Schedule at a Glance
All scientific sessions and exhibits will take place at the Metro Toronto Convention Centre

Friday, April 30, 2010
1:00 p.m. – 5:00 p.m. Registration Open

Saturday, May 1, 2010
6:30 a.m. – 5:00 p.m. Registration Open
8:00 a.m. – 12:00 p.m. Adult Cardiac Skills
8:00 a.m. – 12:00 p.m. General Thoracic Skills
8:00 a.m. – 12:00 p.m. Congenital Skills
1:00 p.m. – 5:00 p.m. Developing the Academic Surgeon Symposium
1:30 p.m. – 4:20 p.m. Professionalism and the Cardiothoracic Specialty
2:00 p.m. – 4:30 p.m. Robotic Cardiothoracic Surgery Symposium

Sunday, May 2, 2010
6:30 a.m. – 6:00 p.m. Registration Open
8:00 a.m. – 5:00 p.m. Adult Cardiac Surgery Symposium
8:00 a.m. – 5:00 p.m. General Thoracic Surgery Symposium
7:55 a.m. – 5:00 p.m. Congenital Heart Disease Symposium
8:00 a.m. – 5:00 p.m. Cardiothoracic Critical Care Symposium
3:00 p.m. – 5:00 p.m. 13th Annual C. Walton Lillehei Resident Forum
5:00 p.m. – 7:00 p.m. Welcome Reception – Exhibit Hall A & B
5:00 p.m. – 7:00 p.m. Operating Rooms: Hybrid Technologies©
7:00 p.m. Various Satellite Post-Activity Symposia*

Monday, May 3, 2010
6:30 a.m. – 5:00 p.m. Registration Open
9:00 a.m. – 4:30 p.m. Exhibits Open – Exhibit Hall A & B
9:00 a.m. – 4:30 p.m. Operating Rooms: Hybrid Technologies©
7:30 a.m. – 7:45 a.m. Business Session (AATS Members Only)
7:45 a.m. – 12:15 p.m. Plenary Scientific Session
Basic Science Lecture — Susan E. Mackinnon, MD, Washington University School of Medicine
Presidential Address — G. Alec Patterson, MD, Washington University School of Medicine
12:15 p.m. – 2:00 p.m. Lunch – Exhibit Hall A & B
12:15 p.m. – 2:00 p.m. Cardiothoracic Residents’ Luncheon
2:00 p.m. – 5:00 p.m. Simultaneous Scientific Sessions
2:00 p.m. – 5:00 p.m. Educational Program: Building a Hybrid OR of the Future©
7:00 p.m. Various Satellite Post-Activity Symposia*

Tuesday, May 4, 2010
6:30 a.m. – 5:00 p.m. Registration Open
9:00 a.m. – 4:00 p.m. Exhibits Open – Exhibit Hall A & B
9:00 a.m. – 4:00 p.m. Operating Rooms: Hybrid Technologies©
7:00 a.m. – 8:45 a.m. Cardiac Surgery Forum
7:00 a.m. – 8:45 a.m. General Thoracic Surgery Forum
8:00 a.m. – 12:00 p.m. Educational Program: Building a Hybrid OR of the Future©
8:45 a.m. – 12:30 p.m. Plenary Scientific Session
Honored Speaker Lecture — David Naylor, MD, University of Toronto
12:30 p.m. – 2:00 p.m. Lunch – Exhibit Hall A & B
2:00 p.m. – 5:00 p.m. Simultaneous Scientific Sessions
5:00 p.m. – 5:45 p.m. Executive Session (AATS Members Only)
7:00 p.m. – 10:00 p.m. Attendee Reception – Hockey Hall of Fame (ticketed event)

Wednesday, May 5, 2010
6:30 a.m. – 12:00 p.m. Registration Open
7:00 a.m. – 8:45 a.m. Emerging Technologies and Techniques Forum
9:00 a.m. – 10:00 a.m. Controversies in Cardiothoracic Surgery Plenary Session
10:00 a.m. – 12:00 p.m. Simultaneous Scientific Sessions
1:00 p.m. – 5:00 p.m. Endobronchial Ultrasound (EBUS) Training Course (Toronto Medical Discovery Tower)

*Industry-sponsored satellite programs are not part of the AATS Annual Meeting
AMERICAN ASSOCIATION FOR THORACIC SURGERY

90th ANNUAL MEETING
Toronto, ON, Canada

TABLE OF CONTENTS

Abstracts .......................................................................................................................... 139
Accreditation ................................................................................................................... 1
Adult Cardiac Skills ....................................................................................................... 64
Adult Cardiac Surgery Symposium ............................................................................. 80
Author Index .................................................................................................................. 19
Basic Science Lecturers .............................................................................................. 429
Building the Hybrid OR of the Future* ...................................................................... 109
By-laws ......................................................................................................................... 445
Cardiothoracic Critical Care Symposium ................................................................... 93
Committees ................................................................................................................... 417
Congenital Heart Disease Symposium ....................................................................... 89
Congenital Skills .......................................................................................................... 70
Council ......................................................................................................................... 416
C. Walton Lillehei Resident Forum Session ................................................................. 97
Developing the Academic Surgeon ............................................................................. 73
Disclosures ..................................................................................................................... 26
Future Meeting Dates ................................................................................................ 600
General Meeting Information ..................................................................................... 1
General Thoracic Skills .............................................................................................. 68
General Thoracic Surgery Symposium ....................................................................... 85
Graham Education and Research Foundation ......................................................... 435
Evarts A. Graham Memorial Traveling Fellowships .................................................... 436
Honored Guest Lectures ............................................................................................. 431
Hospitality Suite .......................................................................................................... 12
Journal of Thoracic and Cardiovascular Surgery ...................................................... 421
Lifetime Achievement Award .................................................................................... 442
Members
  Alphabetically ........................................................................................................... 466
  Geographically ........................................................................................................ 561
  Charter ...................................................................................................................... 587
Necrology .................................................................................................................... 588
Past Meetings and Presidents .................................................................................... 425
Professionalism and the Cardiothoracic Surgery Specialty ....................................... 75
Research Scholars ...................................................................................................... 438
Robotic Cardiothoracic Surgery ................................................................................. 77
Scientific Achievement Award ................................................................................... 441
Scientific Program ...................................................................................................... 64
Speaker and Discussant Guidelines .......................................................................... 14
The Thoracic Surgery Foundation for Research and Education ................................ 589
About AATS

Promoting Scholarship in Thoracic and Cardiovascular Surgery

Founded in 1917, the American Association for Thoracic Surgery (AATS) is an international organization consisting of over 1,200 of the world’s foremost cardiothoracic surgeons representing 35 countries. Surgeons must have a proven record of distinction within the cardiothoracic surgical field and have made meritorious contributions to the extant knowledge base about cardiothoracic disease and its surgical treatment to be considered for membership. The Annual Meeting, research grants and awards, educational symposia and courses, and the AATS official journal, *The Journal of Thoracic and Cardiovascular Surgery*, all strengthen its commitment to science, education and research.

AATS Annual Meeting Accreditation

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

The American Association for Thoracic Surgery designates this educational activity for a maximum of 39 *AMA PRA Category 1 Credits™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.
The American Association for Thoracic Surgery designates the following credit hours:

**Saturday, May 1, 2010 – up to 7.25 hours**
- Adult Cardiac Skills, up to 3.75 hours
- Congenital Skills, up to 3.5 hours
- General Thoracic Skills, up to 3.5 hours
- Developing the Academic Surgeon, up to 3.5 hours
- Professionalism and the Cardiothoracic Surgery Specialty, up to 2.5 hours
- Robotic Cardiothoracic Surgery, up to 2.5 hours

**Sunday, May 2, 2010 – up to 8.5 hours**
- Adult Cardiac Surgery, up to 8.5 hours
- Cardiothoracic Critical Care, up to 8.5 hours
- Congenital Heart Disease, up to 8.25 hours
- General Thoracic Surgery, up to 8 hours
- C. Walton Lillehei Resident Forum, up to 2 hours

**Monday, May 3, 2010 – up to 6.75 hours**
- Plenary Scientific Session, Basic Science Lecture, Presidential Address, up to 4.25 hours
- Adult Cardiac Surgery Simultaneous Session, up to 2.5 hours
- Congenital Heart Disease Simultaneous Session, up to 2.5 hours
- General Thoracic Surgery Simultaneous Session, up to 2.5 hours
Tuesday, May 4, 2010 – up to 7.5 hours
- Adult Cardiac Forum, up to 1.75 hours
- General Thoracic Forum, up to 1.75 hours
- Plenary Scientific Session, Honored Guest Lecture, up to 3.5 hours
- Adult Cardiac Surgery Simultaneous Session, up to 2.25 hours
- Congenital Heart Disease Simultaneous Session, up to 2.25 hours
- General Thoracic Surgery Simultaneous Session, up to 2.25 hours

Wednesday, May 5, 2010 – up to 9 hours
- Emerging Technologies and Techniques Forum, up to 2 hours
- Controversies in Cardiothoracic Surgery, up to 1 hour
- Adult Cardiac: Surgical Therapies for Congestive Heart Failure, up to 2 hours
- Congenital: New Technology in Congenital Heart Disease, up to 2 hours
- General Thoracic: Controversies in the Utilization of New Technology, up to 2 hours
- Endobronchial Ultrasound (EBUS) Training Course, up to 4 hours

For further information on the Accreditation Council for Continuing Medical Education (ACCME) standards of commercial support, please visit www.accme.org.
Perfusionist Accreditation

The American Board of Cardiovascular Perfusion designates this educational activity for a maximum of 38.6 Category 1 CEUs.

CME Kiosks

All physicians may obtain CME (Continuing Medical Education) and Perfusion credits at the CME Pavilion located on the 200 Level of the Convention Center adjacent to Registration. The CME Pavilion computers will allow attendees to manage all of their CME/Perfusion credits for the Annual Meeting and/or email their information to their personal email address. At the end of the Annual Meeting, attendees may print their CME/Perfusion certificate and/or Letter of Attendance. Following the Annual Meeting, attendees may access their CME/Perfusion information and/or Letter of Attendance at http://ww2.expocard.com/aat101/login.asp?SHID=5

Statement of Need

Cancer and cardiovascular disease continue to be the leading causes of mortality and morbidity around the globe. Major advances in these conditions continue to be made at a rapid pace. Improvements in diagnostic techniques as well as interventional approaches to treatment, both surgical and percutaneous, challenge the clinical practitioner to remain current. Increasingly sophisticated technology to accomplish these aims is being developed and introduced into clinical practice. Exciting advances in basic and clinical science offer opportunities for participation in scientific studies and clinical trials. All of these elements create a significant educational need for the practicing cardiothoracic surgeon. The AATS Annual Meeting fills this need through a combination of lectures, original scientific presentations and discussion forums.
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Educational Objectives

At the conclusion of the AATS 90th Annual Meeting, through comprehensive lectures and discussions, participants will be able to:

- Assess the latest techniques and current research specifically related to Adult Cardiac Surgery, General Thoracic Surgery and Congenital Heart Disease.
- Analyze the pros and cons of each paper presented to gain an overall perspective of their current practices.
- Select appropriate surgical procedures and other interventions for their own patients based upon results presented.
- Integrate state-of-the-art knowledge into their current practice.
- Identify basic science developments and emerging technologies and techniques across the spectrum of cardiothoracic surgery.

Target Audience

The AATS Annual Meeting is specifically designed to meet the educational needs of:

- Cardiothoracic Surgeons
- Physicians in related specialties including Cardiothoracic Anesthesia, Cardiology, Pulmonology, Radiology, Gastroenterology and Thoracic Oncology
- Fellows and Residents in Cardiothoracic and General Surgical training programs
- Nurses, Physician Assistants and other Allied Health Professionals involved in the care of cardiothoracic surgical patients
- Critical Care Teams
- Medical students with an interest in Cardiothoracic Surgery
Disclosure Policy

It is the policy of the American Association for Thoracic Surgery that any individual who is involved in planning, presenting or is an author on a program designated for AMA Physician’s Recognition Award Category 1 Credit™ must disclose any financial interest or other relationship (grant, research support, consultant, etc.) that individual has with any manufacturer(s) of any commercial product(s) that may be discussed in the individual’s presentation. This information is disclosed to the audience prior to an activity. The AATS has procedures in place if a conflict of interest should arise. In addition, faculty members are asked to disclose when any discussion of unapproved use of pharmaceutical or medical device occurs. Disclosures can be found on page 26 and at www.aats.org/annualmeeting.

Acknowledgements

We would like to thank the following companies for their support of this educational activity through educational grants:

Medtronic
Olympus Medical Systems Group

We would like to thank the following companies for their exhibit support:

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We would like to thank the following companies for their support of the OR of the Future©:

ATS Medical
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Major Contributors:

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Camera, Recording, Cell Phone and No-Smoking Policies
Due to privacy issues, it is the policy of AATS that no cameras are permitted in the meeting sessions or exhibit hall. Please refrain from taking photos in these locations. Audio and videotaping is also prohibited.
For the courtesy of all faculty and participants, please ensure that cell phone ringers are silenced during all sessions. Smoking is not permitted in the Convention Center, Hotels or Special Event Venues.

Coat and Luggage Check
A complimentary coat and luggage check will be located on Level 100, across from Room 104A (Front Street side) of the Convention Center. Service will be provided during the following dates and times:

- Saturday, May 1 6:30 a.m. – 5:00 p.m.
- Sunday, May 2 6:30 a.m. – 7:00 p.m.
- Monday, May 3 6:30 a.m. – 5:00 p.m.
- Tuesday, May 4 6:30 a.m. – 5:00 p.m.
- Wednesday, May 5 6:30 a.m. – 12:30 p.m.

Course Information
Pre-registration for all of the Saturday Courses and Sunday Symposia is required. Due to the large volume of registrants for these courses, we STRONGLY suggest registering Friday or Saturday to avoid delays.

This year badges will be scanned at all Saturday and Sunday ticketed events in place of tickets. Please remember to have your badge ready at the door.
Saturday Courses

The Adult Cardiac Skills Course will take place on Saturday, May 1, 2010 in Constitution 107 of the Metro Toronto Convention Centre from 8:00 a.m. – 12:00 noon. Pre-registration is required.

The Congenital Skills Course will take place on Saturday, May 1, 2010 in Constitution 105 of the Metro Toronto Convention Centre from 8:00 a.m. – 12:00 noon. Pre-registration is required.

The General Thoracic Skills Course will take place on Saturday, May 1, 2010 in Constitution 106 of the Metro Toronto Convention Centre from 8:00 a.m. – 12:00 noon. Pre-registration is required.

The Developing the Academic Surgeon Symposium will take place on Saturday, May 1, 2010 in Constitution 107 of the Metro Toronto Convention Centre from 1:00 p.m. – 5:00 p.m. Pre-registration is required.

The Professionalism and the Cardiothoracic Surgery Specialty Symposium will take place on Saturday, May 1, 2010 in Constitution 105 of the Metro Toronto Convention Centre from 1:30 p.m. – 4:20 p.m. Pre-registration is required.

The Robotic Cardiothoracic Surgery Symposium will take place on Saturday, May 1, 2010 in Constitution 106 of the Metro Toronto Convention Centre from 2:00 p.m. – 4:30 p.m. noon. Pre-registration is required.

Sunday AATS/STS Postgraduate Symposia

The Adult Cardiac Surgery Symposium will take place on Sunday, May 2, 2010 in Hall C of the Metro Toronto Convention Centre from 8:00 a.m. – 5:00 p.m. There are two luncheon symposia during the Adult Cardiac course: Luncheon Symposia A – How I Do It: Robotic Mitral Valve Repair “Skin-to-Skin” and Luncheon Symposia B – CABG in 2010: Lessons from the Syntax Trial. Lunch A will take place in Hall C. Lunch B will take place in Constitution 107. Pre-registration is required for the course and either luncheon.
The **Congenital Heart Disease Symposium** will take place on Sunday, May 2, 2010 in Constitution 105 of the Metro Toronto Convention Centre from 7:55 a.m. – 5:00 p.m. A boxed lunch will be offered in the room. Pre-registration is required.

The **General Thoracic Surgery Symposium** will take place on Sunday, May 2, 2010 in Constitution 106 of the Metro Toronto Convention Centre from 8:00 a.m. – 5:00 p.m. A boxed lunch will be offered in the room. Pre-registration is required.

The **Cardiothoracic Critical Care Symposium** will take place on Sunday, May 2, 2010 in Room 201 of the Metro Toronto Convention Centre from 8:00 a.m. – 5:00 p.m. A boxed lunch will be offered in the room. Pre-registration is required.

**Cardiothoracic Residents’ Luncheon – Ticketed Event**

**Chairman:** Thomas L. Spray, MD

The 33rd Annual Cardiothoracic Residents’ Luncheon will begin at 12:15 p.m. on Monday, May 3, 2010 in Room 206 of the Metro Toronto Convention Centre. Physicians in cardiothoracic residency programs and medical students interested in attending this luncheon as guests of the AATS must be pre-registered for the luncheon and will receive a ticket with their registration materials. This year’s luncheon will be hosted by the AATS immediate past president, Thomas L. Spray, MD. There is limited seating and on-site participation will be on a space available basis only.

**Exhibits**

The Exhibit Hall will be in Hall A & B on Level 300 of the Metro Toronto Convention Centre. The Exhibit hall will be open during the following hours:

- **Sunday, May 2** 5:00 p.m. – 7:00 p.m.
- **Monday, May 3** 9:00 a.m. – 4:30 p.m.
- **Tuesday, May 4** 9:00 a.m. – 4:00 p.m.
Welcome Reception and Lunches

A Welcome Reception will be held on Sunday, May 2, 2010 from 5:00 p.m. – 7:00 p.m. in Hall A & B of the Metro Toronto Convention Centre. All registered members, non-members, allied health personnel, spouses and guests are invited to attend. Please note that children will be allowed in the Exhibit Hall during the reception only and must be accompanied by an adult. Children under 16 years of age will not be allowed in the exhibit hall at any other time.

Lunch will be available for purchase in the Exhibit Hall on Monday, May 3 and Tuesday, May 4. Professional registrants will be provided with a coupon ($10 value) found with their name badge to offset the cost of lunch. Complimentary morning and afternoon coffee breaks will also be held in Hall A & B.

Hospitality Suite

The Hospitality Suite, located in the York Room of the Fairmont Royal York, will be open during the following dates and times:

- Sunday, May 2: 8:00 a.m. – 4:00 p.m.
- Monday, May 3: 8:00 a.m. – 4:00 p.m.
- Tuesday, May 4: 8:00 a.m. – 4:00 p.m.
- Wednesday, May 5: 8:00 a.m. – 12:00 p.m.

Staff will be on hand throughout the meeting to greet you, answer your questions and be of assistance to you and your family. Special optional tours have been arranged for Saturday, Sunday, Monday and Tuesday.
Registration
Registration will be located on the 200 Level Foyer (Street Level) of the Metro Toronto Convention Centre. Registration will take place during the following hours:

- Friday, April 30 1:00 p.m. – 5:00 p.m.
- Saturday, May 1 6:30 a.m. – 5:00 p.m.
- Sunday, May 2 6:30 a.m. – 6:00 p.m.
- Monday, May 3 6:30 a.m. – 5:00 p.m.
- Tuesday, May 4 6:30 a.m. – 5:00 p.m.
- Wednesday, May 5 6:30 a.m. – 12:00 p.m.

Suggestions for Registration
a.) Badges must be worn at all times and will be required for admission to the Sessions and Exhibit Hall.

b.) To avoid lines in the registration area, we strongly suggest that attendees register on Friday, April 30 and Saturday, May 1.

c.) House Officers, Fellows, Residents and Medical Students will be admitted without payment of the non-member registration fee if they submitted a verification letter from their Chief of Service or Dean. If the AATS did not receive the verification letter prior to the Annual Meeting, registration fees will be assessed. Registration fees will also be assessed for onsite registration of House Officers, Fellows, Residents and Medical Students.

Special Accessibility Needs
If you require special accommodations to fully participate in the meeting, please visit the Registration Area at the Metro Toronto Convention Centre and an AATS staff member will be happy to assist you.
Speaker Ready Room

The Speaker Ready Room is located in Room 204 of the Metro Toronto Convention Centre. It is critical that ALL speakers visit the Speaker Ready Room at least 2 hours before presenting to ensure their presentation has been properly loaded and any embedded video or media files work with the audiovisual system. The Speaker Ready Room will be open during the following dates and times:

- Friday, April 30: 1:00 p.m. – 6:00 p.m.
- Saturday, May 1: 6:30 a.m. – 6:00 p.m.
- Sunday, May 2: 6:30 a.m. – 6:00 p.m.
- Monday, May 3: 6:30 a.m. – 6:00 p.m.
- Tuesday, May 4: 6:00 a.m. – 6:00 p.m.
- Wednesday, May 5: 6:00 a.m. – 12:00 p.m.

Presentation/Discussion Guidelines

Speaker and Discussant Guidelines

Discussion of Papers: Members, non-member physicians and invited speakers have the privilege of discussing papers. Discussants are limited to 2 minutes and must limit their discussion to specific questions directly related to the author’s presentation. All discussion will be presented from floor microphones and may not be accompanied by slides.
1. Submission and acceptance of an abstract constitutes a commitment by the Author(s) to present the material at the AATS Annual Meeting. The work must not have been submitted, presented or published in abstract or manuscript form elsewhere prior to the AATS 90th Annual Meeting in May 2010. Failure to meet this requirement without prior approval of the AATS will jeopardize a presenter’s further acceptance of abstracts for presentation and/or publication. The AATS Council seriously regards and adheres to the submission/presentation policy and will strictly enforce sanctions upon all authors who fail to meet the policies outlined in the rules for submission and presentation of abstracts once submitted. Any questions should be addressed to the Secretary of the Association, Thoralf M. Sundt, III, MD.

2. Papers presented at the meeting in the Plenary and Simultaneous Sessions shall become the property of the AATS. They shall be submitted electronically to the JTCVS Editor prior to presentation (http://www.editorialmanager.com/jtcvs) and NO LATER than MAY 3, 2010 at 5:00 p.m. EST. To expedite review and publication, the Editor requests that submission occur one month prior to the Annual Meeting.

### Program Presentation Total Discussion

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<td>Lillehei Resident Forum</td>
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<td>Controversies in Cardiothoracic Surgery Debate</td>
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*All discussants are limited to 2 minutes
3. The AATS strongly encourages Cardiac Surgery Forum, General Thoracic Surgery Forum and Lillehei Forum presenters to submit their manuscript to the JTCVS Editor although the requirement for manuscript submission to the JTCVS does not apply to these sessions. Should you submit, papers must be submitted electronically to the JTCVS Editor prior to presentation (http://www.editorialmanager.com/jtcvs/) and NO LATER than MAY 3, 2010 at 5:00 p.m. EST. To expedite review and publication, the Editor requests that submission occur one month prior to the meeting.

**SPECIAL EVENTS**

**Sunday, May 2, 2010**

13th Annual C. Walton Lillehei Resident Forum Session  
Room 206 A & C

Welcome Reception  
Hall A & B

3:00 p.m. – 5:00 p.m.

**Monday, May 3, 2010**

Business Session  
(*AATS Members Only*)  
Hall C

Basic Science Lecture  
Hall C

Presidential Address  
Hall C

Exhibit Hall Lunch  
Hall A & B

7:30 a.m.

10:00 a.m.

11:25 a.m.

12:15 p.m. – 2:00 p.m.
Transportation

Shuttle buses will run continuously between the Fairmont Royal York and Sheraton Centre Toronto Hotel and the Metro Toronto Convention Center.

There will be no regular shuttle transportation from the InterContinental Toronto Centre and the Hyatt Regency Toronto on King due to their close proximity to the Center. However, on Tuesday evening, shuttle
buses will run continuously between the Fairmont Royal York, Inter-Continental Toronto Centre, Sheraton Centre Toronto Hotel and Hyatt Regency Toronto on King to the Attendee Reception at the Hockey Hall of Fame.

Business Center
The Business Services Center is located one level above the Level 200 meeting rooms, at the top of the East Escalators (opposite Hall C). Services provided include photocopying, faxing, printing documents from disk, long distance calling cards, limited office supplies and courier services.

Cell Phone Charging Stations
A cell phone and PDA charging station will be located on Level 200 of Metro Toronto Convention Centre.

Emergencies
For any emergency call 8160 from an internal phone or (416) 586-8160 from your cell phone. Do NOT call 911 as responding emergency staff will not know your precise location.

First Aid
A Nurse’s Station will be located on 200 Level of the Metro Toronto Convention Centre.

Foreign Currency Exchange
The Foreign Currency Exchange is located directly across from Room 203B on Level 200 of the Metro Toronto Convention Centre.
American Association for Thoracic Surgery

Author Index

(Note: Authors are listed by last name, first name and final ID)

A
Aaronson, Stuart F16
Abbas, Ghulam T9
Abbas, Hussain F2
Abel, Stuart L4
Adusumilli, Prasad 18
Aggoun, Yacine 26
Aharinejad, Seyedhossien F18
Ailawadi, Gorav A32, F14, T3
Akhter, Shahab F8
Akiri, Gal F16
Albanese, Sonia 51
Alexandrescu, Clara 7
Alghamdi, Abdullah 48
Ali, Rahmat F2
Allen, Mark* 1
Almassi, G. Hossein* 8
Al-Radi, Osman 23, 48
Altorki, Nasser* 20, 41
Amidi, Morteza 8
Amir, Gail F15
Amorim, Paulo F5
Anderson, Lorraine F1
Andrukhova, Olena F18
Anvari, Farshad L7
Aramini, Beatrice 14
Atlin, Cori 23

B
Bailes, Linda T3
Bailey, Leonard* 47
Bailey, Marci 9, 13
Bains, Manjit* 18
Bakaeen, Faisal 4
Ballman, Karla 1

* AATS Member

Barboza, Laura 21
Bartus, Krzysztof T2
Beghetto, Maurice 22
Bendzsak, Anna 6
Bergeron, Sébastien 19
Bhamidipati, Castigliano 32
Bianchi, Cesario F7, L8
Blackstone, Eugene* 11, 25
Bockeria, Leo* T2
Bolling, Steven* T1
Boodhwani, Munir 40
Borger, Michael* 3
Bothe, Wolfgang L6
Bowles, Dawn L5
Breitenbach, Ingo T2
Brown, John* 30
Bryant, Ayesha 42
Bryant, Roosevelt 49
Bueno, Raphael* 45
Burkhart, Harold 5, 34
Byrne, Guerard F4

C
Cabrera, Jesus F1
Caldarone, Christopher* 23, 25, 48
Camp, Christopher L4
Camp, Tiffany T4
Campbell, David* 27
Carere, Ronald T7
Carotti, Adriano 51
Cerfolio, Robert* 42
Chan, K. M. John T5
Chang, Sung Soo F17
Chen, Huiwen 52
Chen, Ying L2
Cheung, Anson T7
<table>
<thead>
<tr>
<th>Name</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chin, Cynthia</td>
<td>F16</td>
</tr>
<tr>
<td>Choong, Cliff</td>
<td>46</td>
</tr>
<tr>
<td>Christenson, Jan</td>
<td>22, 26</td>
</tr>
<tr>
<td>Christos, Paul</td>
<td>41</td>
</tr>
<tr>
<td>Chu, Danny</td>
<td>4</td>
</tr>
<tr>
<td>Chu, Louis</td>
<td>F7, L8</td>
</tr>
<tr>
<td>Cikirikcioglu, Mustafa</td>
<td>22, 26</td>
</tr>
<tr>
<td>Civaia, Filippo</td>
<td>7</td>
</tr>
<tr>
<td>Clark, Sydne</td>
<td>27</td>
</tr>
<tr>
<td>Coady, Michael</td>
<td>L8</td>
</tr>
<tr>
<td>Cohn, Lawrence*</td>
<td>12</td>
</tr>
<tr>
<td>Coles, John*</td>
<td>48</td>
</tr>
<tr>
<td>Collins, Joseph</td>
<td>8</td>
</tr>
<tr>
<td>Colson, Yolonda*</td>
<td>L3</td>
</tr>
<tr>
<td>Coselli, Joseph*</td>
<td>4</td>
</tr>
<tr>
<td>Couper, Gregory*</td>
<td>12</td>
</tr>
<tr>
<td>Crampton, Melanie</td>
<td>F1</td>
</tr>
<tr>
<td>Crawford, Fred*</td>
<td>39</td>
</tr>
<tr>
<td>D'Armini, Andrea</td>
<td>28</td>
</tr>
<tr>
<td>Damiano, Ralph*</td>
<td>9, 13</td>
</tr>
<tr>
<td>Daneshmand, Mani</td>
<td>L5</td>
</tr>
<tr>
<td>Danton, Mark</td>
<td>33, F9</td>
</tr>
<tr>
<td>Dao, Tam</td>
<td>4</td>
</tr>
<tr>
<td>Darling, Gail*</td>
<td>1, 6</td>
</tr>
<tr>
<td>Dartevelle, Philippe*</td>
<td>28</td>
</tr>
<tr>
<td>Davis, R. Duane*</td>
<td>17</td>
</tr>
<tr>
<td>Dawkins, Keith</td>
<td>29</td>
</tr>
<tr>
<td>de Kerchove, Laurent</td>
<td>40</td>
</tr>
<tr>
<td>De Leyn, Paul*</td>
<td>46</td>
</tr>
<tr>
<td>De Rooij, Peter</td>
<td>46</td>
</tr>
<tr>
<td>Dearani, Joseph*</td>
<td>5, 34</td>
</tr>
<tr>
<td>DeCamp, Malcolm*</td>
<td>16</td>
</tr>
<tr>
<td>DeCampill, William*</td>
<td>25</td>
</tr>
<tr>
<td>Decker, Paul</td>
<td>1</td>
</tr>
<tr>
<td>Deeb, G. Michael*</td>
<td>36</td>
</tr>
<tr>
<td>Demirbas, Seval</td>
<td>F13</td>
</tr>
<tr>
<td>Dent, John</td>
<td>T3</td>
</tr>
<tr>
<td>Deslauriers, Jean*</td>
<td>19</td>
</tr>
<tr>
<td>Devilliers, Pierre</td>
<td>11</td>
</tr>
<tr>
<td>Di Donato, Roberto*</td>
<td>51</td>
</tr>
<tr>
<td>Diener, Amy</td>
<td>36</td>
</tr>
<tr>
<td>Dipetrillo, Thomas</td>
<td>15</td>
</tr>
<tr>
<td>Doenst, Torsten</td>
<td>F5</td>
</tr>
<tr>
<td>Dor, Vincent*</td>
<td>7</td>
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<tr>
<td>D'Ovidio, Frank</td>
<td>14</td>
</tr>
<tr>
<td>Downey, Robert*</td>
<td>18</td>
</tr>
<tr>
<td>Dreyfus, Gilles*</td>
<td>T5</td>
</tr>
<tr>
<td>D'Souza, Karen</td>
<td>F8</td>
</tr>
<tr>
<td>Dycoco, Joseph</td>
<td>18</td>
</tr>
<tr>
<td>Dyer, Anne-Marie</td>
<td>16</td>
</tr>
<tr>
<td>Ekvitting, John-Peder</td>
<td>L6</td>
</tr>
<tr>
<td>El Khoury, Gebrine</td>
<td>40</td>
</tr>
<tr>
<td>El Bardissi, Andrew</td>
<td>12</td>
</tr>
<tr>
<td>Emaminia, Abbas</td>
<td>F14</td>
</tr>
<tr>
<td>Ereth, Mark</td>
<td>L4</td>
</tr>
<tr>
<td>Erne, Barbara</td>
<td>F13</td>
</tr>
<tr>
<td>Fedderly, Raymond</td>
<td>24</td>
</tr>
<tr>
<td>Feng, Jun</td>
<td>L8</td>
</tr>
<tr>
<td>Ferland, Sylvie</td>
<td>19</td>
</tr>
<tr>
<td>Fernandez, Lucas</td>
<td>L7</td>
</tr>
<tr>
<td>Fernando, Hiran*</td>
<td>15</td>
</tr>
<tr>
<td>Figliola, Richard</td>
<td>T4</td>
</tr>
<tr>
<td>Filippelli, Sergio</td>
<td>51</td>
</tr>
<tr>
<td>Finley, Christian</td>
<td>6</td>
</tr>
<tr>
<td>Finley, David</td>
<td>18</td>
</tr>
<tr>
<td>Flickinger, John</td>
<td>16</td>
</tr>
<tr>
<td>Flores, Raja*</td>
<td>18</td>
</tr>
<tr>
<td>Frangioni, John</td>
<td>L3</td>
</tr>
<tr>
<td>Franzen, Olaf</td>
<td>T1</td>
</tr>
<tr>
<td>Frommelt, Michele</td>
<td>24</td>
</tr>
<tr>
<td>Gandy, Kimberly</td>
<td>24</td>
</tr>
<tr>
<td>Gangemi, James</td>
<td>32</td>
</tr>
</tbody>
</table>

