The PVSD was crossed with the wire and the wire maneuvered through the aortic valve into the aorta, or advanced into the apex of left ventricle. A 7-French delivery sheath was advanced over the wire into the left ventricle and the wire removed. Appropriate size PVSD device was advanced through the sheath and deployed under epicardial echocardiogram guidance. Two pigs were euthanized after placement of the device and 6 kept alive for 6 months. Cardiac catheterization and angiograms were performed at 3 and at 6 months. The pigs were euthanised after the last angiograms. After euthanasia, the tissue was subjected to gross and microscopic examination.

RESULTS: The procedure was successful in all animals. There was no incidence of device embolization or heart block. There was no aortic insufficiency. Angiograms at 3 and 6 months revealed no residual defect and no encroachment of the aortic valve. Pathologically, the devices were completely covered with neo-endocardium.

CONCLUSIONS: Amplatzer PVSD device appears to be excellent for closure of PVSD in the operating room. This technique appears to be feasible in patients who are high risk candidates for closure in the catheterization laboratory. Morbidity associated with CPB and prolonged hospital

stay is avoided. The procedure can be performed through a mini-thoracotomy. Application of this technique with robotic surgery may obviate the need for sternotomy.

**By Invitation*

T2. Innovative Surgical Modifications to Facilitate a Transcatheter Fontan Completion

Mark Galantowicz*, John P Cheatham*, Craig E Fleishman*, Hamish Munro*, Samuel Weinstein*, Columbus, OH.

Discussant: James Jaggers

OBJECTIVE: An ideal Fontan procedure would minimize complications while maximizing flow through the circuit. We report our experience with a combined surgical/transcatheter approach which enables transcatheter Fontan completion.

METHODS: Modifications of the Glenn procedure include anastomosis of the superior vena-cava(SVC) stump to the pulmonary artery(PA) as a blind pouch, placement of markers at this site as well as the inferior vena-cava(IVC), and placement of an IVC cuff. These modifications allowed successful transcatheter Fontan completion with a covered-stent. At catheterization a guidewire rail is established from the SVC into the blind pouch through the right atrium into the WC. Along this rail a covered-stent is positioned to span the distance from the IVC to the PA. The stent is expanded to establish an intracardiac, non-fenestrated, IVC to PA conduit.

RESULTS: Fourteen children underwent the modified Glenn procedure without any deaths or complications. Five patients, (22-24 months, 10-12.4kg), successfully had a transcatheter Fontan completion. Oxygen saturations increased from 82% to 95%. Mean PA pressures increased 1mmHg. There were no deaths, no blood transfusions, and no effusions. All patients were discharged within 24 hours. Three patients required additional transcatheter procedures, at 2-16weeks, for right to left shunts. All secondary procedures were successful in alleviating shunts and returning oxygen saturations to 95%.

CONCLUSIONS: A collaborative surgical/transcatheter approach can successfully create a non-operative Fontan completion. This approach is conceptually attractive with excellent early results. However, assessment of long-term results in a larger cohort is necessary.

**By Invitation*

T3. Remote Control of Pulmonary Blood Flow: Initial Clinical Experience

Antonio F Corno*, Damien Bonnet*, Nicole Sekarski*, Daniel Sidi*, Pascal Vouhe*, Ludwig K Von Segesser, Lausanne, Switzerland; Paris, France.

Discussant: Duke Edward Cameron

OBJECTIVE: After positive results obtained with a long-term experimental study on mini-pigs, the FloWatch™ (EndoArt S.A., Lausanne, Switzerland), an implantable device for pulmonary artery banding with telemetric control, has been evaluated with a prospective multi-centre clinical trial.

METHODS: From June to September 2002 six patients, mean age 10.6 months (range 1 to 31 months), mean body weight 6.5 kg (range 3.5 to 11.0 kg) underwent pulmonary artery banding with FloWatch™ implantation. Diagnosis was univentricular heart in two patients (one with hypoplastic mitral valve, the other with right isomerism, dextrocardia, total anomalous pulmonary venous connection and patent ductus arteriosus) .complete atrio-ventricular septal defect in two (one with left ventricular dominance and patent ductus arteriosus), one with ventricular septal defect, one with multiple ventricular septal defects and double aortic arch; all of them presented with severe pulmonary hypertension. FloWatch™ implantation was performed through median sternotomy (4) or left thoracotomy (2). Associated procedures were atrioseptectomy on cardio-pulmonary bypass in two patients (in one with division of patent ductus arteriosus), closure of patent ductus arteriosus in one and division of double aortic arch in one.

RESULTS: There were no early or late death, nor re-operation, nor device-related complications, in a mean follow-up of 10 weeks (range 4 to 18 weeks). After the first telemetric regulation of the FloWatch™ the day of surgery, a mean of 3.2 regulations/patient (range 1 to 10) were required to adjust the tightening of the pulmonary artery banding to the clinical needs; 52.6% (=10/19) of telemetric regulations were required within the first post-operative week, 36.8% (=7/19) during the second week and 10.6% (2/19) respectively during the third and the tenth post-operative week; in 79% (=15/19) of cases the regulation was required to further narrow the pulmonary artery, while in 21% (=4/19) to release the pulmonary artery (in 2 of them on emergency basis, because of arterial desaturation and bradycardia).

CONCLUSIONS: a) the initial clinical experience confirmed the adequate functioning of the FloWatch™ as telemetrically adjustable pulmonary artery banding; b) repeated adjustments of the pulmonary artery banding, even weeks after surgery, were dictated by the clinical need in all patients; c) the remote control of pulmonary blood flow was feasible without need for re-operation or invasive procedures; d) in children requiring pulmonary artery banding the therapeutic strategies can be expanded by the use of this promising technology; e) this device should be particularly indicated in patients with transposition of the great arteries requiring left ventricular retraining.

**By Invitation*

T4. Improved Patient Outcomes using a Temporary Atrial Defibrillator for Post Operative Atrial Fibrillation

Amit N Patel*, Baron L Hamman*, Robert F Hebeler Jr.*, Richard E Wood*, Brittany Willey*, Amy Patel*, Harold C Urschel Jr., Dallas, TX.

Discussant: Ralph J Damiano, Jr.

OBJECTIVE: Postoperative Atrial fibrillation (POAF) is the most common complication after cardiac surgery. Medical therapy has significant morbidity with unpredictable results. The goal of this study was to compare epicardial atrial deflbrillation with medical therapy in the management of POAF.

METHODS: An analysis of 200 matched patients undergoing cardiac surgery was performed. Patients with a prior pacemaker/deflbrillator, history of arrhythmia, preoperative antiarrhythmic therapy, and/or IABP were excluded. Temporary epicardial atrial cardioversion wires were placed on the right and left atrium. Bipolar atrial and ventricular pacing wires were also placed. The wires were tested in the operating room. Patients with wires, who went into postoperative AF, were

cardioverted with 6-9 Joules (J) and placed on antiarrhythmic therapy for one week postop. Patients without wires were treated with medical therapy alone.

RESULTS: There were 200 patients enrolled, 100 with cardioversion wires matched with 100 without. Thirty-two patients (32%) with cardioversion wires went into postoperative AF during their hospital stay. Mean time to onset of AF was 2.6±1.4 days after surgery. Thirty-one patients were successfully cardioverted to sinus rhythm with cardioversion, with mean of 6.8±2.1J. Recurrent AF occurred in 5 patients during their hospital stay. All 5 of these patients were cardioverted with a mean of 7.5±1.5J. One patient in the cardioversion wire group converted with medical therapy alone. There were no complications with wire insertion or removal. There were no adverse neurological events in the patients with cardioversion wires. One of the patients in the medical therapy group, who developed AF, had a stroke.

	Cardioversion Wires	**Medical Therapy Only**	**P value**
Postoperative AF (n)	32	32	-
Mean Time to Cardioversion (min)	37 ±23	386 ± 238	<0.05
Recurrence of AF (n)	5	13	<0.05
# of Patients Cardioverted	31	12	
Mean Total Joules Delivered	7.1 ±1.6	310 ±52.2	<0.05
Mean Total Length of Stay for AF pts	6.4 ±2.1	9.2 ±3.1	<0.05
Sinus Rhythm at discharge (n)	32	26	<0.05
Sinus Rhythm at 1 month (n)	32	29	NS

CONCLUSIONS: The use of a temporary atrial defibrillator to resynchronize patients in postoperative AF significantly impacts the return to sinus rhythm and results in an earlier hospital discharge.

**By Invitation*

T5. Successful Performance of the Cox-Maze Procedure on the Beating Heart without Cardiopulmonary Bypass: A Chronic Animal Study Using Bipolar Radiofrequency Ablation

Sydney L Gaynor*, Yosuke Ishii*, Michael D Diodato*, Sunil M Prasad*, Nicholas R Damiano*, Kara M Barnett*, Richard B Schuessler*, Ralph J Damiano Jr., Saint Louis, MO.

Discussant: Michael Argenziano

OBJECTIVE: The Cox-Maze procedure is the gold standard for the surgical treatment of atrial fibrillation (AF) and has proven long-term efficacy. However, its application has been limited by the complexity and invasiveness of the procedure. The cut and sew technique is time consuming and difficult, and requires a prolonged period of cardiopulmonary bypass (CPB). The purpose of this study was to develop a less invasive procedure in which all of the Cox-Maze lesions were performed using bipolar radiofrequency (RF) ablation on the beating heart, off CPB. This study evaluated the efficacy and safety of this new procedure in a chronic porcine model.

METHODS: Six Hanford mini-pigs underwent a median sternotomy and pericardiotomy. All the Cox-Maze lesions were performed on the beating heart utilizing a bipolar RF device. The pigs were survived for 30 days. Atrial function and pulmonary vein anatomy were assessed by high resolution MRI studies pre and post operatively. At reoperation, 256-channel atrial epicardial mapping was performed. Pacing was used to evaluate electrical isolation of the pulmonary veins. Induction of atrial fibrillation was attempted by burst pacing along with the administration of intravenous Neostigmine. The animals were sacrificed and histological assessment was performed with H&E and Masson¢s trichrome stain.

RESULTS: All the animals survived the surgical procedure. There were no perioperative neurological events. At 30 days, burst pacing with cholinergic stimulation failed to induce AF in any animal. Conduction block was confirmed by epicardial mapping. Pulmonary vein isolation was documented by epicardial pacing. MRI assessment revealed no evidence of pulmonary vein stenosis. While atrial ejection fraction decreased from 34 ± 1% preoperatively to 26 ± 4% postoperatively ($p<0.05$), atrial contractility was preserved in every instance. The lesions performed were all transmural. Histological assessment showed no occlusion of the coronary arteries and no significant damage to the coronary sinus, the tricuspid and mitral valves.

CONCLUSIONS: The Cox-Maze procedure can be performed successfully without cardiopulmonary bypass by using bipolar RF energy to create the linear lesions. In this chronic model, this less invasive procedure was safe and did not result in any significant late injury to the pulmonary veins or other vital structures. Clinical trials of this new technology on the beating heart are warranted.

**By Invitation*

T6. Tissue Engineered Trachea Using Sheep Bone Marrow-derived from Mesenchymal Stem Cells with TGF-B2 Released from Biodegradable Microspheres

Koji Kojima*, Ronald A Ignotz*, Toshihiro Kushibiki*, Yasuhiko Tabata*, Lawrence J Bonassar*, Charles A Vacanti*, Worcester, MA; Kyoto, Japan.

Discussant: Paolo Machiarini

OBJECTIVE: Several approaches for tracheal replacement have been described, but a totally satisfactory approach has not yet been achieved. This study was designed to evaluate the feasibility of using autologous sheep bone marrow-derived mesenchymal stem cells (BMSCs) cultured onto polyglycolic acid (PGA) mesh to develop helical engineered cartilage equivalents of a functional tracheal replacement. Several studies have shown that TGF-_2 may help support the chondrocyte phenotype in developing 3D tissue constructs. We also explored the potential benefit of local delivery of TGF-_2 using biodegradable microspheres.

METHODS: Bone marrow was obtained by iliac crest aspiration from 6-month-old sheep and cultured in monolayer for 2 weeks. At confluence, the cells were seeded onto non-woven PGA fiber mesh and cultured *in vitro* for 1 week. Cell-polymer constructs were wrapped around a 20mm diameter x 50 mm length silicone helical template. Constructs were then coated with TGF-_2 (10ng/ml) microspheres. Cell-polymer structures were then implanted into a subcutaneous pocket of a nude rat for 6 weeks. Upon removal GAG content and hydroxyproline were analyzed in both native and tissue engineered trachea (TET). Histologic sections of both native and TET were also stained H&E and Safranin-O.

RESULTS: Cell-polymer constructs using TGF-_2 microspheres formed stiff cartilage *de novo* in the shape of helix after 6 weeks. Control constructs absent of TGF-_2 microspheres appeared to be much stiffer than typical cartilage with an apparently mineralized matrix. The engineered trachea recreated the ring structure similar to that of a normal trachea. Histologi-cal data showed the presence of mature cartilage and no evidence of remaining PGA. Proteoglycan and hydroxyproline contents were also similar to native cartilage levels. Analysis of the composition of non-TGF-_2 supplimented constructs are ongoing.

CONCLUSIONS: This study demonstrated the feasibility of engineered trachea grown using sheep BMSCs as a cell source. Engineering the tracheal equivelants with supplimental TGF-_2 seemed to have a positive effect on retaining a cartilagenous phenotype in the newly forming tissue. This technique appears to be successful at generating a tracheal equivalent that is biochemically, histologically and grossly similar to native tracheal tissue. This has the great potential benefit of an autologous approach for repair of segmental tracheal defects with a minimally invasive harvesting technique.