* AATS Member
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Garcia, Jose T8
Gavino, Jemmyr Therese T5
Geirsson, Arnar F2
Gelman, Andrew F11
Gillinov, A. Marc* 11
Glineur, David F12
Goddrey, Tony F12
Gooding, William F12
Gopaldas, Raja 4
Gorenstein, Lyall 44
Gotoh, Masashi F17
Greason, Kevin T8
Griffith, Bartley* L3
Grinstaff, Mark L3
Grise, Aaron T1
Grube, Eberhard 51
Guccione, Paolo 5
Guo, L Ray 10
Guthrie, Tracey 43

Hain, Claudia F5
Haney, Jason 39
Hanley, Frank* 21
Harringer, Wolfgang T2
Harris, Catherine 44
Hasaniya, Nahidh F2
Hashim, Sabet 47
Hatfield, Brannon 38
Hejal, Rana T10
Hennessy, Sara 35
Heron, Dwight 15
Hillman, Shauna 15
Hirsch, Joy 44
Holmes, David 29
Hong, Haifa 52
Honjo, Osami 23
Hsia, Tain-Yen T4
Huang, James 18
Huh, Joseph 4
Hwang, Scott 38

Iacono, Aldo T8
Ibrahimie, Ali 44
Igai, Hitoshi F17
Igno-Blanchi, Jonathan F7
Ihekweazu, Ugonna F3
Ikeda, Tadashi 39
Ikonomidis, John* L6
Inculet, Richard 31
Ingels, Neil 15
Ismael, Mahmoud 43
Izhar, Uzi F15

Jacobs, Jeffrey* 25
Jacobs, Marshall* 25
Jaksch, Peter F18
Jarrett, Craig 32
Jeewanandam, Valluvan* F8
Jegatheswaran, Anusha 25
Jenkins, David F13
Jetter, Alexander L2
Johnston, Michael* F19
Jonas, Richard* 25
Jones, David* 1

Kalangos, Afkendiyos* 22
Kappetein, A. Pieter 26
Karamichalis, John 27
Kato, Masaaki 37
Katz, Ruth L2
Kelly, Rosemary* F1
Kempfert, Joerg 3, T6
Kem, John* 32, 35, T3
Keshavjee, Shaf* 6
Khullar, Onkar L3

* AATS Member
<table>
<thead>
<tr>
<th>Name</th>
<th>Page Numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kilgo, Patrick</td>
<td>38</td>
</tr>
<tr>
<td>Kim, Min</td>
<td>L2</td>
</tr>
<tr>
<td>Kim, Richard</td>
<td>F2</td>
</tr>
<tr>
<td>Kin, Keiwa</td>
<td>37</td>
</tr>
<tr>
<td>Kinoshita, Osamu</td>
<td>F10</td>
</tr>
<tr>
<td>Kirshberg, Sophie</td>
<td>F15</td>
</tr>
<tr>
<td>Kitahori, Kazuo</td>
<td>50</td>
</tr>
<tr>
<td>Klepetko, Walter*</td>
<td>28, F18</td>
</tr>
<tr>
<td>Kon, Zachary</td>
<td>T8</td>
</tr>
<tr>
<td>Kontos, Christopher</td>
<td>L5</td>
</tr>
<tr>
<td>Kreisel, Daniel</td>
<td>43, F11</td>
</tr>
<tr>
<td>Kron, Irving*</td>
<td>32, 35, F14, L7, T3</td>
</tr>
<tr>
<td>Krupnick, Sasha</td>
<td>43, F11</td>
</tr>
<tr>
<td>Kuratani, Toru</td>
<td>37</td>
</tr>
<tr>
<td>Lacasse, Yves</td>
<td>19</td>
</tr>
<tr>
<td>Laham, Roger</td>
<td>L8</td>
</tr>
<tr>
<td>Landreneau, Rodney*</td>
<td>1, 15, F12, T9</td>
</tr>
<tr>
<td>Lau, Christine</td>
<td>F14</td>
</tr>
<tr>
<td>Laubach, Victor</td>
<td>L7</td>
</tr>
<tr>
<td>Lawton, Jennifer*</td>
<td>9, 13, F6</td>
</tr>
<tr>
<td>Lazarchick, John</td>
<td>39</td>
</tr>
<tr>
<td>Leadley, Katrin</td>
<td>29</td>
</tr>
<tr>
<td>Lebenthal, Abraham</td>
<td>45</td>
</tr>
<tr>
<td>Lee, Paul*</td>
<td>20, 41</td>
</tr>
<tr>
<td>LeMaire, Scott*</td>
<td>4</td>
</tr>
<tr>
<td>Li, Ming</td>
<td>F19</td>
</tr>
<tr>
<td>Lichtenstein, Samuel</td>
<td>T7</td>
</tr>
<tr>
<td>Lila, Nermine</td>
<td>F4</td>
</tr>
<tr>
<td>Lilley, Stuart</td>
<td>F9</td>
</tr>
<tr>
<td>Lim, Scott</td>
<td>T3</td>
</tr>
<tr>
<td>Lin, Shu</td>
<td>17</td>
</tr>
<tr>
<td>Linden, Philip</td>
<td>T10</td>
</tr>
<tr>
<td>Lindman, Brian</td>
<td>9</td>
</tr>
<tr>
<td>Lindner, Jaroslav</td>
<td>28</td>
</tr>
<tr>
<td>Linke, Axel</td>
<td>3</td>
</tr>
<tr>
<td>Lisec, Asher</td>
<td>2</td>
</tr>
<tr>
<td>Little, Virginia</td>
<td>F12</td>
</tr>
<tr>
<td>Liu, Jiang</td>
<td>F19</td>
</tr>
<tr>
<td>Liu, Jinfen*</td>
<td>52</td>
</tr>
<tr>
<td>Lofland, Gary*</td>
<td>25</td>
</tr>
<tr>
<td>Logan, John</td>
<td>F4</td>
</tr>
<tr>
<td>Lopez, Adriana</td>
<td>L2</td>
</tr>
<tr>
<td>Low, Reginald</td>
<td>T1</td>
</tr>
<tr>
<td>Luketich, James*</td>
<td>F12, T9</td>
</tr>
<tr>
<td>Lyall, Fiona</td>
<td>33, F9</td>
</tr>
<tr>
<td>MacArthur, Kenneth</td>
<td>33, F9</td>
</tr>
<tr>
<td>Mack, Michael*</td>
<td>29</td>
</tr>
<tr>
<td>Malhotra, Sunil</td>
<td>21</td>
</tr>
<tr>
<td>Maloney, Ann</td>
<td>12</td>
</tr>
<tr>
<td>Mandrekar, Sumithra</td>
<td>15</td>
</tr>
<tr>
<td>Manning, Peter*</td>
<td>2</td>
</tr>
<tr>
<td>Manson, Roberto</td>
<td>L5</td>
</tr>
<tr>
<td>Marino, Bradley</td>
<td>2</td>
</tr>
<tr>
<td>Marui, Akira</td>
<td>F3</td>
</tr>
<tr>
<td>Mattioli, Sandro</td>
<td>14</td>
</tr>
<tr>
<td>Mayer, Eckhard</td>
<td>28</td>
</tr>
<tr>
<td>McCaig, David</td>
<td>F9</td>
</tr>
<tr>
<td>McCormick, Brian</td>
<td>T8</td>
</tr>
<tr>
<td>McCrindle, Brian</td>
<td>25</td>
</tr>
<tr>
<td>Mcfalls, Edward</td>
<td>F1</td>
</tr>
<tr>
<td>McGregor, Christopher*</td>
<td>F4</td>
</tr>
<tr>
<td>McKellar, Stephen</td>
<td>L4</td>
</tr>
<tr>
<td>McKenna, Robert*</td>
<td>1</td>
</tr>
<tr>
<td>McQuinn, Tim</td>
<td>T4</td>
</tr>
<tr>
<td>Medford-Davis, Laura</td>
<td>16</td>
</tr>
<tr>
<td>Meinertz, Thomas</td>
<td>T1</td>
</tr>
<tr>
<td>Melby, Spencer</td>
<td>9</td>
</tr>
<tr>
<td>Mertens, Luc</td>
<td>23</td>
</tr>
<tr>
<td>Meyers, Bryan*</td>
<td>15, 43</td>
</tr>
<tr>
<td>Mihaljevic, Tomislav*</td>
<td>11</td>
</tr>
<tr>
<td>Milano, Carmelo*</td>
<td>L5</td>
</tr>
<tr>
<td>Miller, O. Craig*</td>
<td>L6</td>
</tr>
<tr>
<td>Minnich, Douglas</td>
<td>42</td>
</tr>
<tr>
<td>Miro, Santiago</td>
<td>19</td>
</tr>
<tr>
<td>Mirza, Farooq</td>
<td>20</td>
</tr>
<tr>
<td>Misaki, Noriyuki</td>
<td>F17</td>
</tr>
<tr>
<td>Mitchell, Max</td>
<td>27</td>
</tr>
</tbody>
</table>

* AATS Member
## American Association for Thoracic Surgery

<table>
<thead>
<tr>
<th>Name</th>
<th>Number</th>
<th>Number 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitchell, Michael</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Miyamoto, Shelley</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Moazami, Nader</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Mohr, Friedrich W*</td>
<td>3, 29, F5, T6</td>
<td></td>
</tr>
<tr>
<td>Moller, James</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>Montiglio, Françoise</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Moon, Marc*</td>
<td>9, 13</td>
<td></td>
</tr>
<tr>
<td>Motomura, Noboru</td>
<td>F10</td>
<td></td>
</tr>
<tr>
<td>Mueller, Michael</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>Mueller, Ralf</td>
<td>T1</td>
<td></td>
</tr>
<tr>
<td>Mulhearn, Thomas</td>
<td>L5</td>
<td></td>
</tr>
<tr>
<td>Munfakh, Nabil</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Murakami, Arata</td>
<td>50, F10</td>
<td></td>
</tr>
<tr>
<td>Murala, Sanjay</td>
<td>F11</td>
<td></td>
</tr>
<tr>
<td>Muratov, Ravil</td>
<td>T2</td>
<td></td>
</tr>
<tr>
<td>Myers, M. Lee</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Myers, Patrick</td>
<td>22, 26</td>
<td></td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Name</th>
<th>Number</th>
<th>Number 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nava, Ruben</td>
<td>F11</td>
<td></td>
</tr>
<tr>
<td>Nguyen, Christopher</td>
<td>36</td>
<td></td>
</tr>
<tr>
<td>Nguyen, Dao*</td>
<td>F20</td>
<td></td>
</tr>
<tr>
<td>Nguyen, T. Dung</td>
<td>F5</td>
<td></td>
</tr>
<tr>
<td>Nichols, Colin</td>
<td>F6</td>
<td></td>
</tr>
<tr>
<td>Nichols, Francis</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Noirhomme, Phillipe</td>
<td>40</td>
<td></td>
</tr>
</tbody>
</table>

**O**

<table>
<thead>
<tr>
<th>Name</th>
<th>Number</th>
<th>Number 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ono, Minoru</td>
<td>50, F10</td>
<td></td>
</tr>
<tr>
<td>Oosterhuis, Jan</td>
<td>46</td>
<td></td>
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* AATS Member
90TH ANNUAL MEETING MAY 1–MAY 5, 2010
TORONTO, ON, CANADA

FACULTY DISCLOSURE LIST

Michael A. Acker, MD  Faculty member is a Consultant for Acorn Cardiovascular. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David H. Adams, MD  Faculty member is a Consultant for Edwards Lifesciences. Faculty member receives Royalties from Edwards Lifesciences. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

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Seyedhossein Aharinejad, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Gorav Ailawadi, MD  Faculty member is a member of the Speakers Bureau for Evalve. Faculty member plans on discussing unlabeled/investigational uses of a commercial product and will disclose this to the audience.

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Faculty member is a Primary Investigator for Edwards Lifesciences and Medtronic Vascular, Inc. Faculty member is on the Scientific Advisory Board for Maquet. Faculty member has a relationship with Sorin/CarboMedics, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

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Steven F. Bolling, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

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<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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<td>Adriano Carotti, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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<td>Stephen D. Cassivi, MD, MSc</td>
<td>Faculty member is a Chair on the Clinical Events Committee for Spiration, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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<tr>
<td>Robert J. Cerfolio, MD</td>
<td>Faculty member is a Consultant for Medela, CT Device Company, Millicore, E Plus Health Care, Neomend, Sealant Company, Deknatel and Covidien. Faculty member is on the Speakers Bureau for OSI Pharmaceuticals. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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<td>Andrew C. Chang, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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<td>Jonathan M. Chen, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
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Aaron M. Cheng, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does **not** plan on discussing unlabeled/investigational uses of a commercial product.

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Paul De Leyn, MD, PhD  
Faculty member is a Consultant for Covidien. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Marc de Perrot, MD, MSc  Faculty member receives an Honoraria from Actelion. Faculty member plans on discussing unlabeled/investigational uses of a commercial product and will disclose this to the audience.

Abe DeAnda, MD  Faculty member is a member of the Speakers Bureau for Zymogenetics, Inc. Faculty member is a member of the Advisory Board for Oxygen Biotherapeutics. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Malcolm M. DeCamp, MD  Faculty member is a member of the Medical Advisory Board for PneumRX and Portaero. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Pedro J. del Nido, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Todd L. Demmy, MD  Faculty member receives Compensation for Intellectual Property from Covidien. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jean Deslauriers, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Eric J. Devaney, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Todd M. Dewey, MD  
Faculty member is a Consultant for Edwards Lifesciences. Faculty member plans on discussing unlabeled/investigational uses of the Edwards Sapien Valve and will disclose this to the audience.

Jessica S. Donington, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Vincent Dor, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Elena Dubcenco, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Christopher T. Ducko, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Andrew W. El Bardissi, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Martin J. Elliott, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Anthony L. Estrera, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Volkmar Falk, MD  Faculty member is a Consultant for Mitral Solutions, Nycomed and Ventor. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Hiran C. Fernando, MD  Faculty member receives Grant/Research Support from Covidien, Veran Medical and Angidynamics. Faculty member is a Consultant for Ethicon. Faculty member plans on discussing unlabeled/investigational uses of a commercial product and will disclose this to the audience.

Christian John Finley, MD, MPH  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Raja M. Flores, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Charles D. Fraser, MD  Faculty member receives Grant/Research Support from Berlin Heart, Inc. Faculty member plans on discussing unlabeled/investigational uses of The Berlin Heart Pediatric EXCOR Ventricular Assist Device (VAD).

Gregory P. Fontana, MD  Faculty member is a Consultant for St. Jude Medical. Faculty member is a member of the Speakers Bureau for Edwards Lifesciences and Sorin. Faculty member receives Grant/Research Support from Edwards Lifesciences. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Anthony P. Furnary, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Henning A. Gaissert, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

James S. Gammie, MD
Faculty member is a Stockholder for Correx, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Kimberly L. Gandy, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Timothy J. Gardner, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

J. William Gaynor, MD
Faculty member is a Stockholder for JNJ, Medtronic, Inc., BMY, Lilly, Pfizer and GE. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Arnar Geirsson, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Raja R. Gopaldas, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
G. Randall Green, MD, JD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Peter J. Gruber, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jonathan W. Haft, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

John W. Hammon, MD  Faculty member is a Consultant for St. Jude Medical. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Frank L. Hanley, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David H. Harpole, Jr., MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Nahidh W. Hasaniya, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Sara A. Hennessy, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Jennifer C. Hirsch, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Chuong D. Hoang, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Wayne Hofstetter, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Osami Honjo, MD, PhD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Richard A. Hopkins, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Keith A. Horvath, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Tain-Yen Hsia, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Sheng-Shou Hu, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Charles B. Huddleston, MD
Faculty member is a Major Stockholder for Medtronic Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Leonard D. Hudson, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Ali N. Ibrahimiye, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

John S. Ikonomidis, MD, PhD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David M. Jablons, MD
Faculty member is a member of the Speakers Bureau for Lilly, Genentech and Sanofi Aventis. Faculty member is a Stockholder for Pinpoint Genomics. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

James Jaggers, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Robert D.B. Jaquiss, MD
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Anusha Jegatheeswaran, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Richard A. Jonas, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David R. Jones, MD  
Faculty member is a member of the Speakers Bureau for Covidien, Inc. Faculty member receives Grant/Research Support from Millennium, Inc., Merck, Inc. and Aventis, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Robert H. Jones, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Kirk R. Kanter, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Saibal Kar, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of a mitral clip and will disclose this to the audience.

John M. Karamichalis, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Nevin M. Katz, MD  
Faculty member is a Consultant for Baxter Pharmaceuticals and Abbott Diagnostics. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Rosemary F. Kelly, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

John A. Kern, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Kemp H. Kernstine, MD, PhD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Shaf Keshavjee, MD, MSc  
Faculty member receives Grant/Research Support from Wyeth Canada, Astellas Canada, Vitrolife and Novalung. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Kenneth A. Kesler, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Onkar Khullar, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Min P. Kim, MD  
Faculty member is a Major Stockholder for ATSI, GE and JNJ. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Osamu Kinoshita, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Paul M. Kirshbom, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Kazuo Kitahori, MD, PhD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Marin H. Kollef, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Zachary N. Kon, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Nicholas T. Kouchoukos, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Daniel Kreisel, MD, PhD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Irving L. Kron, MD  
Faculty member is a Consultant for St. Jude Medical and Edwards Lifesciences. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Francois Lacour-Gayet, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Christine L. Lau, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jennifer S. Lawton, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Paul C. Lee, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Wendy Levinson, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jerrold H. Levy, MD  Faculty member is on the Steering Committee for Cubist, Canyon, and Novo Nordisk. Faculty member receives Grant/Research Support from Eisai, Schering-Plough and Lilly. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Scott Lim, MD  Faculty member is a Consultant for Evalve. Faculty member plans on discussing unlabeled/investigational uses of Evalve Mitraclip and will disclose this to the audience.
Philip Linden, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of vascular coils for intrabronchial fiducial markers and will disclose this to the audience.

Michael J. Liptay, MD  Faculty member is a member of the Speakers Bureau for Covidien. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Virginia R. Little, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jinfen Liu, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jiang Liu, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Georg Lutter, MD, PhD  Faculty member is a Consultant for Edwards Lifesciences. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Bruce W. Lytle, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Paolo Macchiarini, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Michael J. Mack, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Michael A. Maddaus, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Raj Makkar, MD  Faculty member is a Consultant for Cordis. Faculty member is a member of the Speakers Bureau for Medtronic and Lilly. Faculty member plans on discussing unlabeled/investigational uses of an unprotected left main stent and will disclose this to the audience.

Sunil P. Malhotra, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Peter B. Manning, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Justin A. Mariani, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

M. Blair Marshall, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
David P. Mason, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Robert G. Matheny, MD  Faculty member receives a Salary and Stock from CorMatrix Cardiovascular. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Sandro Mattioli, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Constantine Mavroudis, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

John E. Mayer, Jr, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Eckhard Mayer, MD  Faculty member is a member of the Speakers Bureau for Bayer Schering and Actelion. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Patrick M. McCarthy, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Christopher G.A. McGregor, MD  Faculty member is an inventor of technology related to xenotransplantation that has been licensed by the Mayo Clinic to a commercial entity. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Stephen H. McKellar, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of Dabigatran for mechanical heart valve thromboprophylaxis.

Spencer J. Melby, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Franca M.A. Melfi, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Steven J. Mentzer, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Carlos A. Mestres, MD, PhD  Faculty member is a member of the Advisory Board for Novartis Pharma. Faculty member is a Consultant for Edwards Lifesciences. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Bryan F. Meyers, MD, MPH  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Robert E. Michler, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Tomislav Mihaljevic, MD  
Faculty member is a Consultant for Intuitive Surgical, St. Jude Medical and Edwards Lifesciences. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Carmelo A. Milano, MD  
Faculty member receives Grant/Research Support from Thoratec Corporation, Abiomed, Inc., HeartWare, Inc., St. Jude Medical, Edwards Lifesciences and Sorin Group. Faculty member receives Honorarium from St. Jude Medical. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

D. Craig Miller, MD  
Faculty member is a Consultant for Medtronic Heart Valve Division, Inc. and St. Jude Medical. Faculty member is a member of the Executive Committee for the Percutaneous AVR, Edwards Lifesciences, receiving R01 grant, NHLBI, NIH, HL 39589. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Noriyuki Misaki, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Max B. Mitchell, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Friedrich W. Mohr, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Marc R. Moon, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Ralph S. Mosca, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Michael S. Mulligan, MD  Faculty member is a Consultant for Covidien and Boston Scientific. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Patrick O. Myers, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Ruben G. Nava, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

William I. Norwood, Jr, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Isabelle Opitz, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Francis D. Pagani, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Bernard Joon Hahn Park, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Chan B. Park, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Himanshu J. Patel, MD  Faculty member is a Consultant for Gore & Associates. Faculty member receives Grant/Research Support from Medtronic, Inc. and Cook, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

G. Alec Patterson, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Subroto Paul, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Edward W.K. Peng, MBBS  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Arjun Pennathur, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Andrew Pierre, MD, MSc  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Frank A. Pigula, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Alberto Pochettino, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

John D. Puskas, MD  
Faculty member receives Grant/Research Support from Medtronic, Inc. and Maquet. Faculty member receives Royalties from Scanlan. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Joe B. Putnam, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Vivek Rao, MD, PhD  
Faculty member receives Grant/Research Support from Astellas and Medtronic, Inc. Faculty member is a Consultant for Ventracor. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Federico Rea, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty Member does not plan on discussing unlabeled/investigational uses of a commercial product.

Shekar L. Reddy, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Andrew N. Redington, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David C. Rice, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Michael P. Robich, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Mark D. Rodefeld, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Evelio Rodriguez, MD  
Faculty member receives Grant/Research Support from ATS Medical and Cardionet. Faculty member is a member of the Speakers Bureau for ATS Medical. Faculty member is a member of the Advisory Board for Cardionet. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Eric E. Roselli, MD  
Faculty member is a Consultant for Medtronic and Vascutek. Faculty member receives Grant/Research Support from Cook. Faculty member plans on discussing unlabeled/investigational uses of the thoracic stentgrafts for complex and extensive aortic disease (unlabeled indications) and will disclose this to the audience.

Todd K. Rosengart, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Heather J. Ross, MD  
Faculty member is a member of the Data Safety Monitoring Board for Wyeth. Faculty member is a PI for Norvartis. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Jens C. Ruckert, MD, PhD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Joseph F. Sabik, III, MD  
Faculty member is a member of the Speakers Bureau for Medtronic, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Shunji Sano, MD, PhD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Ralph A. Schmid, MD  
Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Joseph D. Schmoker, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Carsten Schroeder, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of WortX18 vascular coils for lung tissue fiducial marker use.

David S. Schrump, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Matthew J. Schuchert, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Angela Sellitto, MS  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Mark Shapiro, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Andrew D. Shaw, MD  Faculty member is a Consultant for Abbott, Baxter and The Medicines Company. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Charles Sheppard, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Kazuo Shimamura, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Joseph B. Shrager, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Candice K. Silversides, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Nicholas G. Smedira, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Craig R. Smith, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

J. Michael Smith, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Robert L. Smith, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of the Edwards SAPIEN transcatheter heart valve.

W. Roy Smythe, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Lorenzo Spaggiari, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Thomas L. Spray, MD  Faculty Member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

John M. Stulak, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David J. Sugarbaker, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Thoralf M. Sundt, III, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Scott J. Swanson, MD  Faculty member is a Consultant for Covidien and Ethicon Endosurgery. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

David Paul Taggart, MD, PhD  Faculty member is a Consultant for Medtronic, Inc., Novadaq, Abbott Diagnostics, and VGS. Faculty member a member of the Speakers Bureau for Medtronic, Inc. and Novadaq. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
<table>
<thead>
<tr>
<th>Faculty Member</th>
<th>Disclosures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Takahide Takeda, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>John M. Toole, MD</td>
<td>Faculty Member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of recombinant factor seven to control hemorrhage after high risk cardiac surgery.</td>
</tr>
<tr>
<td>Hendrik Treede, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Alfredo Trento, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>James S. Tweddell, MD</td>
<td>Faculty member is a Consultant for Cubist Pharmaceuticals and Schering-Plough. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Eric Vallieres, MD</td>
<td>Faculty member is a Consultant for GSK-Bio, Genentech and Uptake. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Glen S. Van Arsdell, MD</td>
<td>Faculty member is a Minor Stockholder for Medtronic. Faculty member is a Consultant for Cubist Pharmaceuticals. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Name</td>
<td>Disclosure</td>
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</tr>
<tr>
<td>Ara A. Vaporciyan, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Robin Varghese, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Javier T. Varona Santos, PhD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Antony Vassalos, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Giulia Veronesi, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Edward D. Verrier, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Luca A. Vricella, MD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
<tr>
<td>Thomas K. Waddell, MD, MSc, PhD</td>
<td>Faculty member has nothing to disclose with regard to commercial support. Faculty member does <strong>not</strong> plan on discussing unlabeled/investigational uses of a commercial product.</td>
</tr>
</tbody>
</table>
Ori Wald, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Thomas Walther, MD, PhD  Faculty Member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of Edwards SAPIEN transcatheter xenograft.

Thomas J. Watson, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Walter Weder, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Richard D. Weisel, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Francis C. Wells, MD  Faculty member is developing a product with St. Jude Medical. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Daniel C. Wiener, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Betsy White Williams, PhD, MPH  Faculty member is a Major Stockholder for Professional Renewal Center. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Y. Joseph Woo, MD  Faculty Member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of Coapsys and will disclose this to the audience.

Brian J. Woodcock, MB  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Bobby Yanagawa, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Kazuhiro Yasufuku, MD, PhD  Faculty member receives Grant/Research Support from Olympus Medical. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Alan Zajarias, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member plans on discussing unlabeled/investigational uses of transcatheter aortic valve implantation which is currently not FDA approved, however it is available in Europe and will disclose this to the audience.

Marco A. Zenati, MD  Faculty member is a Chair on the Scientific Advisory Board for Cardiorobotics. Faculty member is a member of the Data Safety Monitoring Board for the FDA-approved study entitled CURE-AF sponsored by Medtronic, Inc. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.
Yunge Zhao, MD, PhD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Marcin Zielinski, MD  Faculty member has nothing to disclose with regard to commercial support. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

Joseph B. Zwischenberger, MD  Faculty member is a Patent Holder with Avalon Laboratories. Faculty member does not plan on discussing unlabeled/investigational uses of a commercial product.

AATS Staff  AATS Staff have nothing to disclose with regard to commercial support.
PROGRAM INFORMATION

SATURDAY MORNING
MAY 1, 2010

8:00 a.m. – 12:00 p.m. Adult Cardiac Skills
Constitution 107, Metro Toronto Convention Centre
Chair: Gregory P. Fontana, MD
Cedars-Sinai Heart Institute

COURSE OBJECTIVES
At the conclusion of this course, participants will be able to:

- Analyze and apply optimal strategies for patients in need of coronary artery revascularization.
- Consider the adoption and employment of new techniques in the treatment of mitral valve disease, including a greater understanding of methods to prepare surgeons to safely modify their practice.
- Evaluate emerging transcatheter techniques to facilitate the development of postgraduate strategies to broaden future practice opportunities.
- Develop a greater understanding of the surgeon’s role in the rapidly evolving field of congestive heart failure therapy.

8:00 a.m. – 8:05 a.m.
Introduction
Gregory P. Fontana, MD
Cedars-Sinai Heart Institute

CORONARY ARTERY DISEASE

8:05 a.m. – 8:15 a.m.
Total Arterial Revascularization: Is It Really Necessary?
Joseph F. Sabik, III, MD
Cleveland Clinic Foundation
8:15 a.m. – 8:25 a.m.  Hybrid Revascularization: Is it the Best of Both Worlds?  Volkmar Falk, MD  University of Zürich

8:25 a.m. – 8:35 a.m.  Off Pump CABG: Does it Remain Relevant?  John D. Puskas, MD  Emory University

8:35 a.m. – 8:45 a.m.  CROSSFIRE DISCUSSION

8:45 a.m. – 9:15 a.m.  Debate: Left Main Disease Is No Longer an Absolute Surgical Indication  
Pro: Raj Makkar, MD  Cedars-Sinai Heart Institute  
Con: Michael Mack, MD  Cardiothoracic Surgery Associates of North Texas

9:15 a.m. – 9:30 a.m.  Moving from Median Sternotomy to Minimally Invasive Surgery with or without a Robot  Friedrich W. Mohr, MD, PhD  University of Leipzig

9:30 a.m. – 9:50 a.m.  APPROACHES TO THE MITRAL ANNULUS: RINGS, SLINGS AND CATHETERS  
Important Anatomical and Physiologic Considerations  
David H. Adams, MD  Mount Sinai Medical Center  
New Transcatheter Devices  
Saibal Kar, MD  Cedars-Sinai Medical Center
<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>9:50 a.m. – 10:00 a.m.</td>
<td>Is There a Role for Surgeons in Transcatheter Mitral Procedures? Alfredo Trento, MD Cedars-Sinai Medical Center</td>
</tr>
<tr>
<td>10:00 a.m. – 10:15 a.m.</td>
<td>CROSSFIRE DISCUSSION</td>
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<tr>
<td>10:15 a.m. – 10:30 a.m.</td>
<td>BREAK</td>
</tr>
<tr>
<td>10:30 a.m. – 10:40 a.m.</td>
<td>Transcatheter Cardiac Surgery: Where Will You Be in Five Years? Training, Opportunities and Inevitabilities</td>
</tr>
<tr>
<td>10:40 a.m. – 10:50 a.m.</td>
<td>Aortic Disease Eric E. Roselli, MD Cleveland Clinic Foundation</td>
</tr>
<tr>
<td>10:50 a.m. – 11:00 a.m.</td>
<td>Valvular Heart Disease Anson Cheung, MD University of British Columbia</td>
</tr>
<tr>
<td>11:00 a.m. – 11:15 a.m.</td>
<td>CROSSFIRE DISCUSSION</td>
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<tr>
<td></td>
<td>Structural Heart Disease Georg Lutter, MD University of Kiel</td>
</tr>
</tbody>
</table>
## Congestive Heart Disease

### 11:15 a.m. – 11:25 a.m.
**New Mini-VADs Will Become the Mainstay of Surgical Therapy**  
William E. Cohn, MD, PhD  
Texas Heart Institute

### 11:25 a.m. – 11:45 a.m.
**Is the Solution in Cell Therapy or the Matrix?**  
Robert G. Matheny, MD  
Cardiac Surgical Associates  
John V. Conte, MD  
Johns Hopkins Hospital

### 11:45 a.m. – 12:00 p.m.
**CROSSFIRE DISCUSSION**

### 12:00 p.m.
**ADJOURN**
SATURDAY MORNING
MAY 1, 2010

8:00 a.m. – 12:00 p.m. General Thoracic Skills Course
Constitution 106, Metro Toronto Convention Centre
Chair: Yolonda L. Colson, MD, PhD
Brigham & Women’s Hospital

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

☑ Apply new minimally invasive techniques for the endobronchial biopsy and marking of parenchymal lung lesions (navigational bronchoscopy) and the treatment of diaphragmatic paralysis.

☑ Describe the therapeutic potential for lung stem cells, new agents to minimize lung ischemia reperfusion injury and the artificial lung in the future treatment of end-stage lung disease.

☑ Identify the strengths and limitations associated with Natural Orifice Translumenal Endoscopic Surgery (NOTES) and new minimally invasive approaches to GE reflux.

☑ Relate current clinical outcomes with incidence of occult nodal disease in Stage I NSCLCA and develop future nanotech approaches to lymph node targeting.

8:00 a.m. – 8:10 a.m. Introduction and Course Overview
Yolonda L. Colson, MD, PhD
Brigham & Women’s Hospital

8:10 a.m. – 8:30 a.m. Navigational Bronchoscopy and Endobronchial Fiducial Placement
Philip A. Linden, MD
Case Medical Center/University Hospitals
<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker</th>
<th>Institution</th>
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</thead>
<tbody>
<tr>
<td>8:30 a.m. – 8:50 a.m.</td>
<td>Lung Stem Cells</td>
<td>David M. Jablons, MD</td>
<td>University of California, San Francisco</td>
</tr>
<tr>
<td>8:50 a.m. – 9:10 a.m.</td>
<td>What Is New in Lung Ischemia/Reperfusion</td>
<td>Christine L. Lau, MD</td>
<td>University of Virginia Health System</td>
</tr>
<tr>
<td>9:10 a.m. – 9:30 a.m.</td>
<td>Artificial Lung – Experience with Novalung</td>
<td>Shaf Keshavjee, MD, MSc</td>
<td>Toronto General Hospital</td>
</tr>
<tr>
<td>9:30 a.m. – 9:45 a.m.</td>
<td>DISCUSSION</td>
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<tr>
<td>9:45 a.m. – 10:15 a.m.</td>
<td>BREAK</td>
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<tr>
<td>10:15 a.m. – 10:35 a.m.</td>
<td>Nanotechnology and Lymph Node Targeting</td>
<td>Yolonda L. Colson, MD, PhD</td>
<td>Brigham &amp; Women's Hospital</td>
</tr>
<tr>
<td>10:35 a.m. – 11:05 a.m.</td>
<td>What Is NOTES All About?</td>
<td>Elena Dubcenco, MD</td>
<td>University of Toronto</td>
</tr>
<tr>
<td>11:05 a.m. – 11:25 a.m.</td>
<td>New Techniques in GE Reflux</td>
<td>Virginia R. Little, MD</td>
<td>University of Rochester</td>
</tr>
<tr>
<td>11:25 a.m. – 11:45 a.m.</td>
<td>Clinical Advances in Diaphragm Pacing</td>
<td>Christopher Ducko, MD</td>
<td>Brigham &amp; Women's Hospital</td>
</tr>
<tr>
<td>11:45 a.m. – 12:00 p.m.</td>
<td>DISCUSSION</td>
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<tr>
<td>12:00 p.m.</td>
<td>ADJOURN</td>
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</table>
SATURDAY MORNING
MAY 1, 2010

8:00 a.m. – 12:00 p.m. Congenital Skills Course
Constitution 105, Metro Toronto Convention Centre
Chair: James S. Tweddell, MD
Medical College of Wisconsin

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Identify appropriate candidates for rare and/or complex procedures such as; the Senning procedure, 2-ventricle repair of heterotaxy with double outlet right ventricle and pulmonary outflow obstruction, neonatal suprannular mitral valve replacement, interdigitating arch reconstruction, slide tracheoplasty for tracheal stenosis, placement of a Berlin Heart Excor left ventricular assist device, sutureless repair of pulmonary vein stenosis and for extra-anatomic repair of aortic arch obstruction.
- Describe the steps and pitfalls of these rare and/or complex procedures.
- List the appropriate prosthetic materials to accomplish these procedures including patch material and choice of valves or conduits.

<table>
<thead>
<tr>
<th>8:00 a.m. – 8:05 a.m.</th>
<th>Welcome and Introduction</th>
</tr>
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<tbody>
<tr>
<td>James S. Tweddell, MD</td>
<td></td>
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<tr>
<td>Medical College of Wisconsin</td>
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</tbody>
</table>

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<tr>
<th>8:05 a.m. – 8:20 a.m.</th>
<th>The Senning Procedure as Part of a Double Switch for CCTGA</th>
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</thead>
<tbody>
<tr>
<td>David J. Barron, MD</td>
<td></td>
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<tr>
<td>Birmingham Children’s Hospital</td>
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<tr>
<th>8:20 a.m. – 8:30 a.m.</th>
<th>DISCUSSION</th>
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<tbody>
<tr>
<td>Time</td>
<td>Event</td>
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</tbody>
</table>
| 8:30 a.m. – 8:45 a.m. | Two Ventricle Repair of Heterotaxy with DORV/AVC with PS or PA  
Eric J. Devaney, MD  
University of Michigan |
| 8:45 a.m. – 8:55 a.m. | DISCUSSION                                                        |
| 8:55 a.m. – 9:10 a.m. | Supra-Annular Mitral Valve Replacement in a Neonate  
Thomas L. Spray, MD  
Children's Hospital of Philadelphia |
| 9:10 a.m. – 9:20 a.m. | DISCUSSION                                                        |
| 9:20 a.m. – 9:35 a.m. | Norwood Procedure Interdigitating Arch Reconstruction  
Glenn S. Van Arsdell, MD  
The Hospital for Sick Children |
| 9:35 a.m. – 9:45 a.m. | DISCUSSION                                                        |
| 9:45 a.m. – 10:15 a.m. | BREAK                                                             |
| 10:15 a.m. – 10:30 a.m. | Slide Tracheoplasty  
Carl L. Backer, MD  
Children's Memorial Hospital |
| 10:30 a.m. – 10:40 a.m. | DISCUSSION                                                        |
| 10:40 a.m. – 10:55 a.m. | Implantation of a Berlin Heart VAD  
Robert D.B. Jaquiss, MD  
Arkansas Children's Hospital/University of Arkansas for Medical Sciences |
<p>| 10:55 a.m. – 11:05 a.m. | DISCUSSION                                                        |</p>
<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
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</table>
| 11:05 a.m. – 11:20 a.m. | Sutureless Repair of Pulmonary Vein Stenosis Following Repair of TAPVC  
                     | Christopher A. Caldarone, MD                                         |
|              | The Hospital for Sick Children                                        |
| 11:20 a.m. – 11:30 a.m. | DISCUSSION                                                            |
| 11:30 a.m. – 11:45 a.m. | Extra-anatomic Bypass for Complex Aortic Arch Obstruction            |
|              | Harold M. Burkhart, MD                                               |
|              | Mayo Clinic                                                          |
| 11:45 a.m. – 12:00 p.m. | DISCUSSION                                                            |
| 12:00 p.m.   | ADJOURN                                                              |
SATURDAY AFTERNOON
MAY 1, 2010

1:00 p.m. – 5:00 p.m. Developing the Academic Surgeon Symposium
Constitution 107, Metro Toronto Convention Centre
Chair: David H. Harpole, Jr., MD
Duke University

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Plan and implement a successful career in academic cardiothoracic surgery.
- Design and execute a clinical trial.
- Apply basic and clinical research methods to issues in cardiothoracic surgery.

1:00 p.m. – 1:10 p.m. Introduction and Course Overview
David H. Harpole, Jr., MD
Duke University

1:10 p.m. – 1:30 p.m. The Balancing Act of an Academic Career in Cardiothoracic Surgery
John W. Hammon, MD
Wake Forest University

1:30 p.m. – 1:50 p.m. The Cardiothoracic Surgeon as an Educator
Ara Vaporciyan, MD
MD Anderson Cancer Center

1:50 p.m. – 2:10 p.m. Basic Research in Thoracic Surgery
David R. Jones, MD
University of Virginia Health System
<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
</tr>
</thead>
</table>
| 2:10 p.m. – 2:30 p.m. | Partnering with Industry for Clinical Trials and the FDA  
Erle H. Austin, III, MD  
University of Louisville |
| 2:30 p.m. – 2:50 p.m. | Clinical Research as a Pathway to Academic Advancement  
Eugene H. Blackstone, MD  
Cleveland Clinic Foundation |
| 2:50 p.m. – 3:20 p.m. | BREAK                                                                  |
| 3:20 p.m. – 3:40 p.m. | NIH-Sponsored Cooperative Cancer Groups and Thoracic Surgery  
Bryan F. Meyers, MD, MPH  
Washington University School of Medicine |
| 3:40 p.m. – 4:00 p.m. | NIH-Sponsored Cardiothoracic Surgical Trials Network  
Timothy J. Gardner, MD  
Christiana Care Health System |
| 4:00 p.m. – 4:20 p.m. | An Academic Career in Clinical Practice  
Eric Vallieres, MD  
Swedish Health System |
| 4:20 p.m. – 4:40 p.m. | Cardiothoracic Education in the Future  
Edward D. Verrier, MD  
University of Washington |
| 4:40 p.m. – 5:00 p.m. | DISCUSSION                                                             |
| 5:00 p.m.     | ADJOURN                                                                 |
SATURDAY AFTERNOON
MAY 1, 2010

1:30 p.m. – 4:20 p.m. Professionalism and the Cardiothoracic Surgery Specialty
Constitution 105, Metro Toronto Convention Centre
Chair: Pedro J. del Nido, MD
Children’s Hospital Boston

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Utilize newly gained interpersonal skills to deal with conflict in professional relationships.
- Apply the principles for communicating a positive, professional demeanor to patients.
- Communicate the fundamentals of professional integrity to trainees.

<table>
<thead>
<tr>
<th>1:30 p.m. – 1:40 p.m.</th>
<th>Introduction</th>
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<tbody>
<tr>
<td></td>
<td>Pedro J. del Nido, MD</td>
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<td>Children’s Hospital Boston</td>
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<tr>
<th>1:40 p.m. – 2:00 p.m.</th>
<th>The Charter on Professionalism</th>
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<tbody>
<tr>
<td></td>
<td>Richard L. Cruess, MD</td>
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<td></td>
<td>McGill Centre for Medical Education</td>
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<tr>
<th>2:00 p.m. – 2:20 p.m.</th>
<th>The Doctor Patient Relationship</th>
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<tbody>
<tr>
<td></td>
<td>Wendy Levinson, MD</td>
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<td>University of Toronto</td>
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<tr>
<th>2:20 p.m. – 2:40 p.m.</th>
<th>Communication with Colleagues</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Betsy White Williams, PhD, MPH</td>
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<td></td>
<td>Professional Renewal Center</td>
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| 2:40 p.m. – 3:00 p.m. | BREAK |

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<tr>
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<tbody>
<tr>
<td>3:00 p.m. – 3:30 p.m.</td>
<td><strong>Education and Training on Professional Responsibilities</strong></td>
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<tr>
<td></td>
<td>Richard L. Cruess, MD</td>
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<tr>
<td></td>
<td>McGill Centre for Medical Education</td>
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<tr>
<td></td>
<td>Sylvia R. Cruess, MD</td>
</tr>
<tr>
<td></td>
<td>McGill Centre for Medical Education</td>
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<tr>
<td>3:30 p.m. – 3:50 p.m.</td>
<td><strong>Professionalism and The American Board of Thoracic Surgery</strong></td>
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<td></td>
<td>John E. Mayer, Jr., MD</td>
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<td></td>
<td>Children's Hospital Boston</td>
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<tr>
<td>3:50 p.m. – 4:20 p.m.</td>
<td><strong>DISCUSSION</strong></td>
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<tr>
<td>4:20 p.m.</td>
<td><strong>ADJOURN</strong></td>
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SATURDAY AFTERNOON
MAY 1, 2010

2:00 p.m. – 4:30 p.m. Robotic Cardiothoracic Surgery Symposium
Constitution 106, Metro Toronto Convention Centre
Chairs: W. Randolph Chitwood, Jr., MD
East Carolina University
Franca M.A. Melfi, MD
University of Pisa

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Practice the technique of robotic lobectomy.
- Utilize the device for appropriate surgical procedures for their own patients based upon results presented.
- Develop emerging robotic surgical technique for complex procedures in the field of cardiothoracic surgery.
- Apply basic and clinical research methods to issues in robotic cardiothoracic surgery.

2:00 p.m. – 2:10 p.m.
KEYNOTE INTRODUCTORY LECTURE
W. Randolph Chitwood, Jr., MD
East Carolina University

PART I – NON-CARDIAC THORACIC SURGERY: LUNG/ESOPHAGUS/THYMUS
Chairs: Kemp H. Kernstine, MD, PhD
City of Hope National Medical Center and Beckman Research Institute
Ralph A. Schmid, MD, PhD
University Hospital Berne
<table>
<thead>
<tr>
<th>Time</th>
<th>Session Description</th>
<th>Presenters</th>
</tr>
</thead>
<tbody>
<tr>
<td>2:10 p.m. – 2:20 p.m.</td>
<td>Learning Curve in Robot-Assisted Lobectomy for Lung Cancer</td>
<td>Lorenzo Spaggiari, MD, PhD European Institute of Oncology Giulia Veronesi, MD European Institute of Oncology</td>
</tr>
<tr>
<td>2:20 p.m. – 2:30 p.m.</td>
<td>Strategies for Challenging Major Resections for Lung Cancer</td>
<td>Franca M.A. Melfi, MD University of Pisa</td>
</tr>
<tr>
<td>2:30 p.m. – 2:40 p.m.</td>
<td>Robotic versus Open versus VATS Lymphadenectomy – Feasibility versus Validity</td>
<td>Bernard Joon Hahn Park, MD Memorial Sloan-Kettering Cancer Center</td>
</tr>
<tr>
<td>2:40 p.m. – 2:50 p.m.</td>
<td>Da Vinci-Assisted Thoracoscopic Esophagectomy: Basic Principles and Techniques</td>
<td>Kemp H. Kernstine, MD, PhD City of Hope National Medical Center and Beckman Research Institute</td>
</tr>
<tr>
<td>2:50 p.m. – 3:00 p.m.</td>
<td>Robotic Approaches to Anterior Mediastinum for Thymic Diseases</td>
<td>Federico Rea, MD University of Padua</td>
</tr>
<tr>
<td>3:00 p.m. – 3:20 p.m.</td>
<td>DISCUSSION</td>
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</table>

**PART II – ROBOTIC CARDIAC SURGERY**

*Chairs:* Tomislav Mihaljevic, MD Cleveland Clinic Foundation J. Michael Smith, MD Cardiac, Vascular & Thoracic Surgeons, Inc.
<table>
<thead>
<tr>
<th>Time</th>
<th>Session Title</th>
<th>Presenter</th>
<th>Institution</th>
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</thead>
<tbody>
<tr>
<td>3:20 p.m. – 3:30 p.m.</td>
<td>Principles of Robotic Cardiac Surgery</td>
<td>J. Michael Smith, MD</td>
<td>Cardiac, Vascular &amp; Thoracic Surgeons, Inc.</td>
</tr>
<tr>
<td>3:30 p.m. – 3:40 p.m.</td>
<td>Robotic Mitral Valve Repair – Endoballoon Method</td>
<td>Tomislav Mihaljevic, MD</td>
<td>Cleveland Clinic Foundation</td>
</tr>
<tr>
<td>3:40 p.m. – 3:50 p.m.</td>
<td>Robotic Mitral Valve Repair – Chitwood Clamp Method</td>
<td>Evelio Rodriguez, MD</td>
<td>East Carolina University</td>
</tr>
<tr>
<td>3:50 p.m. – 4:00 p.m.</td>
<td>Robotic Coronary Surgery</td>
<td>Johannes Bonatti, MD</td>
<td>University of Maryland</td>
</tr>
<tr>
<td>4:00 p.m. – 4:10 p.m.</td>
<td>Robotic Cryo-MAZE for Atrial Fibrillation</td>
<td>W. Randolph Chitwood, Jr., MD</td>
<td>East Carolina University</td>
</tr>
<tr>
<td>4:10 p.m. – 4:30 p.m.</td>
<td>DISCUSSION</td>
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<tr>
<td>4:30 p.m.</td>
<td>ADJOURN</td>
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</table>
SUNDAY, MAY 2, 2010

8:00 a.m. – 5:00 p.m.  AATS/STS Adult Cardiac Surgery Symposium
Hall C, Metro Toronto Convention Centre
Chair: Marc R. Moon, MD
Washington University School of Medicine

COURSE OBJECTIVES

At the conclusion of this course, the participants will be able to:

- Determine which patients with bicuspid aortic valve disease will require simultaneous ascending aortic replacement, how much of the aortic will need to be resected and the specific surgical techniques necessary to safely perform the procedure.
- Differentiate single leaflet versus bileaflet prolapsed and learn surgical skills necessary to repair both varieties successfully.
- Assess patient subgroups who would be served best by coronary bypass grafting versus a percutaneous intervention.
- Identify patients a priori in whom it may not be cost conscious or appropriate from a quality of life standpoint to implant a left ventricular assist device or repair a thoracoabdominal aneurysm.

SESSION I:
BICUSPID AORTIC VALVE DISEASE

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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</thead>
</table>
| 8:00 a.m. – 8:15 a.m. | Pathobiology and Genetics of Bicuspid Aortopathy  
Joseph D. Schmoker, MD  
University of Vermont |
| 8:15 a.m. – 8:30 a.m. | When to Replace the Ascending Aorta in Bicuspid Disease  
Michael A. Borger, MD  
University of Leipzig |
AMERICAN ASSOCIATION FOR THORACIC SURGERY

SESSION II:
BARLOW’S SYNDROME AND ATRIAL FIBRILLATION

8:30 a.m. – 8:45 a.m.
Aortic Replacement in Bicuspid Disease:
How to Match the Procedure to the Patient
John S. Ikonomidis, MD, PhD
Medical University of South Carolina

8:45 a.m. – 9:05 a.m.
How I Do It: Bicuspid Valve-Sparing Root Replacement “Skin-to-Skin”
D. Craig Miller, MD
Stanford University

9:05 a.m. – 9:20 a.m.
DISCUSSION

9:20 a.m. – 9:40 a.m.
Leonardo Da Vinci and the Mitral Valve
Francis C. Wells, FRCS
Papworth Hospital

9:40 a.m. – 9:55 a.m.
Barlow’s Syndrome: Simple Bileaflet Prolapse or More?
Clifford W. Barlow, MD
Southampton General Hospital

9:55 a.m. – 10:15 a.m.
BREAK

10:15 a.m. – 10:35 a.m.
How I Do It: Repair a Barlow’s Valve “Skin-to-Skin”
Tirone E. David, MD
Toronto General Hospital

10:35 a.m. – 10:50 a.m.
Mitral Regurgitation with Paroxysmal Atrial Fibrillation: Is Concomitant Surgical Ablation Always Required?
Ralph J. Damiano, Jr., MD
Washington University School of Medicine
SESSION III: MALPRACTICE IN CARDIAC SURGERY – DEBATE

10:50 a.m. – 11:05 a.m. Surgical Ablation: Which Lesion Set for Which Patient?
Patrick M. McCarthy, MD
Northwestern University

11:05 a.m. – 11:20 a.m. DISCUSSION

11:20 a.m. – 11:50 a.m.
Malpractice in Cardiac Surgery Is the End-Result of: “Bad” Doctors
G. Randall Green, MD, JD
St. Joseph’s Hospital

Malpractice in Cardiac Surgery Is the End-Result of: “Bad” Lawyers
Abe DeAnda, Jr., MD
Montefiore/Albert Einstein College of Medicine

11:50 a.m. – 1:15 p.m.
LUNCH

LUNCHEON SYMPOSIUM A
Hall C
How I Do It: Robotic Mitral Valve Repair “Skin-to-Skin”
W. Randolph Chitwood, Jr., MD
East Carolina University

LUNCHEON SYMPOSIUM B
Constitution 107
CABG in 2010: Lessons from the Syntax Trial
Michael J. Mack, MD
CT Surgery Associates of North Texas
David Paul Taggart, MD, PhD
University of Oxford
### SESSION IV: TRANSCATHETER VALVE REPAIR AND REPLACEMENT

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
</tr>
</thead>
</table>
| 1:15 p.m. – 1:30 p.m. | Clinical Update: When Will Transcatheter Aortic Valve Replacement be Available in Anytown, USA?  
Alan Zajarias, MD  
Washington University School of Medicine |
| 1:30 p.m. – 1:45 p.m. | How I Do It: Transfemoral/Transapical AVR  
Todd M. Dewey, MD  
Medical City Dallas Hospital |
| 1:45 p.m. – 2:05 p.m. | How I Do It: Minimally-Invasive AVR “Skin-to-Skin”  
Lawrence H. Cohn, MD  
Brigham & Women’s Hospital |
| 2:05 p.m. – 2:20 p.m. | Percutaneous Mitral Valve Repair: Reality, Dream or Nightmare?  
Michael J. Mack, MD  
CT Surgery Associates of North Texas |
| 2:20 p.m. – 2:35 p.m. | DISCUSSION |
| 2:35 p.m. – 2:45 p.m. | With Regard to Afflictions of the Heart, Are Men and Women Created Equal?  
Jennifer S. Lawton, MD  
Washington University School of Medicine |
**SESSION V:**

**“WHEN NOT TO” IN CARDIAC SURGERY**

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>2:45 p.m. – 3:00 p.m.</td>
<td><strong>When Not To: Use the Radial Artery</strong></td>
<td>Hendrick B. Barner, MD</td>
<td>Saint Louis University</td>
</tr>
<tr>
<td>3:00 p.m. – 3:15 p.m.</td>
<td><strong>When Not To: Use Bilateral (or Unilateral) Mammary Arteries</strong></td>
<td>Bruce W. Lytle, MD</td>
<td>Cleveland Clinic Foundation</td>
</tr>
<tr>
<td>3:15 p.m. – 3:35 p.m.</td>
<td><strong>BREAK</strong></td>
<td></td>
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<tr>
<td>3:35 p.m. – 3:50 p.m.</td>
<td><strong>When Not To: Do a Ross Procedure</strong></td>
<td>Nicholas T. Kouchoukos, MD</td>
<td>Missouri Baptist Medical Center</td>
</tr>
<tr>
<td>3:50 p.m. – 4:05 p.m.</td>
<td><strong>When Not To: Implant an LVAD at “End-of-Life”</strong></td>
<td>Nicholas G. Smedira, MD</td>
<td>Cleveland Clinic Foundation</td>
</tr>
<tr>
<td>4:05 p.m. – 4:20 p.m.</td>
<td><strong>When Not To: Replace a “Large” Thoracoabdominal Aneurysm</strong></td>
<td>Thoralf M. Sundt, III, MD</td>
<td>Mayo Clinic</td>
</tr>
<tr>
<td>4:20 p.m. – 4:35 p.m.</td>
<td><strong>When Not To: Do an Off-Pump CABG</strong></td>
<td>Craig R. Smith, MD</td>
<td>Columbia University</td>
</tr>
<tr>
<td>4:35 p.m. – 4:50 p.m.</td>
<td><strong>When Not To: Repair Ischemic Mitral Regurgitation</strong></td>
<td>Irving L. Kron, MD</td>
<td>University of Virginia Health System</td>
</tr>
<tr>
<td>4:50 p.m. – 5:00 p.m.</td>
<td><strong>DISCUSSION</strong></td>
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<tr>
<td>5:00 p.m.</td>
<td><strong>ADJOURN TO WELCOME RECEPTION</strong></td>
<td>Exhibit Hall A &amp; B</td>
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</table>
SUNDAY, MAY 2, 2010

8:00 a.m. – 5:00 p.m. AATS/STS General Thoracic Surgery Symposium
Constitution 106, Metro Toronto Convention Centre
Chair: Bryan F. Meyers, MD, MPH
Washington University School of Medicine

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Outline the ideal preoperative evaluation for a patient referred for surgery for GERD.
- Develop an ideal surgical plan for a giant paraesophageal hernia.
- Describe the pros and cons of the surgical alternatives for the redo hiatal hernia.
- Discuss the risks, benefits and costs of crural buttressing for paraesophageal hernia repair.
- Relate the key elements needed to start a pulmonary thromboendarterectomy program.

SESSION I:

BENIGN ESOPHAGEAL DISEASE

8:00 a.m. – 8:10 a.m. Introduction and Course Overview
Bryan F. Meyers, MD, MPH
Washington University School of Medicine

8:10 a.m. – 8:35 a.m. Modern Evaluation of the GERD Patient for Surgery
Thomas J. Watson, MD
University of Rochester
SESSION II:
LUNG FAILURE AND TRANSPLANTATION

8:35 a.m. – 9:00 a.m.  
Primary Surgical Approach for Giant PEH  
Gail E. Darling, MD  
University of Toronto

9:00 a.m. – 9:25 a.m.  
Multiple Redo Hernia Surgery: Open or Resection?  
Andrew C. Chang, MD  
University of Michigan Health System

9:25 a.m. – 9:50 a.m.  
Crural Buttressing: When, Why and with What Material?  
Michael A. Maddaus, MD  
University of Minnesota Hospital

9:50 a.m. – 10:05 a.m.  
DISCUSSION

10:05 a.m. – 10:35 a.m.  
BREAK

10:35 a.m. – 11:00 a.m.  
Building a Pulmonary Thromboendarterectomy Program  
Marc de Perrot, MD, MSc  
Toronto General Hospital and Princess Margaret Hospital

11:00 a.m. – 11:25 a.m.  
Bilateral versus Single Lung Transplantation  
David Park Mason, MD  
Cleveland Clinic Foundation

11:25 a.m. – 11:50 a.m.  
Experience with Living Donor Lung Transplantation  
Hiroshi Date, MD  
Kyoto University

11:50 a.m. – 12:00 p.m.  
DISCUSSION
# Session III: Esophageal Cancer

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker(s)</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:00 p.m. – 1:30 p.m.</td>
<td>Luncheon Symposium&lt;br&gt;Tracheal Reconstruction with Tissue Engineered Airway</td>
<td>Paolo Macchiarini, MD, PhD&lt;br&gt;Hospital Clinico de Barcelona</td>
<td></td>
</tr>
<tr>
<td>1:30 p.m. – 1:50 p.m.</td>
<td>Optimal Staging of the Patient with Esophageal Cancer</td>
<td>Stephen D. Cassivi, MD, MSc&lt;br&gt;Mayo Clinic</td>
<td></td>
</tr>
<tr>
<td>1:50 p.m. – 2:10 p.m.</td>
<td>Selection of Patients for and Outcomes of Induction Therapy</td>
<td>Richard J. Battafarano, MD, PhD&lt;br&gt;University of Maryland</td>
<td></td>
</tr>
<tr>
<td>2:10 p.m. – 2:30 p.m.</td>
<td>Fast-tracking the Esophagectomy Patient</td>
<td>Henning A. Gaissert, MD&lt;br&gt;Massachusetts General Hospital</td>
<td></td>
</tr>
<tr>
<td>2:30 p.m. – 2:50 p.m.</td>
<td>Approach and Anastomotic Techniques for Esophagectomy</td>
<td>Ara A. Vaporciyan, MD&lt;br&gt;MD Anderson Cancer Center</td>
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<tr>
<td>2:50 p.m. – 3:00 p.m.</td>
<td>Discussion</td>
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<tr>
<td>3:20 p.m. – 3:50 p.m.</td>
<td>Break</td>
<td></td>
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</tbody>
</table>
### SESSION IV: MEDIASTINAL DISEASES

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Speaker</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>3:50 p.m.</td>
<td>Transcervical Extended Mediastinal Lymphadenectomy</td>
<td>Marcin Zielinski, MD</td>
<td>Pulmonary Hospital</td>
</tr>
<tr>
<td>4:10 p.m.</td>
<td>Eliminating Stump Leaks after Right Pneumonectomy</td>
<td>Kenneth A. Kesler, MD</td>
<td>Indiana University</td>
</tr>
<tr>
<td>4:30 p.m.</td>
<td>DISCUSSION</td>
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<tr>
<td>5:00 p.m.</td>
<td>ADJOURN TO WELCOME RECEPTION</td>
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<td>Exhibit Hall A &amp; B</td>
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</tbody>
</table>
SUNDAY, MAY 2, 2010

7:55 a.m. – 5:00 p.m. AATS/STS Congenital Heart Disease Symposium
Constitution 105, Metro Toronto Convention Centre
Chair: Charles B. Huddleston, MD
St. Louis Children's Hospital

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Implement new monitoring techniques for critical care.
- Apply indications and new therapies for treating pulmonary valve insufficiency.
- Discuss the advantages/disadvantages of different techniques for the Fontan procedure.
- Demonstrate best technique for repair of AV valve regurgitation in single ventricle patients, repair of supravalvar AS, repair of AP window and transplantation following failed Fontan.

7:55 a.m. – 8:00 a.m. Introduction
Charles B. Huddleston, MD
St. Louis Children's Hospital

8:00 a.m. – 8:40 a.m. EXTRA-CARDIAC VERSUS INTRA-ATRIAL LATERAL
Tunnel Fontan Extra-Cardiac Is Better
Carl L. Backer, MD
Children's Memorial Hospital
No, it's Not
Richard A. Jonas, MD
Children's National Medical Center

8:40 a.m. – 8:45 a.m. DISCUSSION
<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
<th>Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:45 a.m. – 9:25 a.m.</td>
<td>Adults with CHD PVR Following Repair of TOF – Now When?</td>
<td>Candice K. Silversides, MD</td>
<td>Toronto General Hospital</td>
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<td>Catheter-Based PVR – Where Does this Stand?</td>
<td>Lee N. Benson, MD</td>
<td>The Hospital for Sick Children</td>
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<tr>
<td>9:25 a.m. – 9:35 a.m.</td>
<td>DISCUSSION</td>
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<tr>
<td>9:35 a.m. – 10:15 a.m.</td>
<td>Impact of Fetal Intervention on Neonatal Congenital Heart Surgery</td>
<td>Emile A. Bacha, MD</td>
<td>New York Children’s Hospital</td>
</tr>
<tr>
<td>10:15 a.m. – 10:45 a.m.</td>
<td>BREAK</td>
<td></td>
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<tr>
<td>10:45 a.m. – 11:00 a.m.</td>
<td>Mechanical Cardiac Support for Infants</td>
<td>Charles D. Fraser, MD</td>
<td>Texas Children’s Hospital</td>
</tr>
<tr>
<td>11:00 a.m. – 11:15 a.m.</td>
<td>Novalung Experience</td>
<td>Shaf Keshavjee, MD, MSc</td>
<td>Toronto General Hospital</td>
</tr>
<tr>
<td>11:15 a.m. – 11:30 a.m.</td>
<td>Venous “Assist” for Fontan/Glenn</td>
<td>Mark D. Rodefeld, MD</td>
<td>Indiana University School of Medicine</td>
</tr>
<tr>
<td>11:30 a.m. – 12:00 p.m.</td>
<td>DISCUSSION</td>
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<td>Time</td>
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<td>Speaker</td>
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<tr>
<td>12:00 p.m. – 1:30 p.m.</td>
<td>LUNCHEON SYMPOSIUM</td>
<td><strong>Objective Assessment of Cardiac Output in Infants Following Cardiac Surgery</strong>&lt;br&gt;Desmond Bohn, MD  The Hospital for Sick Children</td>
<td></td>
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<tr>
<td>1:30 p.m. – 1:45 p.m.</td>
<td>AVVR in Single Ventricle</td>
<td>Glen S. Van Arsdell, MD</td>
<td>The Hospital for Sick Children</td>
</tr>
<tr>
<td>1:45 p.m. – 2:00 p.m.</td>
<td>Supravalvar AS in Infants</td>
<td>Max B. Mitchell, MD</td>
<td>The Children's Hospital Heart Institute</td>
</tr>
<tr>
<td>2:00 p.m. – 2:15 p.m.</td>
<td>AP Window and Associated Anomalies</td>
<td>James S. Tweddell, MD</td>
<td>Medical College of Wisconsin</td>
</tr>
<tr>
<td>2:15 p.m. – 2:30 p.m.</td>
<td>Heart Transplantation for Failed Fontan</td>
<td>Ralph S. Mosca, MD</td>
<td>NYU Langone Medical Center</td>
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<tr>
<td>2:30 p.m. – 2:40 p.m.</td>
<td>DISCUSSION</td>
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<td>2:40 p.m. – 3:00 p.m.</td>
<td>BREAK</td>
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<tr>
<td>3:00 p.m. – 3:20 p.m.</td>
<td>Worst Case Scenarios</td>
<td>Constantine Mavroudis, MD</td>
<td>Cleveland Clinic Foundation</td>
</tr>
<tr>
<td>3:20 p.m. – 3:40 p.m.</td>
<td>Worst Case Scenarios</td>
<td>Kirk R. Kanter, MD</td>
<td>Emory University School of Medicine</td>
</tr>
<tr>
<td>3:40 p.m. – 4:00 p.m.</td>
<td>DISCUSSION</td>
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</table>
4:00 p.m. – 4:25 p.m.
**Norwood Procedure – How it Started**
William I. Norwood, Jr., MD, PhD
Nemours Cardiac Center

4:25 p.m. – 4:50 p.m.
**TX for HLHS – How it Started**
Leonard L. Bailey, MD
Loma Linda University Medical Center

5:00 p.m.
**ADJOURN TO WELCOME RECEPTION**
Exhibit Hall A & B
SUNDAY, MAY 2, 2010

8:00 a.m. – 5:00 p.m.  AATS/STS Cardiothoracic Critical Care Symposium
Room 201, Metro Toronto Convention Centre
Chairs: Nevin M. Katz, MD
Washington Institute of Thoracic & CV Surgery
Michael S. Mulligan, MD
University of Washington Medical Center

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Understand unique specialty aspects of Cardiovascular-Thoracic (CVT) Critical Care including ECMO and mechanical circulatory support.
- Integrate nutritional innovations into the practice of CVT Critical Care.
- Apply evidence-based blood and component transfusion guidelines to clinical practice.
- Select appropriate anti-microbial agents and practices for treatment and prevention of nosocomial infection including CVL infections.