**By Invitation*

T7. Harnessing the Potential of Stem Cells: From Bone Marrow to Highly Functional Semilunar Heart Valve

Fraser W Sutherland*, Tjorvi E Perry*, Megan C Sherwood*, Ying Yu*, Yutaka Masuda*, Dawn L McLellan*, Cecilia A Garcia*, George C Engelmayr Jr.*, Michael S Sacks*, John E Mayer Jr., Boston, MA; Pittsburgh, PA.
Discussant: JoÃ£o Q Melo

OBJECTIVES: To develop a clinically acceptable paradigm for tissue engineering heart valves using a readily accessible source of stem cells in combination with existing medical grade, biodegradable polymers as basic building blocks.

METHODS: A composite non-woven textile was fabricated from polyglycolic acid and poly L-lactic acid fibers. Strength and stiffness were evaluated as a function of time at intervals of up to 25 weeks using a uniaxial test of tensile strength and three point bending test respectively. A novel method was devised for assembling the selected textile into heart valve scaffolds recapitulating the normal root, leaflet and sinus geometry. Mesenchymal stem cells (MSC) were isolated from ovine bone marrow and characterized by FACS, immuncytochemistry and by their ability to differentiate along two distinct mesodermal cell lineages in suitable induction media. MSCs were then harvested from sheep characterized, expanded, delivered onto heart valve scaffolds and cultured for a period of four weeks in vitro. A sample of cells were harvested from valves prior to implantation and recharacterized. Resulting 21mm semilunar heart valves were implanted into the main pulmonary artery of juvenile sheep (65 ± 4Kg, n=6) and evaluated by epicardial echocardiography and late cardiac catheterization.

RESULTS: The wall of the scaffold withstood wall tension of 270 ± 20 mmHg after 25 weeks degradation (n=6). Flexural rigidity of scaffold leaflets was 18.2 ± 3.1mNmm^2 (n=6) at the proposed time of implantation. MSCs were CD45, CD29*, confirming their origin distinct from that of more numerous hematopoietic cells. MSCs displayed a pattern of intracytoplasmic filamentous markers, vimentin, _-SMA and desmin similar to normal valvar interstitial cells. Recharacterization of cells at surgical implantation confirmed stability of the cell phenotype during culture. Implanted valves displayed maximum systolic gradient 17 ± 2mmHg, effective orifice area 2.1 ± 0.1cm^2 and regurgitation trivial to mild. Peak to peak gradient at 4 months was 28mmHg with no evidence of dilatation, late aneurysm formation or calcification.

CONCLUSIONS: Cells may be isolated from bone marrow that display a phenotype similar to valvar interstitial cells. Stem cells in combination with existing medical grade polymers present a promising strategy to create autologous semilunar heart valves. Such valves display hemodynamic parameters equivalent or better than current mechanical or bioprostheses.

**By Invitation*

T8. The Impella® Recover Microaxial Left Ventricular Assist Device Reduces Mortality for Postcardiotomy Failure. A Three Center Experience

Michael P Siegenthaler*, Kerstin Brehm*, Thomas Strecker*, Thorsten Hanke*, Axel Noetzold*, Michael Weyand*, Hans H Sievers*, Friedhelm Beyersdorf, Freiburg, Erlangen and Luebeck, Germany.

Discussant: Ludwig K von Segesser

OBJECTIVE: We evaluated patient-outcomes and complications associated with the microaxial Impella® Recover left-ventricular-assist-device for postcardiotomy low-output syndrome (LOS). This low-cost device, with a diameter of 7.3mm, is inserted across the aortic valve using a 10mm Dacron graft sewn to the ascending aorta.

METHODS: Impella® Recover-patients were compared to patients treated with intraaortic balloon-pump (IABP) alone. Two risk-scores were used; the H-score:J.Card.Surg.2001;16:72-77 and the THI-score:Ann.Thorac.Surg,1993;55:908-13. Between 1/2001-9/2002, 18 patients were treated with the Impella® Recover for LOS, 93 with IABP alone. Thirteen Impella-patients could not be separated from cardiopulmonary bypass with IABP, 5 were treated with Impella alone (no IABP due to peripheral vascular disease or deemed unnecessary).

RESULTS: No technical problems with device-insertion occurred. Pump-flow was 3.7+/-1.0L/min at 28,000+/-4,400rpm. Support-time was 50+/-42hrs (range10-152hrs). Two devices required repositioning. One device failed (leaking purge-line) and was removed. Hemolysis was minimal (LDH-levels 542 +/-246U/L, Impella-survivors). Overall mortality of IABP-patients was 26%(24/93). Mortality for 22 high-risk IABP-patients (H-score‰¥2) was 63%(14/22) versus 44%(8/18) for all Impella-patients (p=0.18). Cardiac-output data was available in 16 Impella-patients. Patients unable to raise their cardiac-output ‰¥1L/min above the flow of the Impella-LVAD had a 71 % (5/7) mortality versus a mortality of 11 % (1/9) in patients with intrinsic cardiac-output ‰¥1L/min (p=0.04). Comparison of high-risk lABP-patients to Impella-patients with intrinsic cardiac-output ‰¥1L/min showed a significant reduction in mortality (63%vs.1 1%,p=0.015). No reduction of mortality was seen using risk-stratification with the THI-score.

CONCLUSIONS: The Impella®Recover-LVAD provides 3.5-4.0L/min flow. This support is insufficient for patients with virtually absent myocardial function. Mortality in patients with residual intrinsic cardiac-output ‰¥1L/min over Impella-flow is reduced compared to high-risk IABP-patients.

**By Invitation*

T9. Off-Pump Mitral Valve Repair Using the Coapsys™ Device: A Geometric Evaluation in an Animal Model of Functional Mitral Regurgitation

Masahiro Inoue*, Zoran B Popovic*, Kazuyoshi Doi*, Soren Schenk*, Yoshio Ootaki*, Hassan Nemeh*, Michael W Kopcakjr.*, Raymond Dessoffy*, James D Thomas*, Patrick M McCarthy, Kiyotaka Fukamachi*, Cleveland, OH.

Discussant: Farzan Filsoufi

OBJECTIVE: Functional mitral regurgitation (MR) results from dilatation of the mitral valve annulus and/or lateral papillary muscle displacement in dysfunctional left ventricles. This study used echocardiographic methods to evaluate the ability of the Myocor® Coapsys™ device to alter valve geometry acutely to reduce MR in a canine model of functional MR.

METHODS: The Coapsys device was implanted in 8 dogs after induction of MR by rapid ventricular pacing at 230 bpm for an average of 31 ± 4 days. The device consists of two epicardial pads connected by a sub-valvular chord. The posterior pad was positioned at the annular level and centered on the posterior leaflet. The sub-valvular chord bisected the valve coaptation line. The device was sized by reducing its dimension, drawing the posterior annulus toward the anterior annulus until MR was maximally reduced. The annular diameter corresponding to the A2 and P2 leaflet segments was measured in the long axis view at end diastole and end systole (A-P Dimension). Additionally, the projected annular area of the valve in the short axis was measured at end-diastole. Evaluation of valve function was completed as well. All measurements were made during the implant procedure prior to and after implantation of the device.