8:00 a.m. – 8:05 a.m.  Welcome and Opening Remarks
Michael S. Mulligan, MD
University of Washington Medical Center

8:05 a.m. – 8:30 a.m.  Evolution of CTS Critical Care: Systems Based Management, Training and Certification Implications
Nevin M. Katz, MD
Washington Institute of Thoracic & CV Surgery
<table>
<thead>
<tr>
<th>Time</th>
<th>Session Title</th>
<th>Speaker</th>
<th>Affiliation</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:30 a.m. – 8:55 a.m.</td>
<td>Sedation in the ICU Patient</td>
<td>Brian J. Woodcock, MD</td>
<td>University of Michigan Health System</td>
</tr>
<tr>
<td>8:55 a.m. – 9:20 a.m.</td>
<td>ECMO Support for the Failing Heart/Lung</td>
<td>Jonathan W. Haft, MD</td>
<td>University of Michigan Health System</td>
</tr>
<tr>
<td>9:20 a.m. – 9:45 a.m.</td>
<td>Arrhythmia Management</td>
<td>Ralph Damiano, Jr., MD</td>
<td>Washington University School of Medicine</td>
</tr>
<tr>
<td>9:45 a.m. – 10:00 a.m.</td>
<td>BREAK</td>
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<tr>
<td>10:00 a.m. – 10:25 a.m.</td>
<td>Advanced Ventilator Management (Low Stretch Ventilation and Permissive Hypercapnea)</td>
<td>Leonard D. Hudson, MD</td>
<td>University of Washington Medical Center</td>
</tr>
<tr>
<td>10:25 a.m. – 10:50 a.m.</td>
<td>LVAD and Mechanical Support Post-operative Care</td>
<td>Francis D. Pagani, MD</td>
<td>University of Michigan Health System</td>
</tr>
<tr>
<td>10:50 a.m. – 11:15 a.m.</td>
<td>Lung Transplant Perioperative Care</td>
<td>Michael S. Mulligan, MD</td>
<td>University of Washington Medical Center</td>
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<td>11:15 a.m. – 11:50 a.m.</td>
<td>Optimizing Hemodynamic Support: Are We Aiming for the Right Targets? Post-operative Monitoring/Related Complications</td>
<td>Nevin M. Katz, MD</td>
<td>Washington Institute of Thoracic &amp; CV Surgery</td>
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<td>Time</td>
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<td>Speaker</td>
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<td>12:00 p.m. – 1:30 p.m.</td>
<td>LUNCHEON SYMPOSIUM&lt;br&gt;Hemodynamic Simulation interactive Session</td>
<td>Nevin M. Katz, MD</td>
<td>Washington Institute of Thoracic &amp; CV Surgery</td>
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<td>1:30 p.m. – 1:55 p.m.</td>
<td>Infection Prophylaxis and Prevention</td>
<td>Marin H. Kollef, MD</td>
<td>Washington University School of Medicine</td>
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<td>1:55 p.m. – 2:20 p.m.</td>
<td>Clinical Use of the Artificial Lung</td>
<td>Joseph B. Zwischenberger, MD</td>
<td>University of Kentucky</td>
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<td>2:20 p.m. – 2:45 p.m.</td>
<td>Support for Acute Renal Dysfunction/Failure</td>
<td>Andrew D. Shaw, MD</td>
<td>Duke University</td>
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<td>2:45 p.m. – 3:10 p.m.</td>
<td>Special Considerations for Aortic Surgery, Hybrid Procedures and TEVARs</td>
<td>Joseph E. Bavaria, MD</td>
<td>University of Pennsylvania</td>
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<td>3:10 p.m. – 3:30 p.m.</td>
<td>BREAK</td>
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<td>3:30 p.m. – 3:55 p.m.</td>
<td>Bleeding and Management of Coagulopathy</td>
<td>Jerrold Levy, MD</td>
<td>Emory University</td>
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<td>3:55 p.m. – 4:20 p.m.</td>
<td>Nutritional Support and Its Impact</td>
<td>Aaron M. Cheng, MD</td>
<td>Harborview Medical Center</td>
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| 4:20 p.m. – 4:45 p.m. | Assessment and Management of Neurologic Complications after Cardiac and Aortic Surgery  
Joseph S. Coselli, MD  
Texas Heart Institute/Baylor College of Medicine  
**DISCUSSION**  
**ADJOURN TO WELCOME RECEPTION**  
*Exhibit Hall A & B* |
| 5:00 p.m.         | ADJOURN TO WELCOME RECEPTION |

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*90th Annual Meeting May 1–May 5, 2010*  
*Toronto, ON, Canada*
SUNDAY AFTERNOON
MAY 2, 2010

3:00 p.m. – 5:00 p.m.  13th Annual C. Walton Lillehei Resident Forum
206C, Metro Toronto Convention Centre
(7 minutes presentation, 8 minutes discussion)

Chairs:  
John S. Ikonomidis, MD  
Medical University of South Carolina  
Glen S. Van Arsdell, MD  
The Hospital for Sick Children

L1.  
BRCA1 Is a Novel Regulator of Cardiac Function via Altering Myocardial Substrate Utilization and Mitochondrial Bioenergetics  
Bobby Yanagawa, Praphulla Shukla, Krishna K. Singh, Hwee Teoh, Subodh Verma  
Division of Cardiac Surgery, St. Michael's Hospital, University of Toronto, Toronto, ON, Canada.

L2.  
AP2β Nucleolar Localization Predicts Poor Survival after Lung Cancer Resection in Stage I Patients  
Min P. Kim1, Ying Chen2, Adriana Lopez2, Ignacio Wistuba2, Lin Ji1, Jack A. Roth1*, Ruth L. Katz2  
1. Thoracic and Cardiovascular Surgery, MD Anderson Cancer Center, Houston, TX, USA.  
2. Pathology, MD Anderson Cancer Center, Houston, TX, USA.  
3. Biostatistics, MD Anderson Cancer Center, Houston, TX, USA.

L3.  
Size and Polymer-Dependent Intranodal Localization of Methacrylate Nanoparticles  
Onkar Khullar1, Kimberly Ann V. Zubris2, Aaron P. Griset2, John V. Frangioni3, Mark W. Grinstaff2, Yolonda L. Colson1*  
1. Division of Thoracic Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA.  
2. Departments of Biomedical Engineering and Chemistry, Boston University, Boston, MA, USA.  
3. Division of Hematology Oncology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA.

*AATS Member
L4. **Dabigatran Is Effective for Thromboprophylaxis of Mechanical Heart Valves**
Stephen H. McKellar¹, Stuart Abel¹, Christopher Camp¹, Mark Ereth², Hartzell V. Schaff¹∗

L5. **Safe, Efficient and Durable Transduction of Human and Canine Saphenous Vein Grafts Using a Modified Adeno Associated Viral Vector**
Mani A. Daneshmand¹, Nestor R. Villamizar¹, Roberto J. Manson¹, Thomas Mulhearn², Christopher D. Kontos², Carmelo A. Milano¹, Dawn Bowles¹
1. Surgery, Duke University Medical Center, Durham, NC, USA. 2. Medicine, Division of Cardiology, Duke University Medical Center, Durham, NC, USA.

L6. **Effects of Annuloplasty Ring Implantation on Regional Mitral Leaflet Tenting Area During Acute Myocardial Ischemia**
Wolfgang Bothe¹, Elizabeth H. Stephens¹, John-Peder E. Kvitting¹, Julia C. Swanson¹, Neil B. Ingels², D. Craig Miller¹∗
1. Cardiothoracic Surgery, Stanford School of Medicine, Stanford, CA, USA. 2. Laboratory of Cardiovascular Physiology and Biophysics, Research Institute of the Palo Alto Medical Foundation, Mountain View, CA, USA.

L7. **Pro-Inflammatory Role of A2B Adenosine Receptor in Lung Ischemia-Reperfusion Injury**
Farshad Anvari¹, Ashish K. Sharma¹, Lucas G. Fernandez¹, Katya Ravid², Irving L. Kron¹∗, Victor E. Laubach¹
1. Surgery, University of Virginia, Charlottesville, VA, USA 2. Biochemistry, Boston University School of Medicine, Boston, MA, USA.

L8. **Effects of Nonselective COX and Selective COX-2 Inhibition on Collateral Development in the Heart**
Michael P. Robich¹, Louis M. Chu¹, Jun Feng¹, Cesario Bianchi¹, Roger J. Laham², Michael A. Coady³, Frank W. Sellke³∗
1. Surgery, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA. 2. Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA. 3. Surgery, Warren Alpert Medical School, Brown University, Providence, RI, USA.

5:00 p.m. **ADJOURN TO WELCOME RECEPTION**
Exhibit Hall A & B
**MONDAY, MAY 3, 2010**

7:30 a.m.  
**Business Session**  
(AATS Members Only)  
*Hall C, Metro Toronto Convention Centre*

7:45 a.m.  
**PLENARY SCIENTIFIC SESSION**  
*Hall C, Metro Toronto Convention Centre*  
(8 minutes presentation, 12 minutes discussion)  
**Moderators:**  
G. Alec Patterson, MD  
Thoralf M. Sundt, III, MD

1. **Randomized Trial of Mediastinal Lymph Node Sampling versus Complete Lymphadenectomy During Pulmonary Resection in Patients with N0 or N1 (Less than Hilar) Non-Small Cell Carcinoma: Results of the ACOSOG Z0030 Trial**  
Gail E. Darling1*, Mark S. Allen2, Paul Decker3, Karla V. Ballman3,  
Rodney J. Landreneau4*, Robert J. McKenna5*, David R. Jones7*,  
Richard I. Inculet8, Valerie W. Rusch9*, Joe B. Putnam6*  
**Invited Discussant:**  
Joseph B. Shrager, MD

2. **One Slide Fits All: The Versatility of Slide Tracheoplasty Utilizing Cardiopulmonary Bypass Support for Airway Reconstruction in Children**  
Peter B. Manning1*, Michael J. Rutter2, Asher Lisec3, Bradley S. Marino3  
1. Cardiothoracic Surgery, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA. 2. Otorhinolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA. 3. Cardiology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA.  
**Invited Discussant:**  
Martin J. Elliott, MD

*AATS Member*
3. **Transapical Aortic Valve Implantation at Three Years**  
Thomas Walther, Joerg Kempfert, Michael A. Borger*, Ardawan J. Rastan,  
Axel Linke, Gerhard Schuler, Friedrich W. Mohr*  
*Cardiac Surgery, Heartcenter Leipzig, Leipzig, Germany.*  
*Invited Discussant:* Joseph E. Bavaria, MD

4. **Superior Nationwide Outcomes of Thoracic Endovascular Aneurysm Repair Compared to Open Repair for Isolated Descending Thoracic Aneurysm in a Cohort of 11,000 Patients**  
Raja R. Gopaldas1,2, Joseph Huh1,2, Tam K. Dao1, Scott A. LeMaire1,2*, Danny Chu1,4, Faisal G. Bakaeen1,4, Joseph Coselli1,2*  
1. Division of Cardiothoracic Surgery, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX, USA. 2. Texas Heart Institute at St. Luke’s Episcopal Hospital, Houston, TX, USA. 3. Department of Education Psychology, University of Houston, Houston, TX, USA. 4. Michael E. DeBakey Veterans Affairs Medical Center, Houston, TX, USA.  
*Invited Discussant:* John A. Kern, MD

9:05 a.m. **AWARD PRESENTATIONS**

9:20 a.m. **INTERMISSION – VISIT EXHIBITS/COFFEE BREAK**  
**EXHIBIT HALL A & B**

10:00 a.m. **BASIC SCIENCE LECTURE**  
“Nerve Injury – Changing the Surgical Paradigm through Translational Science”  
Susan E. Mackinnon, MD  
Washington University School of Medicine  
*Introduced By:* G. Alec Patterson, MD

10:40 a.m. **PLENARY SCIENTIFIC SESSION**  
*Moderators:* Irving L. Kron, MD  
Thoralf M. Sundt, III, MD

5. **Identifying Patients at Particular Risk of Injury at Repeat Sternotomy: Analysis of Over 2500 Cardiac Reoperations**  
Chan B. Park, Rakesh M. Suri, Harold M. Burkhart, Kevin L. Greason,  
Joseph A. Dearani, Hartzell V. Schaff*, Thoralf M. Sundt*  
*Mayo Clinic, Rochester, MN, USA.*  
*Invited Discussant:* Irving L. Kron, MD

*AATS Member*
6. The Effect of Regionalization on Outcome in Pulmonary Lobectomy: A Canadian National Study
Christian J. Finley¹, Shaf Keshavjee¹*, David R. Urbach², Anna Bendzsak¹, George Tomlinson², Gail E. Darling¹*
Division of Thoracic Surgery, Toronto General Hospital, Toronto, ON, Canada.
2. Division of Clinical Decision Making and Health Care, Toronto General Hospital, Toronto, ON, Canada.
Invited Discussant: Yolonda L. Colson, MD, PhD

11:25 a.m. PRESIDENTIAL ADDRESS
“Non Solus – A Leadership Challenge”
G. Alec Patterson, MD
Washington University School of Medicine
Introduced By: Irving L. Kron, MD

12:15 p.m. ADJOURN FOR LUNCH – VISIT EXHIBITS
Exhibit Hall A & B

SIMULTANEOUS SCIENTIFIC SESSIONS

2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – ADULT CARDIAC SURGERY
Hall C, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)
Moderators: R. Morton Bolman, III, MD
R. Duane Davis, MD

7. Hemodynamic Results at One Month and Over One Year of Endoventricular Patch Plasty in 117 Ischemic Failing Ventricles Excluded from the STICH Trial
Vincent Dor*, Filippo Civaia, Clara Alexandrescu, Michel Sabatier, Françoise Montiglio
Centre Cardio-Thoracique, Monaco Cedex, Monaco.
Invited Discussant: Robert H. Jones, MD

*AATS Member
8. Endoscopic Vein Harvest is Associated with Compromised Patency Outcomes: Results from the Prospective Randomized ROOBY Trial

Marco A. Zenati1*, Ali F. Sonel2, A. Laurie Shroyer3, Morteza Amidi2, Joseph Collins4, G. Hossein Almassi5*, ROOBY Study3,6

1. Cardiac Surgery, University of Pittsburgh, Pittsburgh, PA, USA. 2. Cardiology, VA Pittsburgh, Pittsburgh, PA, USA. 3. VA Northport, Northport, NY, USA. 4. CSP Data Coordinating Center, Perry Point, MD, USA. 5. Medical College of Wisconsin, Milwaukee, WI, USA. 6. ECHS Denver VAMC, Denver, CO, USA.

Invited Discussant: Carlos A. Mestres, MD, PhD

9. Impact of Pulmonary Hypertension on Outcomes following Aortic Valve Replacement for Aortic Valve Stenosis

Spencer J. Melby, Marci Bailey, Marc R. Moon*, Nader Moazami, Jennifer S. Lawton*, Brian R. Lindman, Ralph J. Damiano*

Cardiothoracic Surgery, Washington University School of Medicine, St. Louis, MO, USA.

Invited Discussant: James S. Gammie, MD

3:00 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK

Exhibit Hall A & B

3:35 p.m. SIMULTANEOUS SCIENTIFIC SESSION – ADULT CARDIAC SURGERY

Hall C, Metro Toronto Convention Centre

Moderators: R. Morton Bolman, III, MD
R. Duane Davis, MD

10. A Randomized Trial of a Restrictive versus Liberal Blood Transfusion Strategy in Older Postoperative Cardiac Surgery Patients

Robin Varghese1, M. Lee Myers1, L. Ray Guo1, Neville Suskin2

2. Cardiology London Health Sciences Centre, London, ON, Canada.

Invited Discussant: Jennifer S. Lawton, MD

11. Robotic Mitral Valve Repair versus Conventional Approaches: Potential Realized

Tomislav Mihaljevic, Craig Jarrett, A. Marc Gillinov*, Sarah Williams, Pierre De Villiers, Eugene H. Blackstone*

Cleveland Clinic Foundation, Cleveland, OH, USA.

Invited Discussant: Volkmar Falk, MD

*AATS Member
12. Minimally Invasive Aortic Valve Replacement in High-Risk Percutaneous Transapical Aortic Valve Replacement Candidates
Andrew W. El Bardissi, Prem Shekar, Gregory S. Couper*, Ann Maloney, Lawrence H. Cohn*
Department of Surgery, Brigham and Women’s Hospital, Boston, MA, USA.
Invited Discussant: Craig R. Smith, MD

13. The Cox-Maze IV Procedure: Predictors of Late Recurrence
Ralph J. Damiano*, Marci Bailey, Forrest H. Schwartz, Nabil A. Munfakh, Jennifer S. Lawton*, Richard B. Schuessler, Marc R. Moon*
Cardiothoracic Surgery, Washington University School of Medicine, St. Louis, MO, USA.
Invited Discussant: Michael Argenziano, MD

5:00 p.m. ADJOURN

2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – GENERAL THORACIC SURGERY
Constitution 106, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)
Moderators: Robert J. Cerfolio, MD
Yolonda L. Colson, MD, PhD

14. Long Term Results of the Heller-Dor Operation with Intra Operative Manometry for the Cure of Esophageal Achalasia
Sandro Mattioli1, Alberto Ruffato1, Vladimiro Pilotti1, Beatrice Aramini1, Frank D’Ovidio2
1. Division of Esophageal and Pulmonary Surgery, Villa Maria Cecilia Hospital, University of Bologna, Bologna, BO, Italy. 2. Division of Cardiothoracic Surgery, Columbia University, New York, NY, USA.
Invited Discussant: Bryan F. Meyers, MD, MPH

*AATS Member
15. The Impact of Adjuvant Brachytherapy with Sublobar Resection on Pulmonary Function and Dyspnea; Preliminary Results from ACOSOG Z4032 Trial
Hiran C. Fernando1*, Rodney J. Landreneau2*, Sumithra Mandrekar3, Shauna Hillman4, Francis C. Nichols4, Bryan Meyers5*, Thomas Dipetrillo6*, Dwight E. Heron7, Joe B. Putnam8*

Invited Discussant: Walter Weder, MD

16. Lobectomy Leads to Optimal Survival in Early-Stage Small Cell Lung Cancer
Malcolm M. DeCamp1*, Abram Recht2, John C. Flickinger3, Laura N. Medford-Davis4, Anne-Marie Dyer5, John M. Varlotto6
1. Division of Cardiothoracic Surgery, Beth Israel Deaconess Medical Center, Boston, MA, USA. 2. Department of Radiation Oncology, Beth Israel Deaconess Medical Center, Boston, MA, USA. 3. Department of Radiation Oncology, Pittsburgh Cancer Institute, Pittsburgh, PA, USA. 4. Harvard Medical School, Boston, MA, USA. 5. Department of Public Health Sciences, Pennsylvania State University, Hershey, PA, USA. 6. Division of Radiation Oncology, Pennsylvania State University, Hershey, PA, USA.

Invited Discussant: Jessica S. Donington, MD

17. Bilateral Lung Transplantation Does Not Improve Survival in Patients 65 and Above
Shekar L. Reddy, Richa Sharma, Shu S. Lin, Scott M. Palmer, David W. Zaas, R. Duane Davis*
Cardiothoracic Surgery, Transplant Division, Duke University Medical Center, Durham, NC, USA.

Invited Discussant: Thomas K. Waddell, MD, MSc, PhD

3:20 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B
3:55 p.m.  SIMULTANEOUS SCIENTIFIC SESSION –
GENERAL THORACIC SURGERY
Constitution 106, Metro Toronto Convention Centre
Moderators:  Robert J. Cerfolio, MD
Yolonda L. Colson, MD, PhD

18. Patterns of Recurrence and Incidence of Second Primary Tumors
after Lobectomy by VATS versus Thoracotomy for Lung Cancer
Raja M. Flores*, Ugonna N. Ihekweazu, Nabil Rizk, Manjit S. Bains*,
Robert J. Downey*, Prasad Adusumilli, David J. Finley, James Huang,
Joseph Dycoco, Valerie W. Rusch*, Bernard Park
Thoracic Surgery, Memorial Sloan-Kettering Cancer Center, Thoracic Surgery,
New York, NY, USA.
Invited Discussant:  Scott J. Swanson, MD

19. Respiratory Function after Pneumonectomy: Results of the
Pneumonectomy Project
Jean Deslauriers*, Steve Provencher, Paula Ugalde, Santiago Miro,
Yves Lacasse, Sébastien Bergeron, Sylvie Ferland
Centre de Pneumologie, IUCPQ (Hôpital Laval), Quebec, QC, Canada.
Invited Discussant:  David J. Sugarbaker, MD

20. Age, Mediastinal Downstaging and Extent of Pulmonary Resection
are Independent Predictors of Survival in Clinical Stage IIIA Patients
after Induction Therapy
Subroto Paul, Jeffrey L. Port*, Brendon M. Stiles, Paul C. Lee*, James
Saunders, Farooq Mirza, Nasser K. Altorki*
Department of Cardiothoracic Surgery, Weill Cornell Medical College, New
York, NY, USA.
Invited Discussant:  Gail E. Darling, MD

5:00 p.m.  ADJOURN
2:00 p.m.  SIMULTANEOUS SCIENTIFIC SESSION – CONGENITAL HEART DISEASE
Constitution 105, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)

**Moderators:** Emile A. Bacha, MD
James S. Tweddell, MD

21. **Application of the Hemi-Mustard Bidirectional Glenn Atrial Switch in the Double Switch Procedure for Congenitally Corrected Transposition of the Great Arteries: Rationale and Midterm Results**
Sunil P. Malhotra1, V. Mohan Reddy2*, Mary Qiu2, Timothy J. Pirolli2, Laura Barboza2, Olaf Reinhartz3, Frank L. Hanley2*
1. Congenital Heart Center, University of Florida, Gainesville, FL, USA.
2. Cardiothoracic Surgery, Stanford University, Stanford, CA, USA.

*Invited Discussant:* David J. Barron, MD

22. **Aortic Valve Repair By Cusp Extension for Rheumatic Aortic Insufficiency in Children: Long-Term Results and Impact of Extension Material**
Patrick O. Myers1, Cecile Tissot3, Jan T. Christenson2, Maurice Beghetti3, Mustafa Cikirkioglu2, Afksendiyos Kalangos2*
1. Cardiac Surgery, Brigham & Women’s Hospital, Boston, MA, USA.
2. Cardiovascular Surgery, Geneva University Hospitals & School of Medicine, Geneva, Switzerland. 3. Unit of Pediatric Cardiology, Geneva University Hospitals & School of Medicine, Geneva, Switzerland.

*Invited Discussant:* Emile A. Bacha, MD

23. **Atrioventricular Valve Repair in Patients with Single Ventricle Physiology: Impact of Ventricular Function and Morphology, Valve Morphology and Mechanism of Insufficiency on Outcomes**
Osami Honjo, Cori Atlin, Luc Mertens, Osman O. Al-Radi, Andrew N. Redington, Christopher A. Caldareone*, Glen S. Van Arsdell*
The Labatt Family Heart Centre, The Hospital for Sick Children, Toronto, ON, Canada.

*Invited Discussant:* Jennifer C. Hirsch, MD

*AATS Member*
24. The Kinetics of the Development of Ventricular Dysfunction in Patients with Hypoplastic Left Heart Syndrome
Heather Underkofler, Michele Frommelt, Raymond T. Fedderly, Pippa M. Simpson, Michael E. Mitchell, James S. Tweddell, Kimberly L. Gandy
1. Surgery, Medical College of Wisconsin, Milwaukee, WI, USA. 2. Pediatrics, Children’s Research Institute, Milwaukee, WI, USA. 3. Children’s Hospital of Wisconsin, Milwaukee, WI, USA.

Invited Discussant: Shunji Sano, MD, PhD

3:20 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B

3:55 p.m. SIMULTANEOUS SCIENTIFIC SESSION – CONGENITAL HEART DISEASE
Constitution 105, Metro Toronto Convention Centre

Moderators: Emile A. Bacha, MD
James S. Tweddell, MD

25. Subsequent Aortic Arch and Left Ventricular Outflow Tract Procedures in Patients after Interrupted Aortic Arch Repair: A Multi-Institutional Study
1. Surgery, Division of Cardiovascular Surgery, The Hospital for Sick Children, Toronto, ON, Canada. 2. Pediatric and Congenital Heart Surgery, Cleveland Clinic Foundation, Cleveland, OH, USA. 3. Cardiothoracic Surgery, Children’s Mercy Hospital, Kansas, MO, USA. 4. Surgery, Division of Thoracic and Cardiovascular Surgery, The Congenital Heart Institute of Florida (CHIF), Cardiac Surgical Associates of Florida (CSAoF), University of South Florida (USF), All Children’s Hospital, Children’s Hospital of Tampa, Saint Petersburg and Tampa, FL, USA. 5. Division of Cardiovascular Surgery, Center for Heart, Lung and Kidney Disease, Children’s National Medical Center, Washington, DC, USA. 6. Pediatrics, Division of Cardiology, The Hospital for Sick Children, Toronto, ON, Canada. 7. Thoracic and Cardiovascular Surgery, Cleveland Clinic Foundation, Cleveland, OH, USA. 8. Surgery, Division of Cardiothoracic Surgery, University of Miami Health Systems, Miller School of Medicine, Miami, FL, USA. 9. Cardiothoracic Surgery, The Congenital Heart Institute at Arnold Palmer Hospital for Children, Orlando, FL, USA.

Invited Discussant: Charles D. Fraser, MD

*AATS Member
26. Leaflet Suspension to the Contralateral Annulus to Address Mitral and Tricuspid Leaflet Restriction or Tethering  
Patrick O. Myers¹, Jan T. Christenson², Mustafa Cikirikcioglu², Yacine O. Aggoun³, Cecile Tissot³, Afksendiyos Kalangos²*  
¹Cardiac Surgery, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA. ²Cardiovascular Surgery, Geneva University Hospital & School of Medicine, Geneva, Switzerland. ³Pediatric Cardiology, Children's University Hospital & School of Medicine, Geneva, Switzerland.  
Invited Discussant: Luca A. Vricella, MD

27. Pediatric Heart Re-Transplantation: Patterns of Primary Graft Failure Differ Between Recipients Initially Transplanted in Infancy Compared to Older Children  
John M. Karamichalis, Shelley Miyamoto, David N. Campbell*, Jilayne Smith, Sydne Clark, Biagio A. Pietra, Max B. Mitchell  
Cardiac Surgery and Cardiology, The Children's Hospital, University of Colorado Denver Health Science Center, Denver, CO, USA.  
Invited Discussant: Jonathan M. Chen, MD
MONDAY, MAY 3, 2010

2:00 p.m.  Building the Hybrid OR of the Future©
Room 201, Metro Toronto Convention Centre
Program Directors: Raphael Bueno, MD
John Byrne, MD

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<th>Time</th>
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| 2:00 p.m. – 2:15 p.m. | Introduction
David Sugarbaker, MD
Brigham and Women's Hospital
John Byrne, MD
Vanderbilt Heart & Vascular Institute
Raphael Bueno, MD
Brigham and Women's Hospital |
| 2:15 p.m. – 2:45 p.m. | Making the Case for a Hybrid OR
Patricia Wynn
The Advisory Board Company
Joseph E. Bavaria, MD
Hospital of the University of Pennsylvania |
| 2:45 p.m. – 3:15 p.m. | Identifying the Players for a Hybrid OR
Marie Hasselblad, RN
Vanderbilt Heart & Vascular Institute
Vinod H. Thourani, MD
Emory University School of Medicine |
| 3:15 p.m. – 3:30 p.m. | DISCUSSION |
| 3:30 p.m. – 3:45 p.m. | BREAK |
| 3:45 p.m. – 4:15 p.m. | The Hybrid OR: Architectural Issues
Charles Martin
NBBJ
Jeff High, RN, BA, ASN
Vanderbilt Heart & Vascular Institute |
4:15 p.m. – 4:45 p.m.
Challenges Encountered Along the Way in Building a Hybrid OR
Ferenc A. Jolesz, MD
Brigham and Women's Hospital
Mathew R. Williams, MD
New York-Presbyterian/Columbia
Georg Nollert, MD
Siemens AG

4:45 p.m. – 5:15 p.m.
Philosophy of Equipment Selection
Julian M. Goldman, MD
CIMIT
Janice Crosby, RN, MBA
CIMIT

5:15 p.m. – 5:30 p.m.
DISCUSSION

5:30 p.m.
ADJOURN
TUESDAY, MAY 4, 2010

7:00 a.m.  Cardiac Surgery Forum Session
Hall C, Metro Toronto Convention Centre
(5 minutes presentation, 5 minutes discussion)

Moderators:  Pedro J. del Nido, MD
             Craig R. Smith, MD

F1.  Despite Successful CABG in a Swine Model of Myocardial
     Hibernation, Maximal Oxygen Consumption and Mitochondrial
     Proteomic Expression Remain Depressed
Rosemary F. Kelly1*, Elizabeth A. Ziemba1, Jesus A. Cabrera1,
Melanie Crampton2, Lorraine B. Anderson1, Edward O. McFalls2,
Herbert B. Ward1*
1. Cardiothoracic Surgery, University of Minnesota, Minneapolis, MN,
   USA. 2. Cardiology, VA Medical Center, Minneapolis, MN, USA.
Invited Discussant:  Keith A. Horvath, MD

F2.  Transforming Growth Factor-β Induces the Expression of
     Extracellular Matrix Molecules in Myxomatous Mitral Valves
Arnar Geirsson1, Hussain M. Abbas2, Rahmat Ali1, Richard W. Kim1,
Juan A. Sanchez2*, Sabet Hashim1, George Tellides1
1. Section of Cardiac Surgery, Yale University, New Haven, CT, USA.
2. Department of Surgery, St. Mary’s Hospital, Waterbury, CT, USA.
Invited Discussant:  Y. Joseph Woo, MD

F3.  Less Invasive and Highly Effective Method for Preventing
     Postoperative Atrial Fibrillation by the Intraoperative Application
     of a Novel Biodegradable Disc Containing Amiodarone
Takahide Takeda1, Takeshi Shimamoto2, Akira Marui1, Naritatu Saito3,
Kyokun Uehara1, Tadashi Ikeda1, Ryuizo Sakata1
1. Department of Cardiovascular Surgery, Graduate School of Medicine,
   Kyoto University, Kyoto, Kyoto, Japan. 2. Department of Cardiovascular
   Surgery, Kurashiki Central Hospital, Kurashiki, Okayama, Japan.
3. Department of Cardiology, Nagai Hospital, Tsu, Mie, Japan.
Invited Discussant:  Ralph J. Damiano, Jr., MD
F4. Cardiac Xenotransplantation Technology Provides Materials for Improved Bioprosthetic Heart Valves
Christopher G. McGregor1*, Nermine Lila2, Michal Vlasin1, John S. Logan1, Guerard W. Byrne1
Invited Discussant: R. Duane Davis, MD

F5. Cardiac Insulin Resistance as a Risk Factor for Heart Failure
Paulo Amorim, T. Dung Nguyen, Andrea Schrepper, Claudia Hain, Yasushige Shingu, Friedrich W. Mohr*, Torsten Doenst
Herzzentrum Leipzig, Leipzig, Germany
Invited Discussant: Todd K. Rosengart, MD

F6. Diazoxide Mediated Maintenance of Myocyte Volume Homeostasis During Stress Requires KATP Channel Subunit SUR1
Angela Sellitto, Haixia Zhang, Richard B. Schuessler, Colin Nichols, Jennifer S. Lawton*
Surgery, Washington University School of Medicine, Saint Louis, MO, USA.
Invited Discussant: Friedhelm Beyersdorf, MD

F7. Myocardial Ischemia-Reperfusion Transcriptional Program in Normal and High Fat Diet Pigs
Cesario Bianchi1, Louis M. Chu1, Robert Osipov1, Shizu Oyamada1, Jonathan Igne Bianchi2, Michael P. Robich3, Frank W. Sellke3*
1. Surgery, Beth Israel Deaconess Medical Center, Boston, MA, USA. 2. Interdisciplinary Medicine, Beth Israel Deaconess Medical Center, Boston, MA, USA. 3. Surgery Cardiothoracic, Warren Alpert School of Medicine/Brown University, Providence, RI, USA.
Invited Discussant: Pedro J. del Nido, MD

Shahab A. Akhter, Karen M. D'Souza, Michelle L. Staron, Jai Raman*, Valluvan Jeevanandam*
Surgery, University of Chicago Medical Center, Chicago, IL, USA.
Invited Discussant: Carmelo A. Milano, MD

*AATS Member
F9. Myocellular Maladaptation to Ischemic Reperfusion Injury Increases Propensity of Post-Operative Right Ventricular Dysfunction in Cyanotic Tetralogy of Fallot
Edward W. Peng1, David McCaig2, Stuart Lilley3, Kenneth J. MacArthur1, James Pollock1, Fiona Lyall2, Mark Danton1
1. Department of Cardiac Surgery, Royal Hospital for Sick Children, Glasgow, United Kingdom. 2. Maternal & Fetal Medicine, Institute of Medical Genetics Yorkhill Hospitals, Glasgow, United Kingdom. 3. Department of Cardiology, Royal Hospital of Sick Children, Glasgow, United Kingdom.
Invited Discussant: James Jaggers, MD

F10. Lanthanum Carbonate, a Phosphate Binder, Inhibits Calcification of Implanted Aortic Allografts
Osamu Kinoshita1, Noboru Motomura1, Haruo Yamauchi2, Minoru Ono1, Arata Murakami1, Shinichi Takamoto2
1. Cardiothoracic Surgery, The University of Tokyo, Tokyo, Japan. 2. Mitsui Memorial Hospital, Tokyo, Japan.
Invited Discussant: Richard A. Hopkins, MD

7:00 a.m. GENERAL THORACIC SURGERY FORUM SESSION
Constitution 106, Metro Toronto Convention Centre
(5 minutes presentation, 5 minutes discussion)
Moderators: David R. Jones, MD
Shaf Keshavjee, MD, MSc

F11. Vascular Endothelium Contributes to Tumor Tolerance Induction
Ruben G. Nava, Sanjay Murala, G. Alec Patterson*, Andrew Gelman, Daniel Kreisel, Sasha A. Krupnick
Surgery, Washington University School of Medicine in St. Louis, St. Louis, MO, USA.
Invited Discussant: David R. Jones, MD
F12. Gene Expression Profiles in Esophageal Adenocarcinoma Predict Survival Following Resection
Arjun Pennathur1, Liqiang Xi2, Virginia R. Little3, William E. Gooding1, Rodney J. Landreneau1*, Tony Godfrey2, James D. Luketich1*
1. The Heart, Lung and Esophageal Surgery Institute, University of Pittsburgh Medical Center, Pittsburgh, PA, USA. 2. University of Rochester Medical Center, Rochester, NY, USA. 3. National Cancer Institute, Rockville, MD, USA.