RESULTS: The table summarizes the pre- and post-implantation results (mean ± SD), along with the p-values of a paired t-test of the data.

Observations	Pre-Implant	Post-Implant	P value
A-P Dimension, End-Diastole (cm)*	2.4 ± 0.1	1.9 ± 0.1	<0.001
A-P Dimension, End-Systole (cm)*	2.1 ± 0.2	1.6 ± 0.1	<0.001
Annular Area (cm^2)*	4.6 ±0.5	3.3 ± 0.5	<0.001
MR grade	3.1 ± 0.7	0.6 ± 0.7	<0.001

* data from one animal was not available due to sub-optimal image quality.

CONCLUSIONS: The Coapsys device significantly reduced A-P dimension throughout the heart cycle and annular area at end-diastole. Further study will be required to assess the chronic stability of the repair in this animal model.

**By Invitation*

T10. Multi-center Robotic Mitral Valve Repair Trial with the da Vinci® System

W. Randolph Chitwood Jr., L Wiley Nifong*, Michael Argenziano*, Craig Smith, Multi-Center Robotic Mitral Repair Group*, Greenville, NC; New York, NY.

Discussant: Friedrich W Mohr

OBJECTIVE: In a prospective phase II FDA trial, robotic mitral valve (MV) repairs were done in 112 patients at 10 centers using the da Vinci® Surgical System (Intuitive Surgical, Sunnyvale, CA). Safety in performing valve repairs using computerized tele-manipulation was studied.

METHODS: After institutional review board approval, informed consent was obtained. Patients had moderate to severe mitral regurgitation (MR). Operative technique included peripheral cardiopulmonary bypass (CPB), a 4-5-cm right mini-thoracotomy, a transthoracic aortic cross-clamp, and antegrade cardioplegia. Successful study endpoint was grade 0 or 1 MR by transthoracic echocardiography (TIE) at one-month post-op.

RESULTS: Valve repairs included quadrangular resections, sliding-plasties, edge-to-edge approximations, and both chordal transfers and replacements. The average age was 56.4 ± 0.09 yrs (mean ± SEM). There were 77 (68.8%) men and 35 (31.2%) women. Valve pathology was myomatous degeneration in 105 (91.1%) with 103 (92.0%) having type II leaflet prolapse. Leaflet repair times averaged 36.7 ± 0.2 mln with annuloplasty times of 39-6 ± 0.1 min. Total robot, aortic cross-clamp, and CPB times were 77.9 ± 0.3 min, 2.1 ± 0.1 hrs, and 2.8 ±0.1 hrs, respectively. On one-month TTE, nine (8.0%) had ‰¥ grade 2 MR, and six (5.4%) of these had re-operations (five replacements, one repair). There were no deaths, strokes, or device related complications.

CONCLUSIONS: Multiple surgical teams performed robotic MV repairs safely early in development of this procedure. Advancements in robotic design and adjunctive technologies may help in the evolution of this minimally invasive technique by decreasing operative times.

**By Invitation*

WEDNESDAY MORNING, MAY 7, 2003

7:00 a.m. CURRENT STATUS OF GLOBAL INITIATIVES IN THE DELIVERY OF CARDIOTHORACIC SURGICAL CARE IN DEVELOPING COUNTRIES
Room 302, Hynes Convention Center

Moderator: James L Cox

8:00 a.m. Activities of the European Academy of Sciences and Arts

Felix H linger, European Academy of Sciences, Salzburg, Austria.

8:10 a.m. Current Status and Initiatives in Southeast Asia and French-Speaking Africa

Alain F Carpentier, Hopital Europeen Georges Pompidou, Paris, France.

8:20 a.m. Current Status and Initiatives in the Russian Federation

J Nilas Young, University of California/Davis, Sacramento, CA.

8:30 a.m. Pediatric Cardiac Surgery Initiatives in Shanghai

Richard A Jonas, Children's Hospital, Boston, MA.

8:40 a.m. The Importance of Research at the Local Level in Developing Countries
Sir Magdi Yacoub, National Health Service, London, UK.

8:50 a.m. Coordination of Global Efforts Among Non-Government-Funded Organizations

Thomas Pezzella, President, The World Heart Foundation, Washington, DC.

9:00 a.m. Panel Discussion

9:30 a.m. Adjourn Session

WEDNESDAY MORNING, MAY 7, 2003

CONTROVERSIES IN CARDIOTHORACIC SURGERY PLENARY SESSION

Ballroom, Hynes Convention Center

9:30 a.m. Topic: Attending Cardiothoracic Surgeons Should Also Be Subject to Work-Hour Regulations
Moderator: D Craig Miller

Pro: David F Dinges, Ph.D.

Con: Irving L Kron

WEDNESDAY MORNING, MAY 7, 2003

CONTROVERSIES IN CARDIOTHORACIC SURGERY: ACQUIRED CARDIAC CONTROVERSIES

Ballroom, Hynes Convention Center

10:30 a.m. Topic: Minimal Access Valve Surgery Compromises the Choice of Operation
Moderator: Freidrich W Mohr

Pro: Tirone E David

Con: W Randolph Chitwood, Jr.

11:15 a.m. Topic: CABG Should be Done On-Pump

Moderator: Hartzell V Schaff

Pro: O. Wayne Isom

Con: Michael J Mack

12:00 noon ADJOURN

WEDNESDAY MORNING, MAY 7, 2003

CONTROVERSIES IN CARDIOTHORACIC SURGERY: GENERAL THORACIC CONTROVERSIES

Room 302, Hynes Convention Center

10:30 a.m. Topic: Induction Therapy is Indicated for Esophageal Cancer
Moderator: Steven J Mentzer

Pro: Richard F Heitmiller

Con: Thomas W Rice

11:15 a.m. Topic: Cancer Screening is Effective and Cost-Effective

Moderator: Keith S Naunheim

Pro: David J Sugarbaker

Con: Mark S Allen

12:00 noon ADJOURN

WEDNESDAY MORNING, MAY 7, 2003

CONTROVERSIES IN CARDIOTHORACIC SURGERY:
CONGENITAL HEART CONTROVERSIES

Room 304, Hynes Convention Center

10:30 a.m. Topic Heart and/or Lung Transplantation in Children Should be Abandoned
Moderator: J William Gaynor

Pro: Constantine Mavroudis

Con: Charles B Huddleston

11:15 a.m. Topic: First Stage Norwood Procedure is Enhanced by Right Ventricular to Pulmonary Artery Conduit
Moderator: Ross M Ungerleider

Pro: Shunji Sano

Con: Edward I Bove

12:00 noon ADJOURN

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* 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.

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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.

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Edgar L. Feinberg, III, M.D., Lafayette, LA

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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 112 individuals to receive Alley-Sheridan Scholarships to attend this course.

THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION and the NATIONAL HEART, LUNG, AND BLOOD INSTITUTE Jointly Sponsored MENTORED CLINICAL SCIENTIST DEVELOPMENT AWARD (K08) provides support for the development of outstanding clinician research scientists for a 5-year period of supervised research experience that integrates didactic studies with laboratory or clinically-based research.

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Browns Mills
McGrath, Lynn B
Camden
Camishion, Rudolph C
DelRossi, Anthony J
Englewood
Ergin, M. Arisan
Fort Lee
Conklin, Edward F
Roberts, Arthur J
New Brunswick
MacKenzie, James W
Scholz, Peter M
Newark
Donahoo, James
Swan, Kenneth G
Pittstown
Garzon, Antonio A
South Orange
Gielchinsky, Isaac
Tenafly
Gerst, Paul H
Wallsh, Eugene
Wyckoff
Adler, Richard H

NEW MEXICO

Albuquerque
Dietl, Charles A
Edwards, W. Sterling
Wernly, Jorge A
Alto
Sutherland, R. Duncan
Buena Vista
Thai, Alan P
Santa Fe
Davila, Julio C
Santa Teresa
Glass, Bertram A
Silver City
Waddell, William R

NEW YORK

Albany
Canver, Charles C
Moores, Darroch W. O.
Bay Shore
Ryan, Bernard J
Bellport
Finnerty, James
Levowitz, Bernard S
Sawyer, Philip N
Buffalo
Bhayana Joginder N
Guiraudon, Gerard M
Hoover, Eddie L
Lajos, Thomas Z
Canandaigua
Craver, William L
Chappaqua
Fell, Stanley C
Dewitt
Parker, Frederick B, Jr
East Amherst
Andersen, Murray N
East Quogne
McCormack, Patricia M
Fayetteville
Effler, Donald B
Fishers Island
Baue, Arthur E
Floral Park
Crastnopol, Philip
Larchmont
Steichen, FeUcien M
Lido Beach
Mines, George L
Millerton
Green, George E
New York
Adams, David H
Altorki, Nasser K
Anagnostopoulos, C. E.
Bains, Manjit S
Boyd, Arthur D
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
Lambert, John J
Lansman, Steven L
Litwak, Robert S
Martini, Nad
McCord, Colin W
Mosca, Ralph S
Oz, Mehraet C
Quaegebeur, Jan M
Redo, S. Frank
Rose, Eric A
Rusch, Valerie W
Smith, Craig R
Spencer, Frank C
Spotnitz, Henry M
Subraraanian, Valavanur
Swanson, Scott J
Tice, David A
Tyras, Denis H
Waters, Paul F
Wichern, Walter, Jr
Wolff, William I
Atlantic Beach
Kerth, Wilham J
Chapel Hill
Bowman, Frederick, Jr
Egan, Thomas M
Keagy, Blair A
Sink, James D
Starek, Peter J
Wilcox, Benson R
Charlotte
Robicsek, Francis
Selle, Jay G
Durham
Anderson, Robert W
D'Amico, Thomas A
Davis, Duane R, Jr
Glower, Donald D
Harpole, David H, Jr
Jones, Robert H
Lowe, James E
Oldham, H. Newland, Jr
Sabiston, David C, Jr
Cleveland
Blackstone, Eugene H
Cosgrove, Delos M
Gillinov, A. Marc
Kirby, Thomas J
Loop, Floyd D
Lyde, Bruce W
McCarthy, Patrick M
Mee, Roger B. B.
Pettersson, Costa B
Rice, Thomas W
Svensson, Lars G
Van Heeckeren, Daniel W
Columbus
Davis, J. Terrance
Kakos, Gerard S
Meckstroth, Charles
Michler, Robert E
Williams, Thomas E, Jr
Wolf, Randall K.
Dayton
DeWall, Richard A

Pittsburgh
Potter, Robert T
Rochester
DeWeese James A
Hicks, George L
Schwartz, Seymour I
Stewart, Scott
Roslyn
Thomson, Norman B, Jr
Wisoff, George
SaranacLake
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
Valhalla
Moggio, Richard A
Reed, George E
Voorheesville
Foster, Eric D

NORTH CAROLINA

Asheville
Kroncke, George M
Takaro, Timothy
Smith, Peter K
Wolfe, Walter G
Young, W. Glenn, Jr
Greensboro
Van Trigt, Peter, III
Greenville
Chitwood, W. Randolph, Jr
Elbeery, Joseph R
High Point
Mills, Stephen A
Highlands
Mullen, Donald C
Winston-Salent
Cordell, A. Robert
Hammon, John W, Jr
Hudspeth, Allen S
Kon, Neal D
Meredith, Jesse H

OHIO

Chagrin Falls
Ankeney, Jay L
Cross, Frederick S
Cincinnati
Albers, John E
Callard, George M
Flege, John B, Jr
Helmsworth, James A
Hiratzka, Loren F
Ivey, Tom D
Merrill, Walter H
Wilson, James M
Wright, Creighton B
Grove City
Kilman, James W
Willoughby
Groves, Laurence K

OKLAHOMA

Jenks
LeBeck, Martin B
Oklahoma City
Elkins, Ronald C
Felton, Warren L, II
Fisher, R. Darryl
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

Abington
Addonizio, V. Paul
Bryn Mawr
Haupt, George J
Templeton, John Y, III
Camp Hill
Pennock, John L
Carlisle
DeMuth, William E, Jr
Chalfont
Frobese, Alfreds
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
Johnstown
Kolff, Jacob
Lancaster
Bonchek, Lawrence I
Philadelphia
Acker, Michael A
Bavaria, Joseph E
Bowles, L. Thompson
Diehl, James T
Edie, Richard N
Edmunds, L. Henry, Jr
Gardner, Timothy J
Gaynor, J. William
Goldberg, Melvyn
Guerraty, Albert J
Hargrove, W. Clark, III
Jacobs, Marshall L
Kaiser, Larry R
MacVaugh, Horace
Sewickley
Clark, Richard E
Wayne
Lemmon, William M
West Chester
DiSesa, Verdi J
Wilkes-Barre
Gmochowski, George E
Wynnewood
Wallace, Herbert W
Yardley
Sommer, George N, Jr

RHODE ISLAND

Providence
Hopkins, Richard A
Moulton, Anthony L
Singh, Arun K

SOUTH CAROLINA

Charleston
Bradham, R. Randolph
Crawford, Fred A, Jr
Kratz, John M
Reed, Carolyn E
Rubin, Joseph W
Sade, Robert M
Swenson, Orvar
Columbia
Almond, Carl H
Greenwood
Bolton J.W. Randolph
Hilton Head Island
Humphrey, Edward W
Landrum
Stayman, Joseph W

TENNESSEE

Nashville
Alford, William, Jr
Bender, Harvey W, Jr
Drinkwater, Davis C, Jr
Gobbel, Walter G, Jr
Philhps, Steven J
Randolph, Judson G
Rankin, J. Scott
Sawyers, John L
Stoney, William S
Thomas, Clarence, Jr

TEXAS

Austin
Tyson, Kenneth R. T.
Bedford
McPhail, Jasper L
Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Jessen, Michael E
Lambert, Cary J
Mack, Michael J
Meyer, Dan M
Plan, Melvin R
Ring, W. Steves
Seybold, William D
Urschel, Harold C, Jr
Dilley
Hood, Richard H, Jr
Galveston
Conti, Vincent R
Zwischenberger, Joseph B
Houston
Burdette, Walter J

Mannion, John D
Rosengard, Bruce R
Shrager, Joseph P
Spray, Thomas L
Wechsler, Andrews
Whitman, Glenn J. R.