Invited Discussant: Chuong D. Hoang, MD

F13. Optimized Intrapleural Cisplatin Chemotherapy with a Fibrin Carrier after Extrapleural Pneumonectomy: A Pre-Clinical Study
Isabelle Opitz1, Barbara V. Erne1, Seval Demirbas1, Alexander Jetter2, Burkhardt Seifert3, Rolf Staeheli1, Walter Weder1*
1. Division of Thoracic Surgery, University Hospital, Zurich, Switzerland. 2. Institute for Pharmacology and Toxicology, University Hospital, Zurich, Switzerland. 3. Department of Oncology, University Hospital, Zurich, Switzerland. 4. Institute for Biostatistics, University, Zurich, Switzerland.

Invited Discussant: W. Roy Smythe, MD

F14. Deletion of Tissue Plasminogen Activator Prevents Lung Ischemia-Reperfusion Injury via Inhibition of Neutrophil Extravasation
Yunge Zhao, Abbas Emaminia, Ashish K. Sharma, John Steidle, Gorav Ailawadi, Irving L. Kron1*, Christine L. Lau1*
Surgery, University of Virginia, Charlottesville, VA, USA.

Invited Discussant: Marc de Perrot, MD, MSc

F15. The Chemokine/Chemokine Receptor Pair, CXCL12/CXCR4 Is a Key Pathway in Lung Adenocarcinoma Tumor Cells Tumor Associated Fibroblasts Crosstalk
Ori Wald1, Uzi Izhar1, Gail Amir4, Sophie Kirshberg2, Zippora Shlomai3, Amnon Peled2, OZ M. Shapira1*
1. Cardiothoracic Surgery, Hadassah University Hospital, Jerusalem, Israel. 2. Goldyne Savad Gene Therapy Institute, Hadassah University Hospital, Jerusalem, Israel. 3. Laboratory for Surgical Research, Hadassah University Hospital, Jerusalem, Israel. 4. Pathology, Hadassah University Hospital, Jerusalem, Israel.

Invited Discussant: Steven J. Mentzer, MD

*AATS Member
F16. **WNT Pathway Activation Predicts Increased Risk of Tumor Recurrence in Patients with Stage I Non-Small Cell Lung Cancer**

Mark Shapiro, Gal Akiri, Cynthia S. Chin, Juan P. Wisnivesky, Todd S. Weiser, Scott J. Swanson, Stuart A. Aaronson

1. Surgery, The Mount Sinai Medical Center, New York, NY, USA.
2. Cardiothoracic Surgery, The Mount Sinai Medical Center, New York, NY, USA.
3. Oncological Sciences, The Mount Sinai Medical Center, New York, NY, USA.
4. Medicine, The Mount Sinai Medical Center, New York, NY, USA.
5. Cardiothoracic Surgery, Brigham and Women’s Hospital, Boston, MA, USA.

*Invited Discussant:* Richard J. Battafarano, MD, PhD

F17. **Development of a Navigation System for Segmentectomy Using Infrared Thoracoscopy**

Noriyuki Misaki, Sung Soo Chang, Hitoshi Igai, Shintaro Tarumi, Masashi Gotoh, Hiroyasu Yokomise

General Thoracic Surgery, Breast and Endocrinological Surgery, Kagawa University, Kagawa, Japan.

*Invited Discussant:* Ralph A. Schmid, MD

F18. **Endothelin-1 Mediates Bronchiolitis Obliterans in Lung Transplant Recipients**

Mohamed Salama, Olena Andrukhova, Peter Jaksch, Shahrokh Taghavi, Walter Klepetko, Seyyedhossien Aharinejad

1. Departments of Cardiothoracic Surgery, Medical University of Vienna, Vienna, Austria.
2. Cardiovascular Research, Center of Anatomy and Cell Biology, Medical University of Vienna, Vienna, Austria.

*Invited Discussant:* Daniel Kreisel, MD, PhD

F19. **Trans-Lymphatic Chemotherapy Controls Lymphatic Metastasis and Prolongs Survival in an Orthotopic Lung Cancer Model**

Jiang Liu, Ming Li, Michael R. Johnston

1. Princess Margaret Hospital/University Health Network, Toronto, ON, Canada.
2. Thoracic Surgery, Queen Elizabeth II Health Sciences Centre, Dalhousie University, Halifax, NS, Canada.

*Invited Discussant:* Michael Liptay, MD

*AATS Member*
F20. Profound Cytotoxicity of the Histone Deacetylase Inhibitor SAHA (Suberoylanilide Hydroxamic Acid) and TRAIL (Tumor Necrosis Factor-Related Apoptosis-Inducing Ligand) Combination in Malignant Pleural Mesothelioma

Javier T. Varona Santos¹, Medhi Wangpaichitr², Min You², Niramol Savaraj², Dao M. Nguyen¹*

¹. Division of Cardiothoracic Surgery, Department of Surgery, Miller School of Medicine, University of Miami, Miami, FL, USA. ². Division of Medical Oncology, Department of Medicine, VA Medical Center, University of Miami, Miami, FL, USA.

Invited Discussant: David S. Schrump, MD

*AATS Member
# TUESDAY, MAY 4, 2010

**8:00 a.m.**  
**Building the Hybrid OR of the Future**  
*Room 201, Metro Toronto Convention Centre*  
**Program Directors:**  
Raphael Bueno, MD  
John Byrne, MD

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| 8:00 a.m. – 8:30 a.m. | **The Hybrid OR: Specific Design Features**  
Michael J. Mack, MD  
CT Surgery Associates of North Texas |
| 8:30 a.m. – 9:00 a.m. | **Marketing a Hybrid**  
Michele Toungette  
Vanderbilt Heart & Vascular Institute |
| 9:00 a.m. – 9:30 a.m. | **Financial Aspects of Building a Hybrid OR**  
Gina Cronin  
Cleveland Clinic  
Michael Schroyer  
St. Vincent Heart Center of Indiana |
| 9:30 a.m. – 10:00 a.m. | **The Hybrid OR: Cardiac Clinical Outcomes**  
Steven J. Hoff, MD  
Vanderbilt Heart Institute  
Mike Davidson, MD  
Brigham & Women's Hospital |
<p>| 10:00 a.m. – 10:15 a.m. | <strong>DISCUSSION</strong> |
| 10:15 a.m. – 10:40 a.m. | <strong>BREAK</strong> |</p>
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| 10:40 a.m – 11:10 a.m. | Brigham & Women's Thoracic Hybrid & Vanderbilt Cardiac Hybrid: Lessons Learned | Raphael Bueno, MD  
Brigham and Women's Hospital  
John Byrne, MD  
Vanderbilt Heart & Vascular Institute |
| 11:10 a.m – 11:25 a.m. | DISCUSSION                                               |                                               |
| 11:25 a.m – 11:45 a.m. | Closing                                                  | Raphael Bueno, MD  
Brigham and Women's Hospital  
John Byrne, MD  
Vanderbilt Heart & Vascular Institute |
8:45 a.m.  PLENARY SCIENTIFIC SESSION
Hall C, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)
Moderators:  G. Alec Patterson, MD
            Thoralf M. Sundt, III, MD

28. Surgical Management and Outcome of Patients with Chronic
    Thromboembolic Pulmonary Hypertension: Results from a
    European Prospective Registry
    Eckhard Mayer¹, Jaroslav Lindner², Andrea M. D’Armini³, David Jenkins⁴,
    Walter Klepetko⁵, Philippe G. Dartevelle⁶*
    1. Department of Thoracic Surgery, Kerckhoff Lung Center, Bad Nauheim,
       Germany.  2. Department of Cardiovascular Surgery, Charles University,
       Prague, Czech Republic.  3. Division of Cardiac Surgery, San Matteo Hospital,
       University of Pavia, Pavia, Italy.  4. Department of Cardiothoracic Surgery,
       Papworth Hospital, Cambridge, United Kingdom.  5. Department of
       Cardiothoracic Surgery, Medical University of Vienna, Vienna, Austria.  6.
       Department of Thoracic and Vascular Surgery, Marie Lannelongue Hospital,
       Le Plessis Robinson, Paris-Sud University, Le Plessis Robinson, France.
    Invited Discussant:  Thoralf M. Sundt, III, MD

29. What Is the Role of Complex Coronary Anatomy in Modern Bypass
    Surgery? Lessons Learned from The SYNTAX Trial after Two Years
    Friedrich W. Mohr¹*, Ardawan J. Rastan¹, Patrick W. Serruys²,
    A. Pieter Kappetein², Elisabeth Stahle³, David R. Holmes⁴, Jose L. Pomar⁵*,
    Stephen Westaby⁶*, Katrin Leadley⁷, Keith D. Dawkins⁷, Michael J. Mack⁸*
    1. Cardiac Surgery, Heart Center Leipzig, Leipzig, Germany.  2. Erasmus
       University Medical Center Rotterdam, Rotterdam, Netherlands.  3. University
       Hospital Uppsala, Uppsala, Sweden.  4. Mayo Clinic, Rochester, MN, USA.
       5. Hospital Clinico Y Provincial, Barcelona, Spain.  6. John Radcliffe Infirmary
       Oxford II, Oxford, United Kingdom.  7. Boston Scientific Corporation, Natick,
       MA, USA.  8. Medical City Hospital, Dallas, TX, USA.
    Invited Discussant:  Sheng-shou Hu, MD

30. Surgical Aortic Valvotomy for Congenital Bicuspid Aortic Valve
    Stenosis: When Is It Time to Replace the Valve?
    John W. Brown*, Mark Ruzmetov, Mark D. Rodefeld*, Mark W. Turrentine*
    Cardiothoracic Surgery, Indiana University School of Medicine, Indianapolis,
    IN, USA.
    Invited Discussant:  Thomas L. Spray, MD
31. Robotic-Assisted Thymectomy – Lessons Learned from 160 Cases
Jens C. Ruckert, Marc Swierzy, Mahmoud Ismail
Department of General, Visceral, Vascular and Thoracic Surgery, Universitätsmedizin Berlin – Charité Campus Mitte, Berlin, Germany.
Invited Discussant: Franca M.A. Melfi, MD

10:05 a.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B

10:40 a.m. PLENARY SCIENTIFIC SESSION
Hall C, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)
Moderators: G. Alec Patterson, MD
Thoralf M. Sundt, III, MD

32. Moderate Control of Hyperglycemia is Superior to Tight Control in Patients Undergoing Coronary Artery Bypass Grafting
Castigliano M. Bhamidipati, Damien J. LaPar*, John A. Kern*, James J. Gangemi, Irving L. Kron*, Gorav Ailawadi
Division of Thoracic and Cardiovascular Surgery, Department of Surgery, University of Virginia School of Medicine, Charlottesville, VA, USA.
Invited Discussant: Anthony P. Furnary, MD

33. Does Pre-Operative Sildenafil Protect Against Pulmonary Related Complications Following Cardiopulmonary Bypass? A Randomized Trial in Children Undergoing Cardiac Surgical Repair
Antony Vassalos1, Edward W. Peng1, David Young3, Kenneth J. MacArthur1, James Pollock1, Fiona Lyall2, Mark Danton1
1. Department of Cardiac Surgery, Royal Hospital for Sick Children, Glasgow, United Kingdom. 2. Maternal & Fetal Medicine, Institute of Medical Genetics Yorkhill Hospitals, Glasgow, United Kingdom. 3. Department of Statistics and Modelling Science, University of Strathclyde, Glasgow, United Kingdom.
Invited Discussant: Paul M. Kirshbom, MD

34. Does Earlier Surgery Improve Left Ventricular Mass Regression Following Mitral Valve Repair for Leaflet Prolapse?
John M. Stulak, Rakesh M. Suri, Joseph A. Dearani*, Harold M. Burkhart, Thoralf M. Sundt*, Maurice Sarano, Hartzell V. Schaff*
Cardiovascular Surgery, Mayo Clinic College of Medicine, Rochester, MN, USA.
Invited Discussant: David H. Adams, MD

*AATS Member
11:40 a.m.  HONORED SPEAKER LECTURE
“Too Big to Fail? Healthcare Reform in the U.S. and Canada”
David Naylor, MD
University of Toronto
Introduced By: G. Alec Patterson, MD

12:30 p.m.  ADJOURN FOR LUNCH – VISIT EXHIBITS
Exhibit Hall A & B
2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – ADULT CARDIAC SURGERY
Hall C, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)
Moderators: Lawrence H. Cohn, MD
Carlos A. Mestres, MD, PhD

35. Cardiac Catheterization within 24 Hours of Valve Surgery Is Significantly Associated with Acute Renal Failure
Sara A. Hennessy, Damien J. LaPar*, George J. Stukenborg, John A. Kern*,
Benjamin B. Peeler*, Gorav Ailawadi, Irving L. Kron*
Department of Surgery, University of Virginia, Charlottesville, VA, USA.
Invited Discussant: John G. Byrne, MD

36. An Analysis of Open Aortic Arch Reconstruction in the Endovascular Era
Himanshu J. Patel, Christopher Nguyen, Amy C. Diener, Mary C. Passow,
G. Michael Deeb*
Surgery, University of Michigan Cardiovascular Center, Ann Arbor, MI, USA.
Invited Discussant: Alberto Pochettino, MD

37. Mid-Term Results of Hybrid Aortic Arch Repair Procedures
Kazuo Shimamura1, Toru Kuratani1, Yukitoshi Shirakawa1, Keiwa Kin1,
Masaaki Kato2, Yoshiki Sawa1
1. Department of Cardiovascular Surgery, Osaka University Graduate School of Medicine, Osaka, Japan. 2. Department of Cardiovascular Surgery, Morinomiya Hospital, Osaka, Japan.
Invited Discussant: Anthony L. Estrera, MD

3:00 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B
3:45 p.m.  SIMULTANEOUS SCIENTIFIC SESSION –
ADULT CARDIAC SURGERY
Hall C, Metro Toronto Convention Centre

Moderators: Lawrence H. Cohn, MD
Carlos A. Mestres, MD, PhD

38. Neurocognitive and Neuroanatomic Changes after Off-Pump versus
On-Pump CABG: Long-Term Follow-Up of a Randomized Trial
John D. Puskas1*, Anthony Stringer1, Scott N. Hwang1, Brannon Hatfield1,
A. Shannon Smith1, Patrick Kilgo2, Willis H. Williams1*
1. Cardiothoracic Surgery, Emory University, Atlanta, GA, USA. 2. Rollins
School of Public Health, Emory University, Atlanta, GA, USA.
Invited Discussant: John W. Hammon, MD

39. Administration of Recombinant Activated Factor VII in the Intensive
Care Unit after Complex Cardiovascular Surgery: Clinical and
Economic Outcomes
John M. Toole1, Jason S. Haney2, Martha R. Stroud1, John Lazarchick3,
Fred A. Crawford1*, Walt Uber2, John S. Ikonomidis1*
1. Cardiothoracic Surgery, Medical University of South Carolina, Charleston,
SC, USA. 2. Pharmacy Services, Medical University of South Carolina, Charleston,
SC, USA. 3. Pathology and Laboratory Medicine, Medical University of South
Carolina, Charleston, SC, USA.
Invited Discussant: Arvind K. Agnihotri, MD

40. Diagnosis and Repair of Aortic Valve Cusp Prolapse: Implications for
Valve Sparing Procedures
Munir Boodhwani, Laurent de Kerchove, David Glineur, Christine Watremez,
Jean Rubay, Jean-Louis Vanoverschelde, Phillipe Noirhomme,
Gebrine El Khoury
Cardiovascular and Thoracic Surgery, Cliniques Universitaires Saint Luc,
Brussels, Belgium.
Invited Discussant: Marc R. Moon, MD

5:00 p.m.  EXECUTIVE SESSION
(AATS Members Only)
Constitution 107, Metro Toronto Convention Centre
2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – GENERAL THORACIC SURGERY  
Constitution 106, Metro Toronto Convention Centre  
(8 Minutes Presentation, 12 Minutes Discussion)  
Moderators: Joseph B. Shrager, MD  
David J. Sugarbaker, MD  

41. Predictors of Recurrence, Time to Progression and Disease-Free Survival in Patients with Completely Resected Esophageal Carcinoma  
Paul C. Lee†*, Jeffrey L. Port†*, Subroto Paul†, Brendon M. Stiles†, James Saunders†, Paul Christos‡, Nasser K. Altorki†*  
1. Cardiothoracic Surgery, Weill Cornell Medical College, New York, NY, USA.  
2. Division of Biostatistics and Epidemiology, Department of Public Health, Weill Medical College of Cornell University, New York, NY, USA.  
Invited Discussant: David C. Rice, MD  

42. The Safety of Thoracic Surgery in Patients Taking Clopidogrel (Plavix)  
Robert J. Cerfolio*, Ayesha S. Bryant, Douglas Minnich  
Cardiothoracic Surgery, UAB, Birmingham, AL, USA.  
Invited Discussant: David P. Mason, MD  

43. Single Center Experience of 1000 Adult Lung Transplants  
Daniel Kreisel†, Sasha A. Krupnick†, Varun Puri†, Tracey J. Guthrie†, Elbert Trulock‡, Bryan F. Meyers†*, G. Alec Patterson†*  
1. Surgery, Washington University School of Medicine in St. Louis, St. Louis, MO, USA. 2. Medicine, Washington University in St. Louis, St. Louis, MO, USA.  
Invited Discussant: Shaf Keshavjee, MD, MSc  

3:00 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK  
Exhibit Hall A & B
3:45 p.m.  SIMULTANEOUS SCIENTIFIC SESSION –
GENERAL THORACIC SURGERY
Constitution 106, Metro Toronto Convention Centre
Moderators:  Joseph B. Shrager, MD
David J. Sugarbaker, MD

44. Retrospective Analysis of Two Endoscopic Thoracic Sympathectomy Techniques for Palmar Hyperhidrosis: Clamping versus Cutting of the Sympathetic Chain
Ali N. Ibrahimie, Ted Yanagihara, Alan Weinberg, Joy Hirsch, Catherine R. Harris, Lyall Gorenstein
Invited Discussant:  M. Blair Marshall, MD

45. Expanding the Indications for Laparoscopic Wedge Gastroplasty with Fundoplication for the Shortened Esophagus
Daniel C. Wiener, Jon O. Wee, Abraham Lebenthal, David J. Sugarbaker, Raphael Bueno
1. Thoracic Surgery, Brigham and Women's Hospital, Boston, MA, USA. 2. Surgical Oncology, Fox Chase Cancer Center, Philadelphia, PA, USA.
Invited Discussant:  Michael A. Maddaus, MD

46. A Prospective European Multicenter Randomized Trial to Evaluate Pleureaseal for Control of Air Leaks after Elective Pulmonary Resection
Paul De Leyn, Michael R. Mueller, Jan W. Oosterhuis, Thomas Schmid, Clifff K. Choong, Walter Weder, Youri Sokolov, Peter De Rooij
1. Department of Thoracic Surgery, University Hospitals Leuven, Leuven, Belgium. 2. Otto Wagner Hospital, Vienna, Austria. 3. VU Medisch Centrum, Amsterdam, Netherlands. 4. Universitaetsklinik Landeskrankenhaus Innsbruck, Innsbruck, Austria. 5. Papworth Hospital, Cambridge, United Kingdom. 6. Division of Thoracic Surgery, University Hospital, Zurich, Switzerland. 7. Service de Chirurgie Thoracique, Hopital Erasme, Brussels, Belgium. 8. Medical Centre Rotterdam Zuid, Rotterdam, Netherlands.
Invited Discussant:  Alessandro Brunelli, MD

5:00 p.m.  EXECUTIVE SESSION
(AATS Members Only)
Constitution 107, Metro Toronto Convention Centre

*AATS Member
2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – CONGENITAL HEART DISEASE
Constitution 105, Metro Toronto Convention Centre
(8 Minutes Presentation, 12 Minutes Discussion)
Moderators: Frank A. Pigula, MD
James S. Tweddell, MD

47. In-Situ Pericardial Extracardiac Lateral Tunnel Fontan Operation: Fifteen-Year Experience
Nahidh W. Hasaniya, Anees J. Razzouk*, Leonard L. Bailey*
Cardiothoracic Surgery, Loma Linda University Medical Center, Loma Linda, CA, USA.
Invited Discussant: Erle H. Austin, III, MD

48. Primary Sutureless Procedure for “Simple” Total Anomalous Pulmonary Venous Connection: Mid-Term Results in a Single Institution
Bobby Yanagawa, Abdullah A. Alghamdi, Christopher A. Caldarone*, John G. Coles*, Osman O. Al-Radi, Glen S. Van Arsdell*
Surgery, Division of Cardiac Surgery, University of Toronto, Toronto, ON, Canada.
Invited Discussant: Francois Lacour-Gayet, MD

49. Repair of Major Cardiac Defects in Low Birth Weight Infants: Is Delayed Surgical Intervention Warranted?
Charles Sheppard2, James H. Moller2, Roosevelt Bryant1, Ronald M. Rosengart*, James D. St. Louis3
1. Department of Surgery, University of Minnesota, Minneapolis, MN, USA.
2. Department of Pediatrics, University of Minnesota, Minneapolis, MN, USA.
Invited Discussant: Peter J. Gruber, MD, PhD

3:00 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B
3:45 p.m.  SIMULTANEOUS SCIENTIFIC SESSION –
CONGENITAL HEART DISEASE
Constitution 105, Metro Toronto Convention Centre
Moderators:  Frank A. Pigula, MD
James S. Tweddell, MD

50. Precise Evaluation of Bilateral Pulmonary Artery Banding for Initial Palliation for High-Risk Hypoplastic Left Heart Syndrome
Kazuo Kitahori¹, Arata Murakami¹, Tetsuhiro Takaoka¹, Shinichi Takamoto², Minoru Ono¹
1. Cardiothoracic Surgery, The University of Tokyo, Hospital, Tokyo, Japan.
2. Cardiothoracic Surgery, Mitsui Memorial Hospital, Tokyo, Japan.
Invited Discussant:  Christopher A. Caldarone, MD

51. Determinants of Outcome after Surgical Treatment of Pulmonary Atresia with Ventricular Septal Defect and Major Aortopulmonary Collateral Arteries
Adriano Carotti¹, Sonia B. Albanese¹, Sergio Filippelli¹, Lucilla Rava², Paolo Guccione¹, Giacomo Pongiglione¹, Roberto M. Di Donato¹*
1. Department of Pediatric Cardiology and Cardiac Surgery, Bambino Gesù Children’s Hospital, Rome, Italy.
2. Epidemiology Unit, Bambino Gesù Children’s Hospital, Rome, Italy.
Invited Discussant:  Frank L. Hanley, MD

52. The Cone Reconstruction of the Tricuspid Valve in Ebstein Anomaly with or without 1.5-Ventricle Repair
Jinfen Liu⁴, Lisheng Qiu, Zhongqun Zhu, Huiwen Chen, Haifa Hong
Shanghai Children’s Medical Center, Shanghai, China.
Invited Discussant:  Pedro J. del Nido, MD

5:00 p.m.  EXECUTIVE SESSION
(AATS Members Only)
Constitution 107, Metro Toronto Convention Centre

*AATS Member
WEDNESDAY MORNING
MAY 5, 2010

7:00 a.m. EMERGING TECHNOLOGIES AND TECHNIQUES FORUM
Hall C, Metro Toronto Convention Centre
(6 Minutes Presentation, 5 Minutes Discussion)

Moderators: Joseph E. Bavaria, MD
Bryan F. Meyers, MD, MPH

T1. Continuing Experience with a Repositionable Inflatable
Transcatheter Valve: Direct Flow Medical One Year and Beyond
Hendrik Treede1, Thilo Tuebler2, Hermann Reichenspurner1*,
Eberhard Grube3, Andrea Pascotto2, Olaf Franzen1, Ralf Mueller3,
Reginald Low4, Steven F. Bolling5*, Thomas Meinertz1, Joachim Schofer2
1. Department of Cardiovascular Surgery and Department of Cardiology,
University Heart Center Hamburg, Hamburg, Germany. 2. Medical Care
Center Prof. Mathey, Prof Schofer, Hamburg University Cardiovascular
Center, Hamburg, Germany. 3. Department of Cardiology Angiology,
HELIOS Heart Center Siegburg, Siegburg, Germany. 4. Division of
Cardiovascular Medicine, University of California Davis Medical Center,
Sacramento, CA, USA. 5. University of Michigan Cardiovascular Center,
Ann Arbor, MI, USA.

T2. Sutureless Aortic Valve Replacement with the Trilogy Trilobal
Aortic Valve System-Multicenter Experience
Ingo Breitenbach1, Jerzy Sadowski3, Gerhard Wimmer-Greinecker5,
Christoph Schmitz2, Leo A. Bockeria4*, Krzysztof Bartus3,
Ravil M. Muratov4, Wolfgang Harringer1
1. Department of Thoracic and Cardiovascular Surgery, Klinikum
Braunschweig, Braunschweig, Germany. 2. Department of Cardiac Surgery,
University of Munich, Munich, Germany. 3. Department of Cardiovascular
Surgery and Transplantology, Jagiellonian University, Krakow, Poland.
4. Bakoulev Scientific Center for Cardiovascular Surgery, Moscow,
Russia. 5. Department of Thoracic and Cardiovascular Surgery, Herzund
Gefässzentrum Bad Bevensen, Bad Bevensen, Germany.

*AATS Member
T3. Closed Chest Intra-Cardiac Mitral Valve Repair with the Mitraclip System By the Valve Interventionalists: Interventional Cardiologists or Cardiac Surgeons at a Single Center
Scott Lim1, Gorav Ailawadi2, Michael Ragosta3, John A. Kern2*, John Dent3, Linda G. Bailes3, Irving L. Kron2*
1. Medicine & Pediatrics, University of Virginia, Charlottesville, VA, USA. 2. Surgery, University of Virginia, Charlottesville, VA, USA. 3. Medicine, University of Virginia, Charlottesville, VA, USA.

T4. A Fluid Diode for Control of Pulmonary Insufficiency
Tain-Yen Hsia1, Tiffany Camp2, Tim McQuinn3, Richard S. Figliola2

T5. Truly Stentless Autologous Pericardial AVR: An Alternative to Standard AVR
K.M. John Chan, Jemyrr Therese A. Gavino, Gilles D. Dreyfus*
Cardiothoracic Surgery, Royal Brompton and Harefield NHS Foundation Trust, Harefield, United Kingdom.

T6. Effect of Transapical Aortic Valve Implantation for Aortic Stenosis on Severity of Mitral Regurgitation
Robert L. Smith2, Arnaud Van Linden1, Joerg Kempfert1, Ines Schimpke1, Gerhard Schuler1, Friedrich W. Mohr1*, Thomas Walther1
1. Department of Cardiac Surgery, Heart Center, University of Leipzig, Leipzig, Germany. 2. Cardiopulmonary Research Science and Technology Institute, Dallas, TX, USA.

T7. Transapical Transcatheter Mitral Valve in Valve Implantation: A Case Series
Anson Cheung, Jian Ye, John Webb, David A. Wood, Ronald G. Carere, Christopher Thompson, Samuel V. Lichtenstein
University of British Columbia, Vancouver, BC, Canada.
Zachary N. Kon, Amod Tendulkar, Zhongjun Wu, Aldo T. Iacono, Brian McCormick, Bartley P. Griffith*, Jose P. Garcia
University of Maryland School of Medicine, Baltimore, MD, USA.

T9. Preliminary Results of Anatomic Lung Resection Utilizing the Liga Sure Energy Based Tissue and Vessel Coagulative Fusion Technology
Matthew J. Schuchert, Ghulam Abbas, Brian L. Pettiford, James D. Luketich*, Rodney J. Landreneau*
Heart, Lung and Esophageal Surgery Institute, University of Pittsburgh Medical Center, Pittsburgh, PA, USA.