Pittsburgh
Hardesty, Robert I
Haider, Brack G, Jr
Keenan, Robert J
Kormos, Robert L
Landreneau, Rodney J
Luketich, James D
Magovern, George J
Magovern, George J, Jr
Magovern, James A
Pontius, Robert G
Rams, James J
Siewers, Ralph D

Johnson City
Pennington, D. Glenn

Jonesborongh
Bryant, Lester R

Knoxville
Blake, Hu Al
Brott, Walter H
Domm, Sheldon E

Memphis
Mainwaring, Richard D
McBurney, Robert P
Pate, James W
Robbins, S. Gwin, Sr
Rosensweig, Jacob
Shochat, Stephen J
Watson, Donald C
Weiman, Darryl S

Cooley, Denton A
Coselli, Josephs
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
Hood, R. Maurice

San Antonio
Cidhoon, John H
Dooley, Byron N
Treasure, Robert L

UTAH

Salt Lake City
Doty, Donald B
Hawkins, John A
Jones, Kent W
Karwande, Shreekanth V
Liddle, Harold V
McGough, Edwin C
Mortensen, J. D.
Nelson, Russell M

VERMONT

Burlington
Leavitt, Bruce J

Hartland
Marrin, Charles A. S.

Richford
Grondin, Claude M

VIRGINIA

Altavista
Pierucd, Louis, Jr

Annandale
Akl, Bechara F
Burton, Nelson A
Lefrak, Edward A

Charlottesville
Crosby, Ivan Keith
Dammann, John F
Daniel, Thomas M
Kron, Irving L
Minor, George R
Muller, William H, Jr
Nolan, Stanton P
Tribble, Curtis G
Wellons, Harry A, Jr

Fredericksburg
Armitage, John M

McLean
Wallace, Robert B

Norfolk
Baker, Lenox D

Reston
Boyd, Thomas F

Richmond
Bosher, Lewis H, Jr
Brooks, James W
Lower, Richard R

Springfield
Mills, Mitchell

WASHINGTON

Belfair
Jones, Thomas W

Bellingham
Varco, Richard L

Issaquah
Gentsch, Thomas O

Kirkland
Mills, Waldo O

Mercer Island
Li, Wei-i

Poulsbo
Malette, William G

Seattle
Aldea, Gabriel S
Allen, Margaret D
Anderson, Richard P
Lupinetti, F. Mark
Manhas, Dev R
Mansfield, Peter B
Merendino, K. Alvin
Miller, Donald W, Jr
Rittenhouse, Edward
Sauvage, Lester R
Thomas, George I
Verrier, Edward D
Wood, Douglas F

Spokane
Berg, Ralph, Jr

WEST VIRGINIA

Charleston
Walker, James H

Huntington
Ferraris, Victor A

Morgantown
Graeber, Geoffrey M
Gustafson, Robert A
Hill, Ronald C
Murray, Gordon F
Warden, Herbert E

Parkersburg
Tarnay, Thomas J

WISCONSIN

Altoona
McEnany, M. Terry

Brookfield
Johnson, W. Dudley

Madison
Chopra, Paramjeet S
Young, William P

Marshfield
Myers, William O

Mequon
Narodick, Benjamin

Milwaukee
Almassi, G. Hossein
Haasler, George B
Litwin, S. Bert
Olinger, Gordon N
Tector, Alfred J
Tweddell, James S

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

SOUTH AUSTRALIA
Beaumont
Sutherland, H D'Arcy

AUSTRIA

Leonding
Bruecke, Peter E

Salzburg
Unger, Felix H

Vienna
Hepetko, Waller
Wolner, Ernst

BELGIUM

Aalst
Vanermen, Hugo K. I.

Bertem
Sergeant, Paul T

Leuven
Flameng, Willem J
Lerut, Antoon E. M. R.

BRAZIL

Rio de Janeiro
Meier, Milton A

Sao Paulo
Jatene, Adib D
Oliveira, Sergio A

Sao JosÃ© do Rio Preto
Braile, Domingo M

CANADA

ALBERTA
Calgary
Bharadwaj, Baikunlh
Miller, George E
Edmonton
Gelfand, Elliot T
Koshal, Anind
Rebeyka, Ivan M
Ross, David B
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

ONTARIO
Almonte
Todd, Thomas R J
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
Ottawa
Hendry, Paul J
Keon, Wilbert J
Sundaresan, R. Sudhir
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-Laurent
DesLauriers, Jean

CHINA

Beijing
Ying-Kai, Wu

ENGLAND

Bath, Avon
Belsey, Ronald
Bristol
Angetini, Gianni D.
Cambridge
Kennedy, John H
Wallwork, John
Harcfield
Dreyfus, Gilles D
Herts
Lennox, Stuart C
London
Braimbridge, Mark V
de Leval, Marc R
Goldstraw, Peter
Lincoln, Christopher R
Ross, Donald N
Stark, Jaroslav F
Taylor, Kenneth M
Montpellier
Thevenet, Andre A
Paris
Bachet, Jean E
Blondeau, Philip
Cabrol, Christian E. A.
Carpentier, Alain F
Chauvaud, Sylvain M
Menasche, Phihppe
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

HONG KONG

Shatin, NT
He, Guo-Wei
Yim, Anthony P

INDIA

Chennai
Cherian, K. Mammen

IRELAND

Dublin
O'Malley, Eoin

ITALY

Bergamo
Parenzan, Lucio
Chieti
Calafiore, Antonio M
Milan
Peracchia, Alberto
Naples
Cotrufo, Maurizio
Pisa
Bortolotti, Uberto
Rome
Marcelletti, Carlo

Yacoub, Magdi
Oxford
Westaby, Stephen
Somerset
Abbey-Smith, R
Worcestershire
Landymore, Roderick W

FINLAND

Grankulla
Manila, Severi P
Helsinki
Harjula, Ari L. J

FRANCE

Bordeaux
Fontan, Francis M
Bordeaux-Pessac
Baudet, Eugene M
Creteil
Loisance, Daniel
Le Plessis Robinson
Binet, Jean-Paul
Dartevelle, Philippe G
Marseille
Metros, Dominique R