T10. The Novel Use of Coil Spring Fiducials Placed via Navigation Bronchoscopy in Inoperable Patients Allows for the Safe and Effective Delivery of Cyberknife Stereotactic Radiation
Carsten Schroeder1, Rana Hejal2, Philip Linden1
1. Thoracic & Esophageal Surgery, Case Medical Center, Cleveland, OH, USA. 2. Pulmonary Critical Care & Sleep Medicine, Case Medical Center, Cleveland, OH, USA.
9:00 a.m.  CONTROVERSIES IN CARDIOTHORACIC SURGERY
Hall C, Metro Toronto Convention Centre
Moderator: Irving L. Kron, MD

Randomized Controlled Clinical Trials are Necessary to Evaluate New Surgical Operations

Pro: Timothy J. Gardner, MD
Con: Joel D. Cooper, MD

10:00 a.m.  ADULT CARDIAC: SURGICAL THERAPIES FOR CONGESTIVE HEART FAILURE
Constitution 107, Metro Toronto Convention Centre
Chairs: Vivek Rao, MD, PhD
University of Toronto
Thoralf M. Sundt, III, MD
Mayo Clinic

COURSE OBJECTIVES

At the conclusion of this course, participants will be able to:

- Employ evidence-based medical management of heart failure patients presenting for cardiac surgical intervention.
- Understand the rationale and role of device therapy (ICD and CRT) for the postoperative management of patients with ischemic cardiomyopathy.
- Compare the medium and long-term outcomes of coronary bypass surgery, surgical ventricular reconstruction and mitral valve repair in patients with ischemic and non-ischemic cardiomyopathy.
- Implement optimal surgical therapy for patients with end-stage heart disease.
10:00 a.m. – 10:20 a.m.  Medical Management of CHF: “What the Surgeon Needs to Know”
Heather J. Ross, MD, MHSc
University of Toronto

10:20 a.m. – 10:40 a.m.  What Is the Role of CRT and ICD after Cardiac Surgery?
Justin A. Mariani, MD
University of Toronto

10:40 a.m. – 11:00 a.m.  Life after STICH: When Do We Repair the Dysfunctional LV?
Robert E. Michler, MD
Albert Einstein Medical College

11:00 a.m. – 11:20 a.m.  Mitral Repair in Non-Ischemic Cardiomyopathy: Is It a Viable Long-Term Solution?
Steven F. Bolling, MD
University of Michigan

11:20 a.m. – 11:40 a.m.  Ventricular Restraint Therapies: Beyond Acorn and HeartNet
Michael A. Acker, MD
University of Pennsylvania

11:40 a.m. – 12:00 p.m.  Mechanical Circulatory Support in 2010: An Update
Nicholas G. Smedira, MD
Cleveland Clinic Foundation

12:00 p.m.  ADJOURN
### AMERICAN ASSOCIATION FOR THORACIC SURGERY

**10:00 a.m.**
**CONGENITAL: NEW TECHNOLOGY IN CONGENITAL HEART DISEASE**

*Constitution 105, Metro Toronto Convention Centre*

**Chairman:** Christopher A. Caldarone, MD  
The Hospital for Sick Children

### COURSE OBJECTIVES

At the conclusion of this course, participants will be able to:

- Select patients appropriate for implantation of mechanical assist devices in children.
- Discuss future applications of cell-based therapies for end-stage heart disease in the congenital population.
- Apply novel modalities of myocardial protection.

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<th>Time</th>
<th>Event</th>
<th>Presenter</th>
<th>Institution</th>
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| 10:00 a.m. – 10:15 a.m. | **Intracardiac Surgery on the Beating Heart**                  | Pedro J. del Nido, MD  
Children's Hospital Boston                        |
| 10:15 a.m. – 10:20 a.m. | **DISCUSSION**                                         |
| 10:20 a.m. – 10:35 a.m. | **Mechanical Assist for Single Ventricle Failure** | Mark D. Rodefeld, MD  
Indiana University                              |
| 10:35 a.m. – 10:40 a.m. | **DISCUSSION**                                         |
| 10:40 a.m. – 10:55 a.m. | **Automated Remote Ischemic Preconditioning in Pediatric Patients** | Andrew N. Redington, MD  
The Hospital for Sick Children                    |
| 10:55 a.m. – 11:00 a.m. | **DISCUSSION**                                         |
11:00 a.m. – 11:15 a.m.  
Robotic Surgery for Congenital Heart Disease  
Johannes Bonatti, MD  
University of Maryland

11:15 a.m. – 11:20 a.m.  
DISCUSSION

11:20 a.m. – 11:35 a.m.  
Real-Time MRI-Guided Procedures  
Keith A. Horvath, MD  
National Heart, Lung and Blood Institute

11:35 a.m. – 11:40 a.m.  
DISCUSSION

11:40 a.m. – 11:55 a.m.  
Cell-Based Therapies for Congenital Heart Disease  
Richard D. Weisel, MD  
Toronto General Hospital

11:55 a.m. – 12:00 p.m.  
DISCUSSION

12:00 p.m.  
ADJOURN
10:00 a.m. GENERAL THORACIC: CONTROVERSIES IN THE UTILIZATION OF NEW TECHNOLOGY
Constitution 106, Metro Toronto Convention Centre
Chairs: Thomas A. D’Amico, MD
Duke University
Shaf Keshavjee, MD, MSc
Toronto General Hospital

COURSE OBJECTIVES
At the conclusion of this course, the participants will be able to:

- Identify the indications and technical aspects for ablative techniques for patients with Barrett’s esophagus.
- Determine the role for RFA or SBRT in marginally operable patients with Clinical Stage I non-small cell lung cancer.
- Utilize mediastinal staging techniques, including EBUS, mediastinoscopy, VAMLA and TEMLA, in appropriately selected patients with lung cancer.

SESSION I:
MANAGEMENT OF BARRETT’S HGD AND T1a CARCINOMA
Moderator: Gail E. Darling, MD, University of Toronto

<table>
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<th>Time</th>
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<th>Speaker</th>
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<tr>
<td>10:00 a.m. – 10:15 a.m.</td>
<td>RFA, EMR and Other Ablative Techniques</td>
<td>Wayne Hofstetter, MD MD Anderson Cancer Center</td>
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<td>10:15 a.m. – 10:30 a.m.</td>
<td>Esophagectomy</td>
<td>Kemp H. Kernstine, MD PhD City of Hope National Medical Center</td>
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<td>10:30 a.m. – 10:40 a.m.</td>
<td>DISCUSSION</td>
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SESSION II: 
MANAGEMENT OF CLINICAL STATE I NSCLC

*Moderator:* Joseph B. Shrager, MD, Stanford University

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<th>Institution/University</th>
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<tr>
<td>10:40 a.m. – 10:55 a.m.</td>
<td>SBRT/RFA</td>
<td>Malcolm M. DeCamp, MD Northwestern Memorial Hospital</td>
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<td>10:55 a.m. – 11:10 a.m.</td>
<td>Thoracoscopic Lobectomy and Segmentectomy</td>
<td>Thomas A. D'Amico, MD Duke University</td>
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<td>11:10 a.m. – 11:20 a.m.</td>
<td>DISCUSSION</td>
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<td>11:20 a.m. – 11:35 a.m.</td>
<td>Role of EBUS</td>
<td>Kazuhiro Yasufuku, MD, PhD Toronto General Hospital</td>
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<td>11:35 a.m. – 11:50 a.m.</td>
<td>Mediastinoscopy, VAMLA, TEMLA</td>
<td>Todd L. Demmy, MD, Roswell Park Cancer Institute</td>
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<td>11:50 a.m. – 12:00 p.m.</td>
<td>DISCUSSION</td>
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SESSION III: 
MEDIASTINAL STAGING

*Moderator:* G. Alec Patterson, MD, *Washington University School of Medicine*

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<td>11:50 a.m. – 12:00 p.m.</td>
<td>DISCUSSION</td>
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<td>12:00 p.m.</td>
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Endobronchial Ultrasound (EBUS) Training Course
Toronto Medical Discovery Tower
MARS Large Animal OR
101 College St, Toronto, Canada

(Separate subscription pre-registration required.)

Course Faculty:
Shaf Keshavjee, MD, MSc
Head, Division of Thoracic Surgery, UHN
Director, Thoracic Surgery Research
Director, Toronto Lung Transplant Program
Professor and Chair, Division of Thoracic Surgery
University of Toronto

Kazuhiro Yasufuku, MD, PhD
Assistant Professor, Division of Thoracic Surgery
University of Toronto

Andrew Pierre, MD
Assistant Professor, Division of Thoracic Surgery
University of Toronto

1:00 p.m. – 2:00 p.m. LECTURE
(Lunch Provided)
Welcome and Introduction
Shaf Keshavjee, MD, MSc
EBUS-TBNA
(Procedure, Results, Pig Anatomy)
Kazuhiro Yasufuku, MD, PhD
Demonstration with Dry Model
BREAK AND CHANGE
(15 minutes)
2:00 p.m. – 5:00 p.m.
HANDS-ON SESSION
(Wet Lab)
(1) Handling of Equipments
   (balloon attachment, needle, etc.)
(2) Training with a Phantom
(3) EBUS-TBNA in Pigs
(4) Rapid On-Site Evaluation of Specimens
(5) Q&A
BREAK
(20 minutes)
CLOSING REMARKS
Shaf Keshavjee, MD, MSc
ADJOURN
SUNDAY AFTERNOON
MAY 2, 2010

3:00 p.m. – 5:00 p.m.  13th Annual C. Walton Lillehei Resident Forum
206C, Metro Toronto Convention Centre
(7 minutes presentation, 8 minutes discussion)

Chairs:
John S. Ikonomidis, MD
Medical University of South Carolina
Glen S. Van Arsdell, MD
The Hospital for Sick Children

L1. BRCA1 Is a Novel Regulator of Cardiac Function via Altering
Myocardial Substrate Utilization and Mitochondrial
Bioenergetics
Bobby Yanagawa, Praphulla Shukla, Krishna K. Singh, Hwee Teoh,
Subodh Verma
Division of Cardiac Surgery, St. Michael’s Hospital, University of
Toronto, Toronto, ON, Canada.

OBJECTIVE: Improving myocardial metabolism is a novel approach to
limit ischemic cardiac dysfunction. We evaluated the effects of BRCA1, a
tumor suppressor gene implicated in DNA repair, to regulate myocardial
substrate utilization and mitochondrial bioenergetics.

METHODS: Mice homozygous for exon 11 floxed BRCA1 allele (BRCA1fl/fl)
were crossed with heterozygous mice expressing Cre recombinase under
the control of the α-myosin heavy chain (αMHC-Cretg/-) promoter. Mice
demonstrating the αMHC-Cretg/-; BRCA1fl/fl were identified as cardio-
myocyte specific BRCA1 homozygous (CM-BRCA1-/-) and with the
αMHC-Cretg/-; BRCA1fl/+ combination as CM-BRCA1+/-. Functional analyses were
performed and hearts were submitted for pathological and molecular
analyses.

RESULTS: Acute and chronic post-MI survival were markedly lower in CM-
BRCA1+/- and CM-BRCA1-/- mice compared to WT controls (7d: 92% and
93% vs. 100%; 180d: 43% and 29% vs. 96%; Figure), with early profound
ventricular thinning and rupture. Infarct sizes were larger in CM-BRCA1+/-
(55 ± 7%) and CM-BRCA1-/- (56 ± 9%) mice compared to WT controls
(32 ± 23%), with a 1.6-fold increase radius-to-septum thickness consistent
with adverse cardiac remodeling. Pressure-volume measurements showed a 2-fold greater end-systolic and end-diastolic volume and a 50% lower ejection fraction in CM-BRCA1-KO mice. Hearts from CM-BRCA1 deficient mice exhibited increased apoptotic Bcl-2 pathway activation and a 3-fold higher expression of \( \gamma \)-H2AX, a marker of double strand DNA damage. Myocardial expression of PPAR\( \alpha \) and \( \gamma \), and PPAR-responsive genes, GLUT1, GLUT 4, and the fatty acid transporters CD36 and carnitine palmitoyltransferase 1 were markedly attenuated (p < 0.01; Figure). Also reduced transcript expression of acetyl-CoA carboxylase 2 and malonyl-CoA decarboxylase, rate limiting enzymes in fatty acid synthesis, along with a 4-fold reduction in AMP-activated protein kinase expression were shown. From a cellular standpoint these changes likely occurred via a reduction in PPAR-\( \gamma \) coactivator 1\( \alpha \), a key regulator of mitochondrial bioenergetics, which was downregulated in CM-BRCA1-KO hearts (p < 0.01).

**CONCLUSION:** We demonstrate an essential role of BRCA1 as a gatekeeper of cardiac function and survival in response to ischemia. BRCA1 deficiency may result in an energy compromised myocardium, primarily through alterations in myocardial carbohydrate and fatty acid metabolism, likely via a PPAR-dependent mitochondrial bioenergetics.
AP2β Nucleolar Localization Predicts Poor Survival after Lung Cancer Resection in Stage I Patients

Min P. Kim¹, Ying Chen², Adriana Lopez³, Ignacio Wistuba², Lin Ji¹, Jack A. Roth¹*, Ruth L. Katz²

1. Thoracic and Cardiovascular Surgery, MD Anderson Cancer Center, Houston, TX, USA. 2. Pathology, MD Anderson Cancer Center, Houston, TX, USA. 3. Biostatistics, MD Anderson Cancer Center, Houston, TX, USA.

OBJECTIVE: Activating enhancer-binding protein-2β (AP2β) is a nuclear transcription factor that activates human telomerase reverse transcriptase (hTERT) expression. It was recently shown that up-regulation of AP2β leads to increased telomerase expression, whereas down regulation of AP2β inhibits both telomerase activity and tumor cell growth. We hypothesized that AP2β expression may be a predictor of poor survival in lung cancer patients.

METHODS: Immunohistochemistry staining (IHC) for AP2β was performed on tissue microarrays prepared from resected stage I NSCLC from 126 patients. There were two distinct patterns of staining: 1) diffuse nuclear staining; 2) nucleolar staining characterized by staining of nucleoli, with or without diffuse nuclear staining. The staining pattern and IHC intensity score were correlated with prospectively collected clinical data. Validation was performed with a non-overlapping group of 115 stage I NSCLC patients.

RESULTS: IHC confirmed co-localization of telomerase and AP2β in lung cancer cells. The IHC intensity score did not correlate with the stage, survival, tumor differentiation, or histology. The patients with a nucleolar pattern had a significantly worse five-year survival (67%) compared to patients with a diffuse nuclear pattern (100%, p = 0.009). Multivariate analysis showed that nucleolar AP2β pattern (p = 0.004, HR 13.9, CI 1.9–1782.1) was an independent predictor for poor survival. The validation set confirmed that patients with a nucleolar pattern had a significantly worse five-year survival (64%) compared to patients with a diffuse nuclear pattern (91%, p = 0.02). Multivariate analysis of the validation set confirmed that the nucleolar AP2β pattern (p = 0.037, HR 2.18, CI 1.05–4.51) was an independent predictor for poor survival.

* AATS Member
CONCLUSION: Genome wide mRNA or proteomic expression array analysis would not have identified the prognostic significance of AP2β since the expression level of the AP2β protein did not correlate with survival. The movement of telomerase to nucleoli is a response to DNA damaging agents and prevents inappropriately added telomere ends during DNA replication. A possible explanation for our results is that tumor cells that maintain AP2β and hTERT in the nucleoli may be more aggressive due to resistance to DNA damage. We conclude that AP2β localization may be a useful biomarker for predicting survival in stage I NSCLC, and that intracellular protein localization should be considered in the evaluation of prognostic biomarkers.

The stage I lung cancer patients with nucleolar pattern had significantly worse five year survival (67%) compared to patients with diffuse pattern (100%).
OBJECTIVE: Despite improvements in current therapy, the presence of nodal disease decreases 5-year survival in NSCLC by 40%. A simple method to concentrate toxic chemotherapy within these lymph nodes (LNs) could substantially improve survival in patients with NSCLC. This study investigates various formulations of acrylate-based nanoparticles (NP) as a method for locoregional lymphatic drug delivery, targeting these LNs at risk.

METHODS: Two distinct NP formulations were synthesized from acrylate monomers with either a trimethoxybenzaldehyde or 12 carbon (C12) side-chain. In order to study to the effects of particle size on migration each formulation was synthesized in two sizes (50–60 nm and >100 nm). A 400 μL suspension of each NP, fluorescently labeled with coumarin and encapsulating the near infrared (NIR) dye IR-786, was injected subcutaneously into the chest wall or hindleg of pigs. Animals were imaged 24 hours post-injection using the FLARE™ NIR imaging system to assess for lymphatic migration. Fluorescent LNs containing NP were excised, cryosectioned, and examined for the presence of coumarin-labeled NPs and IR-786 payload with fluorescent microscopy.

RESULTS: Successful migration of NPs from injection site to the sentinel lymph node (SLN) was size-dependent. 50 nm and 60 nm NP showed migration to the regional SLN(s) within 24 hours of injection in all cases (n = 6 for each polymer; Figure 1), while particles 100 nm or larger, irrespective of polymer side chain, showed minimal migration in only 1 instance (n = 4 for each polymer; p < 0.01, Fisher’s Exact Test). Additionally, the pattern of migration varied depending on the NP polymer. Histologic examination of the SLN confirmed that varying the side chains of the acrylate monomer resulted in localization within different compartments.
of the LN. 50 nm particles with a trimethoxybenzaldehyde side chain remained confined within the subcapsular and sinusoidal spaces of the LN, while similarly sized particles with C12 side chain localized to the germinal centers.

**CONCLUSION:** This study demonstrates the capabilities of polymer NP for drug delivery to various lymphatic targets. Migration characteristics of NP were both size and polymer dependent. By adjusting particle diameter and formulation, NP can be targeted to various compartments within a LN including the subcapsular spaces and germinal centers. Given the differing LN functions in these areas, these targeting methods may prove useful for future oncologic and immunologic lymphatic therapies.

**Figure 1:** NIR-labeled 60 nm methacrylate nanoparticles with a C12 side chain migrate from the subcutaneous injection site (arrowhead) to the regional draining lymph nodes (arrow) through a faint lymphatic channel (open arrow).
L4. **Dabigatran Is Effective for Thromboprophylaxis of Mechanical Heart Valves**

Stephen H. McKellar¹, Stuart Abel¹, Christopher Camp¹, Mark Ereth², Hartzell V. Schaff¹*


**OBJECTIVE:** Warfarin reduces the risk of stroke in patients with mechanical heart valves but increases the risk of hemorrhage and is difficult to use. Dabigatran, a new oral direct thrombin inhibitor, has been shown to be effective in reducing the risk of stroke among patients with atrial fibrillation. No such data exists in the setting of mechanical heart valves. We tested the hypothesis that dabigatran is as effective as heparin for thromboprophylaxis of mechanical valves using a porcine heterotopic aortic valve model.

**METHODS:** Thirty swine underwent implantation of a modified bileaflet mechanical valved conduit bypassing the ligated, native descending thoracic aorta. Animals were randomized to receive no anticoagulation (AC) (n = 10), enoxaparin 2 mg/kg SQ BID (n = 10), or dabigatran 20 mg PO BID (n = 10). The primary end point was the amount of valve thrombus (mg) at 30 days. Secondary endpoints included quantitative measurement of platelet deposition on the valve prosthesis, thromboelastography (TEG), hemorrhagic and embolic events.

**RESULTS:** At 30 days, we observed a mean of 638 ± 895 mg thrombus for the no AC group, 121 ± 128 mg for the enoxaparin group, and 19 ± 31 mg for the dabigatran group (P = 0.01 for enoxaparin vs. dabigatran). Fewer platelets were deposited on the valves from the dabigatran group (2.7 x 10⁸) compared to the enoxaparin group (1.8 x 10⁹), (P = 0.03). No major or occult hemorrhagic or embolic events were observed. TEG analysis demonstrated that dabigatran produced less prolongation of the K value (P = 0.01) and less decrease in the angle (P = 0.01) and MA (P = 0.001) values compared to enoxaparin.

**CONCLUSION:** The direct thrombin inhibitor dabigatran is as effective as enoxaparin for short-term thromboprophylaxis of mechanical valves as it best prevented valve thrombus and platelet deposition at 30-days without increased adverse events. The TEG profile of dabigatran is different than that of enoxaparin. These promising results should be tested in long-term animal studies and, if confirmed, should serve as a foundation for prospective clinical trials with dabigatran as an alternative to warfarin in patients with bileaflet mechanical aortic valves.

*AATS Member*
L5. Safe, Efficient, and Durable Transduction of Human and Canine Saphenous Vein Grafts Using a Modified Adeno Associated Viral Vector

Mani A. Daneshmand¹, Nestor R. Villamizar¹, Roberto J. Manson¹, Thomas Mulhearn², Christopher D. Kontos², Carmelo A. Milano¹, Dawn Bowles¹

¹. Surgery, Duke University Medical Center, Durham, NC, USA. 2. Medicine, Division of Cardiology, Duke University Medical Center, Durham, NC, USA.

OBJECTIVE: The effectiveness of SVGs in coronary artery bypass surgery is compromised by a high incidence of failure. Gene therapy with anti-proliferative agents limits intimal hyperplasia and SVG failure in experimental models. As a gene delivery agent, AAV vectors are notable for their beneficial safety profile and long-term gene expression. The purpose of this study was to determine the optimal conditions for AAV-mediated SVG transduction in anticipation of clinical trials.

METHODS: Cultured human aortic smooth muscle cells (HASMC) (n = 4/group) and segments of human SV (n = 3–7/group) were utilized to evaluate the transduction efficiency of a panel of luciferase-encoding AAV vectors (AAV serotypes 1–9 or SASTG, a modified capsid vector). Human SVs were infected with AAV ex vivo, and residual infectious virus DNA present in serial flushes was quantified by real-time PCR. In vivo, AAV transduction efficiency was evaluated using a canine carotid artery to jugular vein fistula model. SV segments transduced with either AAV2- or SASTG-luciferase (1 × 10¹² total viral particles/vein) were analyzed for luciferase expression by luminometry and bioluminescence imaging 30 or 90 days after gene delivery (n = 5/vector type for both time points).

RESULTS: In HASMC and cultured SV segments the SASTG vector induced significantly greater luciferase transgene activity relative to other AAV serotypes (See Figure for comparison to AAV2; P < 0.05). Greater than 98% of residual vector was removed from the human vein following a single ex vivo PBS flush. In vivo, robust luciferase expression was detected in SVG 30 days after delivery and was sustained at 90 days. Notably, within the same animal, SASTG treated grafts achieve on average 3.7-fold higher transgene expression compared to grafts treated with AAV2 (p = 0.0486) (See Figure).
CONCLUSION: This is the first study to evaluate the transduction properties of different AAV vectors in large caliber veins. In all model systems, a novel genetically modified AAV capsid (SASTG) led to significantly greater transgene expression relative to other AAV serotypes. 90-day expression was observed in vivo, reflecting the longest documented transgene expression in SVG. Little residual infectious virus was detectable prior to anastomosis, supporting the safety of this approach. These findings pave the way for the use of AAV vectors as a safe and effective means of gene delivery for the prevention of vein graft disease.
L6. Effects of Annuloplasty Ring Implantation on Regional Mitral Leaflet Tenting Area During Acute Myocardial Ischemia

Wolfgang Bothe1, Elizabeth H. Stephens1, John-Peder E. Kvitting1, Julia C. Swanson1, Neil B. Ingels2, D. Craig Miller1*

1. Cardiothoracic Surgery, Stanford School of Medicine, Stanford, CA, USA.
2. Laboratory of Cardiovascular Physiology and Biophysics, Research Institute of the Palo Alto Medical Foundation, Mountain View, CA, USA.

OBJECTIVE: Preoperative tenting area (TA) is a predictive factor for recurrent mitral regurgitation (MR) in patients with ischemic MR undergoing annuloplasty. TA decreases with ring implantation, but may increase after surgery and be associated with recurrent MR. Insights into regional alterations in TA before and after ring implantation could improve our understanding of mechanisms leading to ischemic MR and recurrent MR after annuloplasty. We quantified regional changes in mitral leaflet TA during acute myocardial ischemia in an ovine model with and without an annuloplasty ring.

METHODS: 14 radiopaque markers were implanted in 7 adult sheep: one to the mitral annular saddle horn (MASH), 4 to the central septal-lateral (S-L) meridian of the anterior mitral leaflet (AML), 4 to the commissure-commissure (C-C) meridian of the AML, 2 to the central S-L meridian of the posterior leaflet (PML), one to the mid-lateral mitral annulus (MAML) and one each to the anterior and posterior commissures (ACOM, PCOM). True-sized CE Physio annuloplasty rings were inserted in a releasable fashion. Under acute open-chest conditions, marker coordinates were obtained using biplane videofluoroscopy (60 Hz) with ring inserted at baseline (BL + RING) and after 90s of LCx occlusion (ISCH + RING). After ring release, another dataset was acquired at baseline (BL) and after 90s of LCx occlusion (ISCH). Proximal (Area 1), distal (Area 2), total (Area 1 + 2) AML S-L TA, AML C-C TA (Area 3) and PML S-L TA were computed at mid-systole from sums of marker triangles with the midpoint between MASH and MAML being the vertex for the S-L TA triangles and the midpoint between ACOM and PCOM the vertex for the AML C-C TA triangles (Figure A).

RESULTS: Figure B: Compared to BL, MR grade and all measured TAs significantly increased with ISCH. Relative to BL + RING, the AML S-L TAs (Area 1, Area 2, Area 1 + 2) and MR grade did not change in ISCH+RING; surprisingly, however, AML C-C TA and PML S-L TA both increased.

*AATS Member
Ring implantation prevented acute ischemic MR despite the presence of significant commissural AML and S-L PML tenting, most likely due to minimizing AML S-L tenting. Assuming the results from our acute experimental ischemic preparation hold true in patients with chronic IMR, disease-specific annuloplasty rings should aim primarily to reduce the mitral annular S-L dimension. Our new observation suggests that normalizing C-C tethering, e.g., via new ring designs or subvalvular approaches, may also help to optimize long-term outcomes.
L7. Pro-Inflammatory Role of A2B Adenosine Receptor in Lung Ischemia-Reperfusion Injury

Farshad Anvari1, Ashish K. Sharma1, Lucas G. Fernandez1, Katya Ravid2, Irving L. Kron1*, Victor E. Laubach1

1. Surgery, University of Virginia, Charlottesville, VA, USA 2. Biochemistry, Boston University School of Medicine, Boston, MA, USA.

OBJECTIVE: Reperfusion injury after lung transplantation remains a major source of morbidity and mortality. Adenosine receptors have been implicated in both pro-inflammatory and anti-inflammatory roles in ischemia-reperfusion (IR) organ injury. We have previously demonstrated that the A2A adenosine receptor has an anti-inflammatory role in lung IR injury. We hypothesized that the A2B adenosine receptor has a pro-inflammatory role in lung IR injury.

METHODS: An in-vivo mouse left lung hilar clamp model of IR was utilized. Wild-type C57BL6 mice (WT) were used for two groups: WT-Sham (3 hours of perfusion with no ischemia) and WT-IR (1 hour ischemia with 2 hours reperfusion). A third group consisted of A2B receptor knockout (KO) mice (1 hour ischemia with 2 hours reperfusion). At the end of reperfusion, lung function was assessed using an isolated buffer-perfused lung system. Lung inflammation was assessed by measuring pro-inflammatory cytokine levels (IL-6, KC, RANTES, MCP-1) in bronchoalveolar fluid and myeloperoxidase levels in lung tissue.

RESULTS: Compared to wild-type mice, A2B receptor KO mice showed significantly improved lung function after IR as evidenced by lower pulmonary artery pressures and increased lung compliance (Table 1). In addition, the A2B receptor KO mice showed decreased myeloperoxidase levels and reduced pro-inflammatory cytokine levels compared to wild-type mice after IR.

CONCLUSION: Absence of the A2B adenosine receptor provides significant protection against lung IR injury. A2B adenosine receptor contributes to IR lung injury by increasing pro-inflammatory cytokines and increasing neutrophil infiltration as measured by myeloperoxidase level. These results suggest that specific A2B adenosine receptor antagonists may serve as therapeutic agents to prevent IR injury after lung transplantation.

*AATS Member
### Table 1: Pulmonary Function and Cytokine Analysis

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<tr>
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<th>WT-Sham (n = 5)</th>
<th>WT-IR (n = 5)</th>
<th>A2BKO-IR (n = 5)</th>
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<tr>
<td><strong>Pulmonary Function</strong></td>
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<td>PAP (cm H2O)</td>
<td>5.5 ± 0.4</td>
<td>12.7 ± 1.1*</td>
<td>6.9 ± 0.6**</td>
</tr>
<tr>
<td>Compliance (μl/cm H2O)</td>
<td>5.4 ± 0.3</td>
<td>2.9 ± 0.2*</td>
<td>5.1 ± 0.2**</td>
</tr>
<tr>
<td>Myeloperoxidase (ng/ml)</td>
<td>88.6 ± 24.6</td>
<td>150.8 ± 23.9</td>
<td>113.4 ± 28.7</td>
</tr>
<tr>
<td><strong>Cytokine Levels (pg/ml)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IL-6</td>
<td>3851 ± 927</td>
<td>6756.1 ± 571.1*</td>
<td>4693.7 ± 410.8</td>
</tr>
<tr>
<td>KC</td>
<td>1104.6 ± 160</td>
<td>4749.1 ± 491*</td>
<td>2779.1 ± 505**</td>
</tr>
<tr>
<td>MCP-1</td>
<td>59 ± 5.8</td>
<td>272.1 ± 72*</td>
<td>64.3 ± 6.1**</td>
</tr>
<tr>
<td>RANTES</td>
<td>58.8 ± 9.3</td>
<td>198.4 ± 30.8*</td>
<td>44.1 ± 3.6**</td>
</tr>
<tr>
<td>TNF-α</td>
<td>21.8 ± 1.5</td>
<td>31.5 ± 2.9</td>
<td>22.7 ± 1.5</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SEM. PAP, pulmonary artery pressure.

*p < 0.05 vs. WT-Sham, **p < 0.05 vs. WT-IR.
L8. Effects of Nonselective COX and Selective COX-2 Inhibition on Collateral Development in the Heart

Michael P. Robich1, Louis M. Chu1, Jun Feng1, Cesario Bianchi1, Roger J. Laham2, Michael A. Coady3, Frank W. Sellke3*

1. Surgery, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA. 2. Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA. 3. Surgery, Warren Alpert Medical School, Brown University, Providence, RI, USA.

OBJECTIVE: Selective cyclooxygenase-2 (COX-2) inhibitors have been purported to increase the risk of myocardial infarction and death. We sought to explore the effects of nonselective cyclooxygenase (COX) and selective COX-2 inhibition on collateral development in a model of chronic myocardial ischemia. We hypothesized that COX-2 inhibitors will negatively effect angiogenesis and pro-angiogenic pathways.

METHODS: The hearts of Yorkshire swine were made chronically ischemic by placing an ameroid constrictor on the left circumflex coronary artery (LCx). Swine were divided into three groups and given no drug (control, n = 7), a nonselective COX inhibitor (naproxen 400 mg PO daily, NSAID, n = 7), or a selective COX-2 inhibitor (celecoxib 200 mg PO daily, COX-2, n = 7). After 7 weeks, coronary angiography was performed and images graded for TIMI flow scores of collateral formation. Myocardial function and perfusion and microvascular reactivity were assessed. Serum prostacyclin (PGI2) levels and immunoblotting for protein markers of angiogenesis were examined.

RESULTS: The COX-2 group demonstrated significantly increased mean arterial pressure (MAP) when compared to the control and NSAID groups. Animals in the COX-2 group had similar TIMI score vs. control, but the NSAID group had a trend toward increased TIMI flow. Myocardial perfusion in the COX-2 group was similar to that in the control, but less than that in the NSAID group. Coronary microvascular relaxation in the collateral dependent territory was diminished in the COX-2 group vs. NSAID or control groups. The COX group had a trend toward decreased serum PGI2. Protein expression of angiogenic markers showed decreased levels of VEGF and phospho-eNOS (ser1177) in both the COX-2 and NSAID groups, though only significantly in the COX-2 group. The NSAID group had decreased expression of endostatin, an anti-angiogenic protein (Table 1).
CONCLUSION: COX-2 inhibitors resulted in increased MAP and endothelial microvascular dysfunction, but similar collateral formation and myocardial perfusion vs. control. NSAID was associated with an increase in collateral formation and perfusion vs. control. Thus, selective COX-2 inhibition in this model is not associated with impairment in collateral formation, but is associated with decreased circulating levels of PGI2 and tissue levels of phospho-eNOS and VEGF.