Beyersdorf, Friedhelm
Hasse, Joachim T. W.
Hamburg
Lacour-Gayet, Francois
Hannover
Haverich, Axel
Leipzig
Mohr, Friedrich W
Loiching
Sebening, Fritz
Munich
Borst, Hans G
Neuss
Bircks, Wolfgang H

GREECE

Athens
Palatianos, George M
Sards, George E

GUATEMALA

Guatemala
Castaneda, Aldo R
Herrera-Llerandi, Rodolfo

Rendina, Erino Angelo

JAPAN

Hamamatsu
Kazui, Terushisa
Kanazawa
Iwa, Takashi
Watanabe, Yoh
Kitakyushushi
Miyamoto, Alfonso T
Kobe
Okita, Yutaka
Minoo City
Kawashima, Yasunaru
Miura Gun
Suma, Hisayoshi
Osaka
Bando, Ko
Kilamura, Soichiro
Matsuda, Hikaru
Sendai
Fujimura, Shigefumi
Mohri, Hitoshi

Shinjuku-ku
Iraai, Yasuharu
Tokyo
Koyanagi, Hiloshi
Kurosawa, Hiromi
Naruke, Tsuguo
Wada, Juro J

KOREA

Seoul
Cho, Bum-Koo

MONACO

Monaco Cedex
Dor, Vincent

NETHERLANDS

Leiden
Dion, Robert
Utrecht
Jansen, Erik W. L.
Wassenaar
Brom, A. Gerard

NEW ZEALAND

Waiwera Auckland
Barratt-Boyes, Brian G

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, Zohair Y

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

Lausanne
vonSegesser, Ludwig K
Fully
Naef, Andreas P
Zurich
Turina, Marko I
Weder, Walter

SYRIA

Damascus
Kabbani, Sami S

VENEZUELA

Caracas
Tricerri, Fernando E

AMERICAN ASSOCIATION FOR THORACIC SURGERY
CHARTER MEMBERS

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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
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

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
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, President-Elect, Vice-President, Secretary, Treasurer, five Councilors and the Editor of the Association shall be a member ex-officio without vote. All members of the Council must be Active or Senior Members of the Association, except that the Editor may be an Honorary Member.

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

a. It may not alter the initiation fees or annual dues, or levy any general assessments against the membership, except that it may, in individual cases, waive annual dues or assessments.
b. It may not change the Articles of incorporation or By-Laws.

c. It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article 111, Section 8.

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

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

ARTICLE V. OFFICERS

Section 1. The officers of the Association shall be President, a President-Elect, 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, President-Elect, 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, President-Elect, and the Vice-President shall be elected for a one-year term of office. The Secretary and the Treasurer shall be elected for a one-year term of office and may be reelected for not more than four additional terms.

Section 4. The President of the Association shall perform all duties customarily pertaining to the office of President. He shall preside at all meeting of the Association and at all meetings of the Council.

Section 5. The President-Elect of the Association shall, in the absence or inability of the President to serve, perform all duties customarily pertaining to the office of President. In this instance the Council shall advance the Vice-President to the office of the President-Elect and appoint an interim Vice-President as necessary.

Section 6. The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve as Secretary of the Association and as Secretary of the Council. When deemed appropriate an Active or Senior Member may be elected to serve as an understudy to the Secretary in anticipation of the latter's retirement from office.

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve a Treasurer of the Association.

Section 8. The Editor of the Association is not an officer of the Association. He shall be appointed by the Council at its annual meeting; provided, however, that such appointment shall not become effective until approved by the Association at the annual meeting of the Association. The Editor shall be appointed for a five-year term and may not be appointed to more than two successive terms; provided, however, that an Editor completing two years or less of the unexpired term of a previous Editor may be appointed for two successive five-year terms. The Editor shall serve as the Editor of the Official Journal and Shall be ex officio the Chairman of the Editorial Board and a member of the Council of the Association without vote.

Section 9. Vacancies occurring among the officers named in Section I or a vacancy in the position of Editor shall be temporarily filled by the Council, subject to approval of the Association at the next meeting of the Association.

ARTICLE VI. COMMITTEES

Section 1. The Council is empowered to appoint a Membership Committee, A Program Committee, a Necrology Committee and such other committees as may in its opinion be necessary or desirable. All such committees shall render their reports at an executive session of the Association, except that no ad hoc committee need report unless so directed by the Council.

Section 2. The Membership Committee shall consist of seven Active or Senior Members. The Council may appoint not more than one of its own members to serve on this Committee. The Duties of the Membership Committee are to investigate all candidates for membership in the Association and to report its findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than five of the members may be reappointed to succeed themselves. This Committee is also charged with maintaining a record of membership attendance and participation in the scientific programs and reporting to the affected members and to the Council any deviations from the requirement of Article VIII, Section 4, of these By-Laws.

Section 3. The Program Committee shall consist of at least 14 members: the President, the President-Elect, the Vice President, the Secretary and the Editor and at least 9 members-at-large, three each representing the areas of adult cardiac, pediatric cardiac and general thoracic surgery. Three of these members-at-large shall be appointed each year by the President for a three-year term. Additional Committee members shall be appointed for one or two-year terms. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

Section 4. The Necrology Committee shall consist of one or more Active or Senior Members. Appointments to this Committee shall be for a one-year term of office. Any or all members of this committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as Chairman of this Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials upon all deaths of members of the Association and to report such deaths at every annual meeting.

Section 5. The Nomination Committee shall consist of the five (5) immediate Past Presidents of the Association. The most senior Past President shall serve as Chairman. This Committee shall prepare a slate of nominees for Officers and Councilors upon instruction from the Council as to the vacancies which are to be filled by election and shall present its report at the Second Executive Session of the Annual Meeting.

Section 6. The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such Committees shall not exceed such annual or special appropriations as may be specifically voted for such purposes by the Association as a whole. Members are urged to cooperate with all Scientific or Research Committees of the Association.

Section 7. The Evarts A Graham Memorial Traveling Fellowship Committee shall consist of eight members: two cardiac surgeons, two general thoracic surgeons, two transplant surgeons, and two pediatric heart surgeons, two to be appointed each year for four year terms with the senior two members of the Committee serving as Co-Chairs. The duties of the Committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and Fellows, past, present and future.

Section 8. The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, ex offlcio, 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 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 te 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 7, 2002

DISCLOSURE INDEX

PROG #	NAME	CONFLICT
1	Aftab R. Kherani, MD	NOTHING TO DISCLOSE
2	Vaughn A. Starnes, MD	NOTHING TO DISCLOSE
3	Alain Serraf, MD, PhD	Did not Disclose
4	Kwang Ree Cho, MD	NOTHING TO DISCLOSE
5	Richard G. Ohye, MD	Did not Disclose
6	Bryan F. Meyers, MD	NOTHING TO DISCLOSE
7	Harold L. Lazar, MD	Did not Disclose
8	Ram Sharony, MD	Did not Disclose
9	Nirav C. Patel, MD	Did not Disclose
10	Lynda L. Mickleborough, MD	Did not Disclose
11	James S. Gammie, MD	Did not Disclose
12	Soon J. Park, MD	Thoratec - Trasming Center, Consultant
13	Sydney L. Gaynor, MD	Atricure - Consultant, Research Grant
14	Marco DiEusanio, MD	Did not Disclose
15	Matthias Karck, MD	Did not Disclose
16	Marc De Perrot, MD	Did not Disclose
17	Steven M. Keller, MD	Did not Disclose
18	Carolyn E. Reed, MD	Did not Disclose
19	Cliff K. Choong, MD	NOTHING TO DISCLOSE
20	Nicolas Aubree, MD	Did not Disclose
21	Steven M. Woolley, MRCS	NOTHING TO DISCLOSE
22	Ara A. Vaporciyan, MD	NOTHING TO DISCLOSE
23	Seth Force, MD	NOTHING TO DISCLOSE
24	Joachim Weipert, MD	NOTHING TO DISCLOSE
25	Kristin K. Galli, MD	Did not Disclose
26	Ali Dodge-Khatami, MD	Did not Disclose
27	Massimo Griselli, MD	Did not Disclose
28	Scott M. Bradley, MD	NOTHING TO DISCLOSE
29	Christian Olsson, MD	Did not Disclose
30	George M. Hoffman, MD	NOTHING TO DISCLOSE
31	Max B. Mitchell, MD	Did not Disclose
32	Gilles D. Dreyfus, MD	Did not Disclose
33	Malcolm V. Brock, MD	Did not Disclose
34	David A. Ashburn, MD	Did not Disclose
35	Claudio Muneretto, MD	Did not Disclose
36	Marc Ruel, MD, MPH	Did not Disclose
37	John S. Ikonomidis, MD, PhD	Did not Disclose
38	Francesco Maisano, MD	Did not Disclose
39	Matthew A. Romano, MD	Did not Disclose
40	Robert S. Farivar, MD, PhD	Did not Disclose
41	Kenji Minakata, MD	NOTHING TO DISCLOSE
42	Sunil K. Bhudia, MD	Did not Disclose
43	George M. Palatianos, MD	Did not Disclose
44	Sudish C. Murthy, MD, PhD	Did not Disclose
45	Michaela Tutic, MD	NOTHING TO DISCLOSE

PROG#	NAME	CONFLICT
46	Peter S. Dahlberg, MD	Did not Disclose
47	Richard J. Battafarano, MD, PhD	NOTHING TO DISCLOSE
48	David J. Sugarbaker, MD	Did not Disclose
49	Thomas W. Rice, MD	Did not Disclose
50	Michael B. Wallace, MD, MPH	Did not Disclose
51	Mark K. Ferguson, MD	NOTHING TO DISCLOSE
52	Bassem N. Mora, MD	Did not Disclose
53	John W. Brown, MD	Did not Disclose
54	Richard G. Ohye, MD	Did not Disclose
55	Kenji Minakata, MD	Did not Disclose

56	Jonathan M. Chen, MD	Did not Disclose
57	Igor E. Konstantinov, MD	Did not Disclose
58	Jan T. Christenson, MD, PhD	NOTHING TO DISCLOSE
59	Anne-Marie McMahon, MBBS	NOTHING TO DISCLOSE
T01	Zahid Amin, MD	Did not Disclose
T02	Mark Galantowicz, MD	NOTHING TO DISCLOSE
T03	Antonio F. Corno, MD	NOTHING TO DISCLOSE
T04	Amit N. Patel, MD	Did not Disclose
T05	Sydney L. Gaynor, MD	NOTHING TO DISCLOSE
T06	Koji Kojima, MD, PhD	Did not Disclose
T07	Fraser W. H. Sutherland, MA, FRCS	Did not Disclose
T08	Michael P. Siegenthaler, MD	Did not Disclose
T09	Masahiro Inoue, MD, PhD	Myocor, Inc./Coapsys - Research grant
T10	W. Randolph Chitwood, Jr., MD	NOTHING TO DISCLOSE
F01	Y. Joseph Woo, MD	NOTHING TO DISCLOSE
F02	Ivan Salvador B. Contreras, MD	NOTHING TO DISCLOSE
F03	Richard J. Myung, MD	NOTHING TO DISCLOSE
F04	Christopher G. A. McGregor, MD	Did not Disclose
F05	Joel S. Corvera, MD	Did not Disclose
F06	Vesa Anttila, MD	NOTHING TO DISCLOSE
F07	Manuel Castella, MD	NOTHING TO DISCLOSE
F08	William M. Yarbrough, MD	Did not Disclose
F09	Akio Ikai, MD	NOTHING TO DISCLOSE
F10	Thomas Yeh, Jr., MD, PhD	Did not Disclose
Fll	Shinji Kanemitsu, MD	Did not Disclose
F12	Takahiko Sakamoto, MD	NOTHING TO DISCLOSE
F13	Elie Fadel, MD, PhD	Did not Disclose
F14	Koji Ueyama, MD	Did not Disclose
F15	Adriano Carotti, MD	NOTHING TO DISCLOSE
F16	Thomas B. Reece, MD	NOTHING TO DISCLOSE
F17	Gavin J. Gordon, PhD	Did not Disclose
F18	Tsutomu Tagawa, MD, PhD	Did not Disclose
F19	Chadrick E. Denlinger, MD	NOTHING TO DISCLOSE
F20	Nirmal Veeramachaneni, MD	NOTHING TO DISCLOSE
F21	Robert E. Merritt, MD	Did not Disclose
F22	Jhingook Kim, MD	Did not Disclose
F23	Daniel R. Meldrum, MD	Did not Disclose

PROG#	**NAME**	**CONFLICT**
F24	Sunil Singhal, MD	NOTHING TO DISCLOSE
F25	Domenico Galetta, MD	NOTHING TO DISCLOSE
F26	Joseph Friedberg, MD	Did not Disclose
F27	Eero I. Sihvo, MD	NOTHING TO DISCLOSE
F28	Thorsten Wittwer, MD	NOTHING TO DISCLOSE
F29	Dao M. Nguyen, MD	Did not Disclose
F30	P. P. Brega Massone,	NOTHING TO DISCLOSE
L01	Jason A. Petrofski, MD	Did not Disclose
L02	Sunil Singhal, MD	Did not Disclose
L03	Matthew R. Mulloy, MD	NOTHING TO DISCLOSE
L04	Subodh Verma, MD, PhD	Did not Disclose
LOS	Thomas S. Maxey, MD	Did not Disclose
L06	Mohsen Karimi, MD	Did not Disclose
L07	Chuong D. Hoang, MD	Did not Disclose
L08	Had R. Mallidi, MD	Did not Disclose