5:00 p.m.   ADJOURN TO WELCOME RECEPTION

Exhibit Hall A & B

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>NSAID</th>
<th>COX-2</th>
<th>p value</th>
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<tr>
<td><strong>Functional Studies</strong></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Mean Arterial Pressure</td>
<td>55.3 ± 3</td>
<td>58 ± 3</td>
<td>84 ± 5</td>
<td>&lt;0.001</td>
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<td>+dP/dT (mmHg/sec)</td>
<td>954 ± 105</td>
<td>1065 ± 80</td>
<td>754 ± 87</td>
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<tr>
<td>TIMI Flow Score</td>
<td>0.3 ± 0.2</td>
<td>0.8 ± 0.5</td>
<td>0.2 ±0.2</td>
<td>0.23</td>
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<tr>
<td>LCx Blood Flow- Rest (mL/min/g)</td>
<td>0.12 ± 0.02</td>
<td>0.19 ± 0.03</td>
<td>0.12 ± 0.04</td>
<td>0.18</td>
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<tr>
<td>LCx Blood Flow- Pace (mL/min/g)</td>
<td>0.08 ± 0.02</td>
<td>0.15 ± 0.01</td>
<td>0.08 ± 0.01</td>
<td>0.006</td>
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<tr>
<td><strong>Microvessel Studies</strong></td>
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<tr>
<td>Sodium Nitroprusside (SNP)</td>
<td>94 ± 6</td>
<td>80 ± 8</td>
<td>75 ± 8</td>
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<tr>
<td>Adenosine Diphosphate (ADP)</td>
<td>73 ± 7</td>
<td>75 ± 6</td>
<td>55 ± 6</td>
<td>0.04</td>
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<td><strong>Serum Prostacyclin</strong></td>
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<td></td>
<td></td>
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<tr>
<td>PGI2 (pg/mL)</td>
<td>282 ± 81</td>
<td>105 ± 82</td>
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<td>0.19</td>
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<td><strong>Angiogenesis Markers</strong></td>
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<tr>
<td>(fold change)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>VEGF</td>
<td>3.4</td>
<td>2.5</td>
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<tr>
<td>phospho-eNOS (ser1177)</td>
<td>3.2</td>
<td>1.3</td>
<td>1</td>
<td>0.04</td>
</tr>
<tr>
<td>Endostatin</td>
<td>1.7</td>
<td>1</td>
<td>1.5</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Summary of results. PGI2- prostacyclin I2, VEGF- vascular endothelial growth factor, eNOS- endothelial nitric oxide synthase. Statistics performed using 1 way ANOVA with Newman-Keuls multiple comparison test (NSAID vs. COX-2)
7:30 a.m.  
**Business Session**  
(AATS Members Only)  
Hall C, Metro Toronto Convention Centre

7:45 a.m.  
**PLENARY SCIENTIFIC SESSION**  
Hall C, Metro Toronto Convention Centre  
(8 minutes presentation, 12 minutes discussion)

**Moderators:** G. Alec Patterson, MD  
Thoralf M. Sundt, III, MD

1. Randomized Trial of Mediastinal Lymph Node Sampling versus Complete Lymphadenectomy During Pulmonary Resection in Patients with N0 or N1 (Less than Hilar) Non-Small Cell Carcinoma: Results of the ACOSOG Z0030 Trial  
Gail E. Darling¹*, Mark S. Allen², Paul Decker³, Karla V. Ballman³,  
Rodney J. Landreneau⁴**, Robert J. McKenna⁵*, David R. Jones⁷*,  
Richard I. Inculet⁸, Valerie W. Rusch⁹*, Joe B. Putnam⁶*


**Invited Discussant:** Joseph B. Shrager, MD

**OBJECTIVE:** To evaluate whether mediastinal lymph node dissection (MLND) improves overall survival compared to mediastinal lymph node sampling (MLNS) in patients undergoing pulmonary resection for N0 or non-hilar N1, T1 or T2 NSCLC.

**METHODS:** Patients with proven NSCLC underwent sampling of lymph node stations 2R, 4R, 7, and 10R for right sided tumors; and 5, 6, 7 and 10L for left sided tumors. If these lymph node stations were negative for malignancy, patients were randomized to no further lymph node resection (MLNS) or complete MLND. All surgeons were required to adhere to
the technique described in written instructions and demonstrated in an approved instructional video. Following surgery, patients were followed for a minimum of 5 years.

RESULTS: A total of 1,111 patients were randomized (555 MLNS and 556 MLND). After final eligibility review, 1,023 (498 MLNS and 525 MLND) patients were classified as eligible/evaluable. There were no significant differences between the two groups in terms of gender, race, age or ECOG performance status. The right upper lobe was the most common tumor location (MLNS: 213 vs. MLND: 205) and adenocarcinoma was the most common histologic type in both arms (MLNS: 210 vs. MLND: 235). There was no significant difference between the two arms in terms of type or extent of resection, stage, length of stay, morbidity or mortality. In the MLND group 20 patients (3.8%) were found to have occult N2 disease in the lymphadenectomy specimen. At a median follow-up of 6.3 years, 431 (42.1%) patients have died: 214 (42.9%) in the MLNS arm and 217 (41.3%) in the MLND arm. The median survival was 8.1 years (MLNS) versus 8.5 (MLND) (p = 0.531). There were 493 recurrences including deaths: 54 local; 73 regional; and 224 distant. The median time to recurrence was 5.7 years in the MLNS group (243 recurrences; 24 local; 42 regional; and 110 distant) versus 6.1 years in the MLND group (250 recurrences; 30 local; 31 regional; and 114 distant) (p = 0.655). There also was no difference for local (p = 0.527) or regional recurrence (p = 0.126) between the two groups.

CONCLUSION: MLND does not improve survival in patients with early stage NSCLC when a thorough preresection sampling of the mediastinal lymph nodes is negative. MLND also does not decrease the incidence of local or distant recurrences. These results are not generalizable to higher stage tumors.
2. One Slide Fits All: The Versatility of Slide Tracheoplasty Utilizing Cardiopulmonary Bypass Support for Airway Reconstruction in Children

Peter B. Manning1*, Michael J. Rutter2, Asher Lisec3, Bradley S. Marino3

1. Cardiothoracic Surgery, Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA. 2. Otorhinolaryngology, Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA. 3. Cardiology, Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA.

Invited Discussant: Martin J. Elliott, MD

OBJECTIVE: Distal tracheal reconstruction in children is commonly associated with significant mortality and morbidity. Slide tracheoplasty with cardiopulmonary bypass (CPB) support uses all native tissue allowing growth potential and results in a more stable airway, facilitating early extubation. The purpose of this study is to describe our results with this approach and identify predictors for adverse outcomes.

METHODS: Patients undergoing slide tracheoplasty with CPB from 4/01–8/09 were reviewed. Preoperative characteristics, operative variables, and outcome measures [mortality, total hospital length of stay (LOS), and significant airway reintervention (>1 endoscopic dilation, stenting, and/or tracheal re-operation)] were collected. Predictors of worse outcomes were identified using bivariate analysis. Multivariable modeling was performed for predictors of prolonged LOS.

RESULTS: The cohort included 76 patients (median age 8.7 months, 7d-20 yrs); 71 (93%) had congenital tracheal lesions and 19 (25%) had prior tracheal operations. Length of tracheal pathology was <50%, 50–80%, and >80% of the total tracheal length in 22, 35, and 19 patients respectively. 45 patients had associated cardiac or great vessel anomalies; 23 patients had simultaneous repair of a cardiovascular anomaly at the time of tracheal reconstruction. Median CPB time was 95 min (range 49–318) for the entire group and 86 min for tracheoplasty only. 47 (62%) patients were extubated within 48 hours of their operation. Median LOS was 19 days (range 7–119). 21 patients (28%) required significant airway reintervention during a median follow-up duration of 12 months (range 1 m–7.8 yrs). There were 4 deaths; 2 early and 2 late. In bivariate analysis, age (p = .015) and CPB duration (p = .029) were predictors of mortality, while duration of postoperative mechanical ventilation was associated with need for significant airway reintervention (p = .015). Multivariable analysis showed...
that preoperative ventilatory support (p < .001), longer CPB duration (p = .003), and the need for significant airway reintervention (p = .001) were predictors of longer LOS.

**CONCLUSION:** Slide tracheoplasty with CPB may be performed with low mortality in a diverse population of pediatric patients including those with full-length congenital stenosis, acquired lesions, and reoperative tracheoplasty. Utilizing this technique minimizes the need for early significant airway reintervention in the majority of patients.
3. Transapical Aortic Valve Implantation at Three Years

Thomas Walther, Joerg Kempfert, Michael A. Borger*, Ardawan J. Rastan, Axel Linke, Gerhard Schuler, Friedrich W. Mohr*
Cardiac Surgery, Heartcenter Leipzig, Leipzig, Germany.

Invited Discussant: Joseph E. Bavaria, MD

OBJECTIVE: Transapical (TA) aortic valve implantation (AVI) has been introduced into clinical practice in 2006 to treat high risk patients with symptomatic aortic stenosis. The aim of this study was to evaluate the results of minimally invasive TA-AVI at three years.

METHODS: From February 2006 until October 2009 a total of 267 high risk patients with symptomatic aortic stenosis received TA-AVI using the Edwards SAPIEN™ transcatheter xenograft. Patient age was 82 ±6 years, 70% were female. Logistic EuroSCORE was 32 ±16% and STS score 12 ±7%. All patients referred that presented with an aortic annulus diameter ≤24 mm were included into this study. Initially ten patients were treated on-pump, then an off-pump protocol with safety net femoral wires to allow for potential conversion was used. All procedures were performed in a hybrid operative theatre.

RESULTS: Transapical access was feasible in all patients. TA-AVI was performed completely off-pump in 90% of the patients, 6.6% had to be converted on-pump. Five patients required conversion to sternotomy, three of them survived. Survival at 30 days was 90%, at one year 70%, at two years 68% and at three years 56%. There were two minor strokes on POD 1 and 2, both with good functional recovery. One patient presented with endocarditis at three weeks post AVI, after conversion she lateron died due to sepsis. Systematic evaluation revealed that patients reached a good quality of life at three months after TA-AVI, quite comparable to an age matched control population.

CONCLUSION: TA-AVI can be performed with good outcome in high risk patients with aortic stenosis. Optimal imaging and a team approach are essential for success.
4. Superior Nationwide Outcomes of Thoracic Endovascular Aneurysm Repair Compared to Open Repair for Isolated Descending Thoracic Aneurysm in a Cohort of 11,000 Patients

Raja R. Gopaldas1,2, Joseph Huh1,2, Tam K. Dao1, Scott A. LeMaire1,2*, Danny Chu1,4, Faisal G. Bakaeen1,4, Joseph Coselli1,2*
1. Division of Cardiothoracic Surgery, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX, USA. 2. Texas Heart Institute at St. Luke’s Episcopal Hospital, Houston, TX, USA. 3. Department of Education Psychology, University of Houston, Houston, TX, USA. 4. Michael E. DeBakey Veterans Affairs Medical Center, Houston, TX, USA.

Invited Discussant: John A. Kern, MD

OBJECTIVE: Thoracic endovascular aneurysm repair (TEVAR) was introduced for the treatment of descending thoracic aortic aneurysm (DTAA) in 2005. Little is known about TEVAR’s nationwide impact on patient outcomes. We evaluated the nationwide estimates of short-term outcomes of TEVAR and open DTAA procedures performed in the US during a 2-year period.

METHODS: From the weighted Nationwide Inpatient Sample databases, we identified patients who underwent surgery for isolated DTAA in 2006–2007. Patients with vasculitis, connective tissue disorders, or concomitant aneurysms in other aortic segments were excluded. Of the remaining 11,669 patients, 9106 underwent conventional open aortic repair (OAR) and 2563 underwent TEVAR. Hierarchic logistic and multivariable regression were used to assess the effect of TEVAR vs. OAR after adjusting for potential confounding factors. Primary outcomes were mortality and length of stay (LOS); secondary outcomes were disposition, morbidity, and hospital charges.

RESULTS: Patients who underwent TEVAR were older (69.5 ± 12.7 y vs. 60.2 ± 14.2 y; P < 0.001) and had higher Charlson-Deyo comorbidity scores (4.6 ± 1.8 vs. 3.3 ± 1.8; P < 0.001). Unadjusted LOS was shorter for TEVAR patients (7.7 ± 11 d vs. 8.8 ± 7.9 d), but unadjusted mortality was similar (TEVAR 2.3%, n = 59; vs. OAR 2.3%, n = 209; P = 1.0). The proportion of non-elective interventions was similar between groups (TEVAR 15.9%, n = 405; vs. OAR 15.8%, n = 1446; P = 0.9). After risk adjustment, TEVAR and OAR produced similar mortality rates, but TEVAR patients had a lower overall complication rate and a shorter LOS (by 1.3 days); however, hospital charges were higher by $6713/patient (95% CI $1869–$11,556; P < 0.001).
TEVAR patients were 4 times more likely to have a routine discharge to home, and they had fewer intra-operative, neurologic, and respiratory complications, but no differences in the incidence of pulmonary embolism ($P = 0.5$). Overall, TEVAR was associated with a 60% lower risk of any complication than OAR (OR = 0.39; $P < 0.001$).

**CONCLUSION:** The nationwide data on TEVAR for DTAA associate this procedure with better short-term outcomes than OAR, even though TEVAR is selectively performed in patients who are almost a decade older than OAR patients. TEVAR is associated with a shorter hospital LOS and 1 fewer complication for every 3 patients, but significantly higher, hospital charges. Future studies should assess the long-term success of TEVAR vs. the gold standard OAR.

<table>
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<tr>
<th>Parameter</th>
<th>Odds Ratio/B</th>
<th>Sig</th>
<th>95% CI Lower Bound</th>
<th>95% CI Upper Bound</th>
<th>$R^2$ Square</th>
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<tbody>
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<td>Intra-Operative complications</td>
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<td>&lt;.001</td>
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<td>Infections</td>
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<td>Renal complications</td>
<td>0.87</td>
<td>0.314</td>
<td>0.67</td>
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<td>Respiratory complications</td>
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<td>&lt;0.001</td>
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<td>Pulmonary embolism</td>
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<tr>
<td>Any complication</td>
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<td>&lt;0.001</td>
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<td>Total complications</td>
<td>−0.334/patient</td>
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<td>Died during hospitalization</td>
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<td>0.879</td>
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<td>Length of stay</td>
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<td>−0.8</td>
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<td>Routine home discharge</td>
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<td>Cost in 2009 dollars</td>
<td>56713.09</td>
<td>&lt;0.01</td>
<td>1869.52</td>
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10:00 a.m.  BASIC SCIENCE LECTURE

“Nerve Injury – Changing the Surgical Paradigm through Translational Science”
Susan E. Mackinnon, MD
Washington University School of Medicine

Introduced By:  G. Alec Patterson, MD

10:40 a.m.  PLENARY SCIENTIFIC SESSION

Moderators:  Irving L. Kron, MD
Thoralf M. Sundt, III, MD

5. Identifying Patients at Particular Risk of Injury at Repeat Sternotomy: Analysis of Over 2500 Cardiac Reoperations
Chan B. Park, Rakesh M. Suri, Harold M. Burkhart, Kevin L. Greason, Joseph A. Dearani, Hartzell V. Schaff*, Thoralf M. Sundt*
Mayo Clinic, Rochester, MN, USA.

Invited Discussant:  Irving L. Kron, MD

OBJECTIVE:  Reoperative sternotomy is an increasingly common clinical challenge. A variety of protective strategies have been proposed, however it remains unclear in whom such strategies are warranted.

METHODS:  Using our STS database, we identified adults undergoing repeat median sternotomy for routine cardiac surgery between 1/1/1996 and 12/31/2007 excluding transplants, ventricular assist device implants, and procedures for congenital defect. All operative notes and perioperative outcomes were reviewed.

RESULTS:  Of 2555 patients, 1537 (60%) had undergone prior CABG, 700 (27%) mitral valve surgery (MVR), and 643 (25%) aortic valve surgery (AVR); 61 patients (2%) had prior mediastinal radiation, and 424 (17%) >1 prior sternotomy. There were 267 injuries in 231 (9.0%) patients. Of 87 (33%) injuries during sternal re-entry, 21 (24%) were to saphenous vein bypass grafts (SVG) or ITA, 20 (23%) to the innominate vein, 17 (20%) to the right ventricle (RV), and 17 (20%) to the aorta. Injury was more common, however, during pre-pump dissection (n = 135), of which 29 (21%) were to SVG, 28 (21%) to ITA, 15 (11%) to the innominate vein and 15 (11%) to the aorta. Patients with injury had a higher hospital mortality (18.6% vs. 6.5%, p < 0.001), particularly when the injury occurred during sternal re-entry (2.4% survivors vs. 9.8% mortalities, p < 0.001) compared

*AATS Member
with injury during dissection (4.3% vs. 10.3%, p < 0.001), however only injury to the RV was a predictor of hospital death (p < 0.001). The mortality rate associated with injury to the RV was 35% (11/31), aorta 21% (7/34), innominate vein 18% (7/38), SVG 18% (8/45) and ITA 17% (7/41). Injuries were more common after prior CABG (70% with injury vs. 59% without, p = 0.0012) but not prior AVR, MVR or aortic surgery. Injury was also more common when the current operation was AVR (51% with injury vs. 44% without, p = 0.04) or aortic surgery (16% vs. 10%, p = 0.004). By multivariate analysis, however, only prior radiation (OR 4.3), and increased number of prior median sternotomies (OR1.6) were risk factors for injury. Institution of CPB prior to sternotomy was used in 40 patients did not assure prevention of injury (p = 0.44).

CONCLUSION: Patients with prior radiation and >1 prior sternotomy are at increased risk of complications during sternal re-entry and accordingly warrant consideration for protective strategies, as may patients with prior CABG. Given the association between RV injury and death, strategies to prevent this complication are particularly important.
6. The Effect of Regionalization on Outcome in Pulmonary Lobectomy: A Canadian National Study

Christian J. Finley¹, Shaf Keshavjee¹*, David R. Urbach², Anna Bendzsak¹, George Tomlinson², Gail E. Darling¹*

¹Division of Thoracic Surgery, Toronto General Hospital, Toronto, ON, Canada.
²Division of Clinical Decision Making and Health Care, Toronto General Hospital, Toronto, ON, Canada.

Invited Discussant: Yolonda L. Colson, MD, PhD

OBJECTIVE: To examine the relationship between changing hospital volume and in-hospital mortality (IHM) and length of stay (LOS) after lobectomy.

METHODS: Lobectomy patients in the Canadian Institute for Health Information Discharge Abstract Database from 1998–2007 were included. Random effects logistic regression was used for the IHM analysis and a random effects linear regression was used for the log-transformed LOS analysis. The changes in IHM and LOS over time were examined using a hierarchical model. A cross-sectional analysis of hospital volume and IHM and LOS was then done controlling for clustering. A model controlling for Charlson comorbidity index, other confounders, and clustering was used to estimate the effects of within-hospital changes in annual lobectomy volume and its association with outcome. The proportion of cases done in high-volume centres over time was then tabulated and displayed graphically.

RESULTS: The study included 22,915 patients; 12147 (53%) males, and the average (Standard Deviation) age was 62.4 (14.8) years. There was a 32% (95% confidence interval (CI) 7–50%; p = 0.014) relative risk reduction in IHM over the study period and a 21% reduction in LOS (95% CI: 18–23; p < 0.001). Lower IHM and LOS were seen in higher-volume centres in the cross-sectional analysis with a 31% (95% CI 19–41%, p < 0.001) relative risk reduction for IHM and 10% relative decrease (95% CI 5–15%; p < 0.001) in LOS for every 50 additional cases performed.

The multivariate model found no significant effect of volume changes within-hospital on mortality, a 12% relative increase (95% CI: 60–21%; p = 0.52) in mortality for each additional 50 cases. For each increase in volume of 50 cases, there was a 9% relative decrease in LOS (95% CI: 4–15%; p < 0.001).

The change in percentage of patients treated in differing centre volumes is shown in the following Figure.

*AATS Member
CONCLUSION: IHM and LOS for lobectomies have decreased in Canada, with high-volume centres having improved overall outcomes. However, there was no reduction in mortality within-hospitals where volumes increased. A decrease in LOS did occur in hospitals where volume increased. The improved overall mortality over time is likely related to a increasing number of patients being treated in high-volume centres, rather than improvements within individual centres.

11:25 a.m.  
**PRESIDENTIAL ADDRESS**

“Non Solus – A Leadership Challenge”
G. Alec Patterson, MD
Washington University School of Medicine

*Introduced By:* Irving L. Kron, MD

12:15 p.m.  
**ADJOURN FOR LUNCH – VISIT EXHIBITS**

*Exhibit Hall A & B*
7. Hemodynamic Results at One Month and Over One Year of Endoventricular Patch Plasty in 117 Ischemic Failing Ventricles Excluded from the STICH Trial

Vincent Dor*, Filippo Civaia, Clara Alexandrescu, Michel Sabatier, Françoise Montiglio
Centre Cardio-Thoracique, Monaco Cedex, Monaco.

Invited Discussant: Robert H. Jones, MD

OBJECTIVE: To demonstrate early and late efficiency of left ventricular reconstruction (LVR) by endoventricular patch plasty for post myocardial infarct (MI) end stage ischemic failing ventricle (IFV) in acute or chronic grade IV, or advanced heart failure (HF) beyond drugs (and/or devices) and revascularization therapies.

METHODS: From 2002 to May 2008, among 274 LVR for post MI scarred ventricles, 117 IFV, mean age 64 (34–83), were responding to criteria of exclusion from Stich Trial:

- 12 no coronary vessel suitable for CABG
- 17 first MI month, including 11 acute HF:8 septal ruptures, 3 ventricular tachycardia (VT)
- 48 IV inotropes and/or intra aortic balloon pumping (IABP)
- 15 bifocal or posterior scar
- 4 heart transplantation scheduled
- 21 for 5 other criteria (see article)
Patients had pre operative assessment by MRI of percentage of scarred asynergic LV circumference, mean 49% (35–75). Systolic and diastolic functions of LV are analysed by LVEF and LV and left atrial volume indexes. Mitral regurgitation mild to severe in 65 patients is related in 61 to annulus dilatation (≥35 mm). Pulmonary pressure is assessed by echo and right heart catheterisation (+ programmed ventricular stimulation: 39 spontaneous/inducible VT). 36 patients with previous implantable defibrillator or pace maker or IAPB parameters were evaluated by cine angio, CT scan or echo.

LVR by endoventricular suture and patch excludes asynergic LV wall (cause of HF) restores curvature of contractile myocardium (suppressing eccentric motion mechanism of dilatation) and keeps up physiological diastolic volume (50ml/m²) with balloon sizing. Feasibility is evaluated by the contractile area diastolic volume index calculated on gadolinium late enhancement map which also helps the guiding of the endoventricular suture (contractility trail). Patch is circular for antero septo apical lesion and triangular for posterior localisations. Mitral valve was repaired in 64 patients, 82 had endocardectomy with cryotherapy in 41 for VT, coronary revascularization in 107 (arterial in 95, mixed in 12, patent stents on LAD or thrombosed LAD after endarterectomy being bypassed by safety).

RESULTS: 4 hospital and 2 delayed deaths occurred during the first year. 111 patients had hemodynamic control after 1 month and 1 year by MRI (104) or CT Scan (7) showing evident progressive remodelling reversion (Table ).

| Table 1: Reverse Remodeling in 111 IFV |
|------------------|-----------------|-----------------|-----------------|-----------------|
| Advanced HF n = 101 | EF (%) | EDVol (ml/m²) | ESVol (ml/m²) | LAVol (ml/m²) |
| Pre op | 26 ± 4 (9–34)* | 130 ± 43 (62–342)* | 95 ± 37 (45–289)* | 53 ± 22 (17–94)* |
| >1 month | 40 ± 8 (21–64)* | 84 ± 2 1 (46–170)** | 51 ± 17 (24–118)^ | 40 ± 14 (13–72) |
| >1 year | 44 ± 11 (20–69) | 85 ± 30 (33–217) | 48 ± 20 (15–128) | |
| Acute HF n = 10 | EF (%) | EDVol (ml/m²) | ESVol (ml/m²) | PAP's (mmHg) |
| Pre op | 41 (32–50) | 79 (54–122) | 46 (31–83) | 58.5 (38–85) |
| >1 month | 52 (38–68) | 48 (28–79) | 24 (16–49) | 34.6 (30–49) |
| >1 year | 54 (38–72) | 69 (32–105) | 33 (9–66) | |

*p < 0.001, **p = 0.44, ^p = 0.25

CONCLUSION: LVR, being an etiological surgical treatment, can reverse durably and stably severe ischemic HF.
8. Endoscopic Vein Harvest Is Associated with Compromised Patency Outcomes: Results from the Prospective Randomized ROOBY Trial

Marco A. Zenati1*, Ali F. Sonel2, A. Laurie Shroyer3, Morteza Amidi2, Joseph Collins4, G. Hossein Almassi5*, ROOBY Study3,6

1. Cardiac Surgery, University of Pittsburgh, Pittsburgh, PA, USA. 2. Cardiology, VA Pittsburgh, Pittsburgh, PA, USA. 3. VA Northport, Northport, NY, USA. 4. CSP Data Coordinating Center, Perry Point, MD, USA. 5. Medical College of Wisconsin, Milwaukee, WI, USA. 6. ECHS Denver VAMC, Denver, CO, USA.

Invited Discussant: Carlos A. Mestres, MD, PhD

OBJECTIVE: In the controlled, single-blinded, Randomized On/Off Bypass (ROOBY) Trial, 2,203 patients were randomized to on-pump vs. off-pump CABG. The saphenous vein graft harvesting technique (endoscopic or open) was recorded. We describe the patency outcomes of bypass graft according to the harvesting technique.

METHODS: Of the 2,127 patients that survived 12 months, 1,371 (64.4%) had one year control coronary catheterization. Most of these patients (1,335) had catheterization at the 12 month end of study visit. Grafts were evaluated for patency (open vs. closed) and assigned a Fitzgibbon grade of A (excellent graft), B (impaired graft with stenosis >50%) or O (occluded). Overall, 4,093 grafts were analyzed including 2,601 saphenous vein grafts.

RESULTS: All cause 30-day mortality was low in both off-pump (1.6%) and on-pump (1.2%) groups. The number of grafts was 3.0 ± 0.9 grafts/patient. 912 patients enrolled and randomized in ROOBY with recorded harvesting technique had 12 month follow up including control angiography. In 61.8% (564/912) the SVG harvesting technique was open and in the remaining 38.2% it was endoscopic. The LITA-LAD patency was 95.3% in the off-pump group and 96.2% in the on-pump group (p = NS). The percentage of patients will "all grafts open" at 12 months when at least one of the grafts was a SVG was 75.9% for the open SVG harvest (420/553) and 61.3% (209/341) for endoscopic harvest (p < 0.05).

CONCLUSION: We conclude that the 12 month graft patency of SVG harvested using the endoscopic technique was lower compared to open technique. Further analysis is warranted to determine the relationship with other variables.

*AATS Member
9. Impact of Pulmonary Hypertension on Outcomes following Aortic Valve Replacement for Aortic Valve Stenosis

Spencer J. Melby, Marci Bailey, Marc R. Moon*, Nader Moazami, Jennifer S. Lawton*, Brian R. Lindman, Ralph J. Damiano*
Cardiothoracic Surgery, Washington University School of Medicine, St. Louis, MO, USA.

Invited Discussant: James S. Gammie, MD

OBJECTIVE: The presence of chronic pulmonary hypertension (CPH) historically has been considered a significant risk factor affecting early and late outcomes following valve replacement. However, few studies have rigorously looked at its effect on outcome. Moreover, there have been a number of recent advances in the management of pulmonary hypertension and right heart failure following cardiac surgery. The purpose of this study was to determine if pulmonary hypertension remains a risk factor in the modern era in outcomes following aortic valve replacement (AVR) for aortic valve stenosis.

METHODS: From January 1996 to June 2009, 1,080 patients underwent AVR for primary aortic valve stenosis, of which 574 (53%) had normal systolic pulmonary artery pressures (sPAP) and 506 (47%) had CPH. CPH was defined as mild (sPAP 35–44 mmHg), moderate (45–59 mmHg), or severe (≥60 mmHg). In the group of patients with CPH, 204 had postoperative echocardiograms.

RESULTS: Operative mortality was significantly higher in patients with CPH (47/506, 9% vs. 31/574, 5%, p = 0.02). The incidence of postoperative stroke was similar (p = 0.14), but patients with CPH had an increased median hospital LOS (8 vs. 7 days, p = 0.001) and an increased incidence of prolonged ventilation (26% vs. 17%; p < 0.001). In the mild CPH group, sPAP did not fall following AVR (39 ± 3 vs. 37 ± 11 mmHg, p = 0.2). In contrast, sPAP fell after AVR in patients with moderate CPH (51 ± 4 vs. 45 ± 16, p = 0.01) and severe CPH (69 ± 12 vs. 45 ± 14, p < 0.001). Five-year survival (Kaplan-Meier) was 78 ± 6% with normal sPAP and 77 ± 7% with mild CPH postoperatively, compared to 64 ± 8% with moderate CPH and 45 ± 12% with severe CPH (p < 0.001, Figure).

*AATS Member
CONCLUSION: In patients undergoing AVR for aortic valve stenosis, CPH increased operative mortality, and patients with persistent moderate or severe CPH after AVR had decreased long-term survival. The presence of CPH does not currently factor into STS risk scores. This data suggests that CPH has significant impact on outcomes in patients undergoing AVR and should be considered in preoperative assessment.

3:00 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B
10. A Randomized Trial of a Restrictive versus Liberal Blood Transfusion Strategy in Older Postoperative Cardiac Surgery Patients

Robin Varghese¹, M. Lee Myers¹, L. Ray Guo¹, Neville Suskin²
2. Cardiology London Health Sciences Centre, London, ON, Canada.

Invited Discussant: Jennifer S. Lawton, MD

OBJECTIVE: Does a liberal transfusion strategy improve quality of life, functional status, and clinical outcome in older postoperative cardiac surgery patients?

METHODS: In this single-blinded trial, cardiac surgery patients over 70 years with an initial postoperative hemoglobin (Hgb) of 70–90 g/L were randomly assigned to receive blood based on a liberal (transfusion if Hgb < 100g/L) or restrictive (transfusion if Hgb < 70g/L) transfusion strategy. Patients otherwise received routine postoperative care. Exercise capacity was measured by performing a 6-minute walk distance test (6MWT) on postoperative day (POD) 5 when possible, and at the 6-week mark. Quality of life was measured using the 36-item short form health survey (SF-36). Secondary outcomes with respect to postoperative clinical course were also evaluated.

RESULTS: A total of 73 patients were analyzed and the groups were well matched at baseline. The mean age was 77.3 in the liberal arm and 75.1 in the restrictive arm. Although there was no difference in the mean initial postoperative Hgb (82g/L vs. 84g/L; p = 0.28), the levels in the liberal group remained significantly higher throughout the study period: POD1 104 vs. 86; POD2 109 vs. 85; 6 weeks 126 vs. 115 (p < 0.0001 for all comparisons). The mean number of red cell units transfused after randomization was significantly different (2.6 vs. 0.28 units; p < 0.0001). A comparison of exercise capacity measured by the 6MWT revealed no significant differences in distance walked between the liberal and restrictive groups at 6-weeks follow-up (359 m vs. 341m; p = 0.48). There were no differences in the incidence of postoperative complications (p = 0.86).
Median post-operative hospital length of stay was 6.0 days in both groups. Quality of life measured at six weeks using the SF-36 questionnaire revealed no significant difference between groups in the patients’ perceived functional status.

CONCLUSION: The use of a restrictive postoperative transfusion strategy in older patients following cardiac surgery has no deleterious effect on postoperative length of stay, complication rate or functional capacity at 6-weeks postoperatively. A relatively restrictive transfusion strategy appears safe and generally appropriate in this population of cardiac surgery patients.
11. Robotic Mitral Valve Repair versus Conventional Approaches: Potential Realized

Tomislav Mihaljevic, Craig Jarrett, A. Marc Gillinov*, Sarah Williams, Pierre Devilliers, Eugene H. Blackstone*

Cleveland Clinic Foundation, Cleveland, OH, USA.

Invited Discussant: Volkmar Falk, MD

OBJECTIVE: Robotic mitral valve (MV) repair provides the least invasive surgical approach for treating myxomatous mitral valve disease, yet there are few data comparing its outcomes to those of conventional approaches. Therefore, we sought to compare outcomes of robotic (ROB) MV repair to those using complete sternotomy (CST), partial sternotomy (PST), and mini-anterolateral thoracotomy (MIN) approaches.

METHODS: From 1/2006 to 1/2009, 743 patients with degenerative MV disease and posterior leaflet prolapse underwent primary isolated MV surgery by ROB (n = 253), CST (n = 113), PST (n = 263), or MIN (n = 114) approaches. Outcomes were compared on an intent-to-treat basis using a propensity score study design based on 60 preoperative factors to obtain well-matched patient pairs.

RESULTS: Mitral valve repair was achieved in all patients except for one in the CST group. Among matched patients, ROB patients had the longest operative times (median 389 min), 117 min, 107 min, and 60 min longer than CST (P < .0001), PST (P < .0001), and MIN (P < .0001), respectively. There were no in-hospital deaths. Occurrences of STS-defined neurologic, pulmonary, renal, vascular, and infectious complications were similar (P > .07). ROB patients had the shortest postoperative hospital stays (median 4.2 days), 0.9 days, 1.2 days, and 0.8 days shorter than CST (P < .0001), PST (P < .0001), and MIN (P = .001), respectively. Effectiveness of MV repair was similar among groups (P = .4).

CONCLUSION: Robotic MV repair is as safe and effective as repair using conventional approaches. Technical complexity of the robotic approach, with resulting increase in operative times, is compensated for by lesser invasiveness and shorter postoperative stay.

*AATS Member
OBJECTIVE: Risk-stratifying algorithms are currently used to determine which patients may be too high-risk for surgery, and thus candidates for percutaneous/transapical aortic valve replacement (AVR). Yet, the utilization of minimally invasive surgical approaches has been successful in reducing morbidity and improving survival following AVR. We sought to describe the outcome following minimally invasive aortic valve replacement (MiniAVR) in high-risk octogenarians who may be candidates for percutaneous/transapical AVR.

METHODS: From 1996–2009 MiniAVR was performed in 249 octogenarians. We utilized the Modified Euroscore and STS score to risk-stratify patients.

RESULTS: As shown in Table 1, mean age at operation was 84 ± 3 years and 21% (n = 52) had previous cardiac surgery. Operative mortality was 3% (n = 8/249). The Modified Euroscore (14 ± 13%) and STS Score (17 ± 13%) were not predictive of 30-day mortality in this cohort of patients (Euroscore p = 0.96, STS score p = 0.15). Despite their poor predictive power, the STS score and Euroscore were strongly correlated with each other (F = 58.03, p < 0.0001). Postoperative complications included stroke in 10 (4%), pneumonia in 3 (1%), renal failure requiring dialysis in 2 (1%), cardiac arrest in 2 (1%), PE in 1 (1%), and sepsis in 1 (1%). Follow-up was available for 238 (96%) patients and extended up to 12 years. Overall, long-term survival after MiniAVR at 1, 5, and 10 years was 93%, 77%, and 56%, respectively (Figure 1a). There was no significant difference in long-term survival compared to that of a US age and gender matched population (Figure 1a, standardized mortality ratio 1.01, 95% CI: 0.76, 1.37, p = 0.88). A multivariate Cox-proportional hazards model indicated that increasing age (HR 1.10, p = 0.008) and severe COPD (HR 2.52, p < 0.007) to be significant predictors of survival. Using these factors, a clinical prediction model (p = 0.02) was developed (Figure 1b) and demonstrated that low-risk patients (1st quartile prediction score) had 1, 5, and 8 year survival of 94%, 84%, and 67% while high-risk patients (3rd quartile prediction score) had 1, 5, and 8 year survival of 89%, 74%, and 49%, respectively.
CONCLUSION: Patients thought to be high-risk candidates for surgical AVR have excellent outcomes after minimally invasive surgery with long-term survival that is no different than that of an age and gender matched US population. These data provide a benchmark against which outcomes of percutaneous/transapical aortic valve replacements should be compared.
13. The Cox-Maze IV Procedure: Predictors of Late Recurrence

Ralph J. Damiano*, Marci Bailey, Forrest H. Schwartz, Nabil A. Munfakh, Jennifer S. Lawton*, Richard B. Schuessler, Marc R. Moon*
Cardiothoracic Surgery, Washington University School of Medicine, St. Louis, MO, USA.

Invited Discussant: Michael Argenziano, MD

OBJECTIVE: The Cox-Maze procedure (CMP) was introduced for the surgical treatment of atrial fibrillation (AF) in 1987. The final iteration, the CMP III, achieved high cure rates and became the surgical gold standard. Due to its invasiveness, a more simplified ablation-assisted procedure (CMP IV) has been performed since January, 2002. This report describes a large, prospectively-collected, single institution experience for the treatment of atrial fibrillation. The study examined multiple preoperative variables to determine predictors of late recurrence.

METHODS: Data were collected prospectively on 264 patients (mean age 63 ± 12 years) who underwent CMP IV for atrial fibrillation from January 2002 through August 2009. Forty-one percent of patients had paroxysmal and 59% had either persistent or long-standing persistent atrial fibrillation. The mean AF duration was 6.5 ± 7.7 (range- 0.1–46.0) years. All data were entered prospectively into a database containing 386 variables. All patients were available for follow-up. Follow-up included ECGs in all patients. Since 2006, prolonged monitoring was obtained in 92% of patients at 3, 6 and 12 months. Data were analyzed by logistic regression analysis at 12 months with preoperative variables, including age, gender, left atrial (LA) diameter, NYHA class, AF type and duration, used as co-variants. All patients with reported outcomes data had a minimum of 3 months follow-up.

RESULTS: Sixty-six percent of patients had concomitant procedure, while the rest had lone AF. Following an ablation-assisted CMP, the freedom from AF was 90%, 92%, and 90% at 3, 6, and 12 months respectively. The freedom from both arrhythmias and antiarrhythmic drugs was 62%, 77%, and 78% at 3, 6, and 12 months. There was no difference in late success rate for patients with paroxysmal versus persistent or long-standing AF (p = 0.233). There was also no difference in success rates for patients with lone versus concomitant atrial fibrillation (p = 0.485). The only risk factor for AF recurrence at one year was enlarged LA diameter (p = 0.003).

*AATS Member
CONCLUSION: The CMP IV, which replaces most of the surgical incisions with linear lines of ablation, maintains the success rate of the CMP III. The only predictor of AF recurrence was enlarged LA diameter. This occurred despite aggressive attempts at LA size reduction and may suggest the need for a more extensive or different procedure in these patients.

5:00 p.m.        ADJOURN
OBJECTIVE: In the last 10 years English literature, the Heller-Dor operation is by far the technique more often adopted for the cure of esophageal achalasia. Quality of outcomes is sometimes different, possibly for technical reasons. Aim of the study is to analyze the details of myotomy and fundusplation in relation to the cure of dysphagia (D) and the occurrence of postoperative reflux esophagitis (RE) achieved in a 30 years single center experience.

METHODS: The period January 1979–December 2008 was considered in which the same technique was performed by five staff surgeons and several residents. Intraoperative manometry was used in 100% of the 262 patients in order to: 1) abolish the high pressure zone (HPZ) with a long esophago-gastric myotomy 2) protect the surface of the myotomy with a long but soft anterior fundusplation (6–8 sutures each side of the myotomy, trimmed to avoid RE without impairing the esophageal emptying). 202 patients (97 men, median age 55.5 yrs. r. 7–94) were operated on by laparotomy and 60 (24 men, median age 56 yrs. r. 16–80) by laparoscopy. Follow-up consisted of clinical interview, endoscopy, barium-swallow at given intervals and manometry if required. A semiquantitative scale graded results (Table 1).
RESULTS: Mortality was 1/202 in the laparotomy group (severe portal hypertension in congenital cardiopathy) and 0/60 in the laparoscopy group with 3 conversions. All patients had follow-up; laparotomy group median 96 months r. 12–324, laparoscopy group median 48 months r. 6–161.

At intraoperative manometry, myotomy achieved the complete abolition of HPZ in 100%; the Dor related HPZ length and mean pressure were 4.5 (±0.4) cm and 13.3 (±2.2) mmHg in laparotomy and 4.5 (±0.5) cm and 13.2 (±2.2) mmHg in laparoscopy (p = 0.75). Outcome is summarized in Table 1. In laparotomy poor results (19/201 9.5%) were secondary to RE in 15/201 (7.5%), in 2 RE was diagnosed after 184 and 252 months and to recurrent D in 4/201 (2%) all with end stage sigmoid achalasia. In laparoscopy 2/60 (3.3%) had RE and none recurrent D.

CONCLUSION: A long esophago-gastric myotomy protected by Dor funduspslication performed with reference to manometric parameters is the optimal cure for D and efficiently controls postoperative RE. Although the follow up is shorter the literature data on the timing of postoperative D and RE allow to conclude that the laparoscopic technique is as efficient as the laparotomic one. Intraoperative manometry is probably the key factor for achieving the reported results.
15. The Impact of Adjuvant Brachytherapy with Sublobar Resection on Pulmonary Function and Dyspnea; Preliminary Results from ACOSOG Z4032 Trial

Hiran C. Fernando1*, Rodney J. Landreneau2*, Sumithra Mandrekar3, Shauna Hillman4, Francis C. Nichols4, Bryan Meyers5*, Thomas Dipetrillo6, Dwight E. Heron3, Joe B. Putnam7*


Invited Discussant: Walter Weder, MD

OBJECTIVE: ACOSOG Z4032 is a prospective randomized clinical trial, comparing sublobar resection with intraoperative brachytherapy (SRB) to sublobar resection (SR) alone in high-risk patients with stage I lung non-small cell cancer (NSCLC) 3 cm or less. This report examines the impact of brachytherapy on pulmonary function tests (PFT) and dyspnea.

METHODS: All eligible patients had either a wedge or segmental resection. For patients randomized to SRB, following resection, Iodine 125 seeds were implanted at the resection margin. Dyspnea scores (using the UC San Diego Shortness of Breath Questionnaire) and pulmonary function, specifically DLCO% and FEV1 (ml) were assessed at baseline, and at months 3, 12 and 24 (M3, M12, and M24). The dyspnea and spirometry values at baseline, 3 and 12 months were compared between the arms using a Wilcox on rank sum test. Additionally, a 10 point change in dyspnea score, a 10% change in DLCO%, and a 200ml change in FEV1 were deemed “clinically meaningful” and compared between the arms using the Fisher’s exact test.

RESULTS: As of October, 2009, 216 /226 patients have been enrolled. No differences were seen between treatment groups in baseline characteristics (age, performance, stage), rate of thoracotomy or tumor location, and baseline PFTs and dyspnea scores. At M3, the median FEV1 and DLCO% were significantly better in the SR group (see Table). No difference was seen at M12. Compared to baseline values, there was no “clinically meaningful” significant change in PFT or dyspnea score between arms at M3 or M12.

*AATS Member
CONCLUSION: The addition of brachytherapy to SR may transiently impair pulmonary function at 3-months, but not at 12 months. There was no clinically significant change from baseline on either PFT or dyspnea score at any time point, demonstrating that the use of SRB is reasonable for patients with impaired pulmonary function in a multicenter setting.

Table 1: Study was Supported by Grant #U10CA76001

<table>
<thead>
<tr>
<th>Measure</th>
<th>SRB Month 3</th>
<th>SR Month 3</th>
<th>p-Value</th>
<th>SRB Month 12</th>
<th>SR Month 12</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median FEV1 (ml)</td>
<td>1210</td>
<td>1515</td>
<td>0.05</td>
<td>1270</td>
<td>1260</td>
<td>0.90</td>
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<tr>
<td>Median DLCO%</td>
<td>42</td>
<td>50</td>
<td>0.03</td>
<td>45</td>
<td>47</td>
<td>0.32</td>
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<tr>
<td>Median Dyspnea Score (0–100)#</td>
<td>73</td>
<td>77</td>
<td>0.87</td>
<td>77</td>
<td>75</td>
<td>0.72</td>
</tr>
</tbody>
</table>

Measure (Change from baseline) | SRB change at Month 3 | SR change at Month 3 | p-value | SRB change at Month 12 | SR change at Month 12 | p-value |
<table>
<thead>
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<tbody>
<tr>
<td>200ml change in FEV1</td>
<td>34 (47%)</td>
<td>30 (44%)</td>
<td>0.87</td>
<td>25 (51%)</td>
<td>26 (56%)</td>
<td>0.68</td>
</tr>
<tr>
<td>10 point change in DLCO%</td>
<td>35 (48%)</td>
<td>25 (37%)</td>
<td>0.23</td>
<td>19 (39%)</td>
<td>23 (51%)</td>
<td>0.30</td>
</tr>
<tr>
<td>10 point change in Dyspnea</td>
<td>46 (63%)</td>
<td>32 (51%)</td>
<td>0.17</td>
<td>28 (58%)</td>
<td>18 (43%)</td>
<td>0.20</td>
</tr>
</tbody>
</table>

Comparison of Median values; Wilcoxon Rank Sum Comparison of "change from baseline" Fisher's Exact #lower score indicates worse dyspnea.
16. Lobectomy Leads to Optimal Survival in Early-Stage Small Cell Lung Cancer
Malcolm M. DeCamp1*, Abram Recht2, John C. Flickinger3, Laura N. Medford-Davis4, Anne-Marie Dyer5, John M. Varlotto6
1. Division of Cardiothoracic Surgery, Beth Israel Deaconess Medical Center, Boston, MA, USA. 2. Department of Radiation Oncology, Beth Israel Deaconess Medical Center, Boston, MA, USA. 3. Department of Radiation Oncology, Pittsburgh Cancer Institute, Pittsburgh, PA, USA. 4. Harvard Medical School, Boston, MA, USA. 5. Department of Public Health Sciences, Pennsylvania State University, Hershey, PA, USA. 6. Division of Radiation Oncology, Pennsylvania State University, Hershey, PA, USA.
Invited Discussant: Jessica S. Donington, MD

OBJECTIVE: Small cell lung cancer (SCLC) rarely presents as Stage I or II disease. While chemotherapy is the cornerstone of therapy, local management is variable. We sought to evaluate the contemporary incidence of early-stage SCLC and to define its optimal local therapy.

METHODS: We analyzed the relative incidence, treatment patterns and outcomes of 2214 patients with early-stage SCLC (1690 Stage I and 524 Stage II) identified from the Surveillance, Epidemiology, and End Results (SEER) database from 1988–2005.

RESULTS: Early-stage SCLC constituted a relatively stable proportion of all SCLC (3–5%), of all lung cancers (0.10–0.17%), and of all Stage I lung cancers (1–1.5%) from 1988–2003, but increased significantly after that to 7–7.5%, 0.29% and 2.2% respectively (p < 0.0001 for each comparison). Utilization of surgical resection for early-stage SCLC therapy peaked at 47% of reported cases in 1990 and steadily declined to 16% by 2005. Patients treated with lobectomy or greater resections (L+) without radiotherapy (RT) had longer median survival time (50 months) than those treated with segmental or wedge resections (L-) without RT (30 months, p = 0.006) or those treated with RT alone (20 months, p = 0.0001) (Figure). Patients undergoing L- without RT also had significantly longer median survival than patients receiving RT alone (p = 0.002). Median survival time was shorter (37 months) in patients receiving RT after L+ resection than when RT was not used, but was not statistically significant (p = 0.201). The use or omission of RT made no difference after limited resection (30 versus 28 months, p = 0.585). Multivariable analysis found survival significantly related to age, year of diagnosis, tumor size, stage, and treatment (L+ without RT vs. RT alone, and vs. L- without RT).

*AATS Member
CONCLUSION: Surgery is an underutilized modality in the management of early-stage SCLC. Lobectomy provides optimal local control and leads to superior survival for these patients. While sub-lobar resection proved inferior to lobectomy, it conferred a survival advantage superior to RT-alone. The addition of RT to resection provided no additional benefit underscoring the need for multi-disciplinary care coordination for all lung cancer patients.
17. Bilateral Lung Transplantation Does Not Improve Survival in Patients 65 and Above

Shekar L. Reddy, Richa Sharma, Shu S. Lin, Scott M. Palmer, David W. Zaas, R. Duane Davis*
Cardiothoracic Surgery, Transplant Division, Duke University Medical Center, Durham, NC, USA.

Invited Discussant: Thomas K. Waddell, MD, MSc, PhD

OBJECTIVE: To study the outcomes following lung transplantation in patients aged 65 and above as a function of disease etiology and choice of operation.

METHODS: Data from United Network of Organ Sharing between 2000–2009 was analyzed. Survival of patients 65 and above was plotted. Further analysis of survival between COPD and IPF groups was performed and compared between single and double lung transplants. Survival data is presented as median days & inter-quartiles and compared with log-rank test. Variables were further explored to create a log-linear regression model for survival.

RESULTS: There were 7743 patients in the study period, of which 762 were ≥65 years and formed study group. Survival in the ≥65 group: 1194 days (IQR: 383–2108) was worse than those aged <65 yrs: 1874 days (IQR: 596–3300) p = <0.0001). In patients under 65, survival was significantly better with COPD as compared to IPF. Survival for COPD was 1976 (IQR: 696–3336) vs. IPF: 1694 (IQR: 486–3167) (p < 0.001). In the younger cohort, bilateral lung transplant as compared to single lung transplants provided a survival advantage, median survival in days BLT: 2229 (IQR: 681–* vs. SLT 1717 (IQR: 578–3228) (p < 0.0001). In contrast, patients ≥65 years, COPD patients did not have improved survival median survival COPD 1176 (IQR: 421–2108) vs. IPF 1427 (IQR: 339–1969) (p = 0.77). In this older cohort, double lung transplant did not provide a significantly better survival compared to single lung transplant (p = 0.8). Figure 1. Univariate analysis revealed age to be the only variable that was approaching significance. With log-linear regression, age achieved significance with p = <0.001 and a parameter estimate of ~36.1 and standard error of 10.9.

*AATS Member
CONCLUSION: 1) Survival following lung transplantation in patients aged 65 and above is comparable between COPD and IPF groups as well as between single and double lung transplants.

2) In older patients age appears to be a more dominant predictor on survival than diagnosis or choice of operation.

3:20 p.m. I NTERMISSION – V ISIT E XHIBITS/C OFFEE B REAK

Exhibit Hall A & B
18. Patterns of Recurrence and Incidence of Second Primary Tumors after Lobectomy by VATS versus Thoracotomy for Lung Cancer

Raja M. Flores*, Ugonna N. Ihekweazu, Nabil Rizk, Manjit S. Bains*, Robert J. Downey*, Prasad Adusumilli, David J. Finley, James Huang, Joseph Dycoyo, Valerie W. Rusch*, Bernard Park

Thoracic Surgery, Memorial Sloan-Kettering Cancer Center, Thoracic Surgery, New York, NY, USA.

Invited Discussant: Scott J. Swanson, MD

OBJECTIVE: Reports have questioned the oncologic efficacy of VATS when compared to thoracotomy despite similar survival results. Therefore, we investigated the pattern of recurrent disease and the incidence of second primaries after lobectomy by VATS and thoracotomy.

METHODS: All patients were selected for VATS or thoracotomy by a surgeon at initial evaluation at a single institution. Two surgeons exclusively performed lobectomy by thoracotomy while 7 surgeons performed VATS lobectomy. All patients who underwent lobectomy for clinical stage 1A non-small cell lung cancer by CT and PET were identified from a prospective database. Patient characteristics, recurrences, and second primary tumors were recorded. Variables were compared by student’s t-test, Pearson chi squared, and Fisher’s exact test. A logistic regression model was constructed to identify variables influencing the development of recurrent disease and metachronous tumors.

RESULTS: From 2002 to 2009, 520 patients underwent lobectomy by VATS and 652 by thoracotomy. Synchronous primaries identified at initial surgery were 34 (7%) by VATS and 78 (12%) by thoracotomy (p = 0.01). Final pathological stage was similar in VATS and thoracotomy groups. Logistic regression demonstrated a lower risk (OR = 0.67, p = 0.04) of recurrent disease and metachronous lesions in VATS patients after adjusting for age, stage, and gender.
CONCLUSION: VATS was associated with lower recurrence rates and metachronous tumors when compared to thoracotomy after controlling for age, gender, and stage. These data support VATS lobectomy as an oncologically sound technique.

<table>
<thead>
<tr>
<th>Stage</th>
<th>VATS n = 520</th>
<th>Thoracotomy n = 652</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>67 years</td>
<td>67 years</td>
<td>0.8</td>
</tr>
<tr>
<td>Female gender</td>
<td>331 (64%)</td>
<td>402(62%)</td>
<td>0.5</td>
</tr>
<tr>
<td>Size (cm)</td>
<td>2.1</td>
<td>2.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Stage IA</td>
<td>344 (66%)</td>
<td>420 (65%)</td>
<td>0.2</td>
</tr>
<tr>
<td>IB</td>
<td>92 (18%)</td>
<td>94 (14%)</td>
<td></td>
</tr>
<tr>
<td>III A</td>
<td>22 (5%)</td>
<td>36 (6%)</td>
<td></td>
</tr>
<tr>
<td>IIB</td>
<td>18 (3%)</td>
<td>28 (4%)</td>
<td></td>
</tr>
<tr>
<td>IIIA</td>
<td>31 (6%)</td>
<td>47 (7%)</td>
<td></td>
</tr>
<tr>
<td>IIIB</td>
<td>11 (2%)</td>
<td>27 (4%)</td>
<td></td>
</tr>
<tr>
<td>Locoregional Recurrence</td>
<td>20 (4%)</td>
<td>36 (6%)</td>
<td>.01</td>
</tr>
<tr>
<td>Distant Recurrence</td>
<td>32 (6%)</td>
<td>68 (10%)</td>
<td>.01</td>
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<tr>
<td>Metachronous tumors</td>
<td>15 (3%)</td>
<td>19 (3%)</td>
<td>0.5</td>
</tr>
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</table>

Logistic regression: recurrence and metachronous (dependent variable)

<table>
<thead>
<tr>
<th>OR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.02</td>
</tr>
<tr>
<td>Gender</td>
<td>.92</td>
</tr>
<tr>
<td>Pathological stage</td>
<td>1.5</td>
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<tr>
<td>VATS</td>
<td>.67</td>
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</tbody>
</table>
19. Respiratory Function after Pneumonectomy: Results of the Pneumonectomy Project

Jean Deslauriers*, Steve Provencher, Paula Ugalde, Santiago Miro, Yves Lacasse, Sébastien Bergeron, Sylvie Ferland
Centre de Pneumologie, IUCPQ (Hôpital Laval), Quebec, QC, Canada.

Invited Discussant: David J. Sugarbaker, MD

OBJECTIVE: Although more than 75 years have elapsed since the first successful one-stage pneumonectomy for lung cancer, little information is available regarding long-term changes in pulmonary function in such individuals. The objectives of this study were to analyse these changes so that surgical myths dating back to the early days of thoracic surgery could be revisited.

METHODS: Among 523 consecutive patients who underwent pneumonectomy for lung cancer between 01–92 and 09–01, 117 were alive at the time of study (2006) and thus had 5 years minimum follow-up. Of these, 17 were excluded, leaving 100 individuals available for study (mean follow-up time of 9.1 ± 2.8 years). Over one day, each patient underwent complete medical history, standard chest radiographs, thoracic MRI, pulmonary function studies, arterial blood gas analysis, a 6-minute walk test, and cardiac ultrasonography. Each also completed the ATS respiratory questionnaire form.

RESULTS: In comparison with pre-operative values, the functional percentage losses in expiratory lung volumes were 38 ± 19% for FEV1, 31 ± 24% for FVC and 33 ± 18% for DLCO. There was a significant correlation between pre and post-operative FEV1 (p < 0.001) although there was a wide range of variation between individuals (R = 0.47). In a multivariate linear regression analysis, normal ipsilateral diaphragmatic motion, higher pre-operative FEV1, more hyperinflation of the remaining lung, female gender and younger age were significant factors for better post-operative FEV1 (p < 0.01). Gas exchange at rest was normal (PaO2 = 88 ± 10 mm Hg; PaCO2 : 42 ± 3 mmHg), and exercise tolerance as assessed by the 6-minute walk test was also normal (83 ± 17% of predicted values). Most patients (73%) had no or only minimal dyspnea (ATS grade 0–2). Twenty eight patients (36%) with measurable ultrasonographic PAP had pulmonary hypertension but in most it was mild to moderate (mean of 37 ± 8) and not associated with significant differences in lung function (p = 0.34 for FEV1), gas exchange (p = 0.01) and exercise tolerance (p = 0.99).

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CONCLUSION: These findings are important because they show that despite a worsening of lung function by 30–35%, gas exchange at rest and exercise tolerance remain normal after pneumonectomy. Most importantly, hyperinflation of the remaining lung is not detrimental to residual lung function. Pulmonary hypertension is uncommon and in most cases it is only mild to moderate.
Objective: In clinical stage IIIA non-small cell lung cancer, the role of surgical resection, particularly pneumonectomy, after induction therapy remains controversial. The objective of this report is to determine the factors predictive of survival following induction therapy.

Methods: This is a retrospective review of a prospectively collected database of 136 patients who underwent surgical resection after induction chemotherapy (n = 119) or chemoradiation (n = 17) from 6/1990 and 7/2009. Survival was calculated using the Kaplan-Meier method and multivariate predictors were determined using Cox regression analysis.

Results: Median age was 64 years (female (n = 79 (58%)). 109 (bi) lobectomies and 27 pneumonectomies were performed. There was one hospital death (0/109 lobectomy; 1/27 pneumonectomy). Sixty-seven patients were downstaged to N0/N1 nodal status (49%). There were 2 complete pathological responses. Median followup was 29 months (range 0.36 to 161 months). Overall 5-year survival for the entire cohort was 40% (44% lobectomy, 26% pneumonectomy, p = 0.002). Patients who were pathologically downstaged to pN0/N1 had improved 5 year survival (54% vs. 27%, p = 0.008, respectively). For patients with ypN0/N1 disease, survival following lobectomy was better than after pneumonectomy (5 yr survival 58% vs. 30%, p = 0.02) (Table 1). In patients with residual N2 disease, there was no statistically significant survival difference seen between lobectomy and pneumonectomy (5 yr survival 28% vs. 23%, respectively, p = 0.06) (Table). Multivariate analysis showed the following to be independent predictors of survival: age (Hazard Ratio (HR) 1.06; p = 0.005), extent of resection (HR 2.72; p = 0.002), and residual pN2 (HR 1.73; p = 0.027).
CONCLUSION: After induction therapy for patients with clinical stage IIIA disease, both pneumonectomy and lobectomy can be safely performed. Although survival after lobectomy is better, long-term survival can be accomplished following pneumonectomy in appropriately selected patients.

5:00 p.m. ADJOURN

Table 1: Five Year Survival

<table>
<thead>
<tr>
<th>pN</th>
<th>Lobectomy</th>
<th>Pneumonectomy</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pN0/N1</td>
<td>53% (n = 57)</td>
<td>30% (n = 10)</td>
<td>0.02</td>
</tr>
<tr>
<td>pN2</td>
<td>28% (n = 52)</td>
<td>23% (n = 17)</td>
<td>0.06</td>
</tr>
</tbody>
</table>
2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – CONGENITAL HEART DISEASE
Constitution 105, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)

Moderators: Emile A. Bacha, MD
James S. Tweddell, MD

21. Application of the Hemi-Mustard Bidirectional Glenn Atrial Switch in the Double Switch Procedure for Congenitally Corrected Transposition of the Great Arteries: Rationale and Midterm Results
Sunil P. Malhotra, V. Mohan Reddy, Mary Qiu, Timothy J. Pirolli, Laura Barboza, Olaf Reinhartz, Frank L. Hanley
1. Congenital Heart Center, University of Florida, Gainesville, FL, USA.
2. Cardiothoracic Surgery, Stanford University, Stanford, CA, USA.

Invited Discussant: David J. Barron, MD

OBJECTIVE: The double switch operation for congenitally corrected transposition of the great arteries (cc-TGA) prevents long-term systolic dysfunction of the systemic ventricle. The Senning and Mustard procedures for simple transposition have demonstrated significant long-term morbidity, specifically related to sinus node dysfunction and superior vena caval baffle obstruction. To avoid these complications, we favor a modified atrial switch, consisting of a hemi-Mustard to baffle IVC return to the tricuspid valve in conjunction with a bidirectional Glenn (BDG). Moreover, the hemi-Mustard is more ideally suited to the technical challenges presented by the high incidence of mesocardia, dextrocardia, and situs inversus observed with cc-TGA. In cases of pulmonary atresia where a RV to PA conduit is indicated, the reduced RV volume resulting from the BDG promotes conduit longevity.

METHODS: Between January 1994 and September 2009, 56 patients with congenitally corrected transposition of the great arteries (cc-TGA) were managed surgically. An anatomic repair was achieved in 48 patients (87%). The Rastelli/atrial switch was performed in 22 patients with pulmonary atresia and an arterial/atrial switch was performed in 26 patients. A

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hemi-Mustard with BDG was the atrial switch for 63% of anatomic repairs (30/48). A Senning procedure was performed in 18 patients when BDG was contraindicated.

RESULTS: There was one in-hospital death following anatomic repair (1/48). There have been no late deaths to date. No patients have required cardiac transplantation. At a median follow-up of 41.4 months (range 3 m–14.2 y), 46/48 late survivors are in NYHA class I status and the remaining 2 are NYHA Class II. Normal LV systolic function was demonstrated by follow-up echocardiography in 92% (44/48). Major reoperations included revision of the coronary button in 2 patients and BDG takedown in one patient due to development of a circular shunt. There have been no baffle-related reoperations. Tricuspid regurgitation decreased from a mean grade 2.3 to 1.2 after repair (p = 0.00002). To date, 17 of 24 patients (71%) that underwent left ventricle training have proceeded to anatomic repair.

CONCLUSION: We describe a 15-year experience with anatomic repair for cc-TGA using a modified atrial switch with favorable midterm results. Baffle-related complications have been minimized using the hemi-Mustard/BDG atrial switch. Cardiac transplantation was avoided in all cases and excellent functional status was observed at follow-up.
22. **Aortic Valve Repair by Cusp Extension for Rheumatic Aortic Insufficiency in Children: Long-Term Results and Impact of Extension Material**

Patrick O. Myers¹, Cecile Tissot³, Jan T. Christenson², Maurice Beghetti³, Mustafa Cikirikcioglu², Afksendiyos Kalangos²*

1. Cardiac Surgery, Brigham & Women’s Hospital, Boston, MA, USA.
2. Cardiovascular Surgery, Geneva University Hospitals & School of Medicine, Geneva, Switzerland. 3. Unit of Pediatric Cardiology, Geneva University Hospitals & School of Medicine, Geneva, Switzerland.

**OBJECTIVE:** Aortic valve repair has good mid term results in selected patients. However, neither the long-term results of pericardial cusp extension in children nor the durability of different pericardial fixation techniques have been reported. Our goal was to evaluate the long-term results of aortic valve repair by tailoring of cusp extension, and assess the impact of using different pericardial materials for cusp extension.

**METHODS:** Seventy-eight children with severe rheumatic aortic regurgitation (mean age 12 ± 3.5, range 3–18 years) underwent aortic valve repair using triple cusp extension over a 12 year period. The aortic cusps were extended using fresh autologuous pericardium in 53 patients (67.9%), glutaryldehyde-fixed bovine pericardium (St. Jude Medical Inc., St. Paul, MN) in 9 (11.5%) and Photofix bovine pericardium (CardioFix, Sorin Carbomedics, Milano, Italy) in 16 (20.5%). Fifty-seven patients (73.1%) underwent concomitant mitral valve repair for associated rheumatic mitral valve disease, and 8 (10.3%) underwent tricuspid valve repair. All patients were then followed-up by transthoracic echocardiography at 6 months and at yearly intervals thereafter.

**RESULTS:** There was one operative death from left ventricular failure. Follow-up was complete in 98.7% of patients (76 of 77). During a median follow-up of 10.7 years (range 1 months–16.4 years), 1 late death (1.3%) occurred and 15 patients (19.7%) required reoperation for aortic valve replacement at a mean of 43 ± 33.7 months (range 1 month–9 years), 9 within the autologuous pericardium group (18%), 3 within the bovine pericardium group (33%) and 3 within the Photofix pericardium group (19%). Actuarial freedom from reoperation was 96 ± 2.3% at 1 year, 87.5 ± 3.9% at 5 years, 84.6 ± 4.3% at 7 years, 80.7 ± 4.9% at 10 years, 78.7 ± 5.2% at 12 years and 75.3 ± 6% at 15 years, with a significantly higher reoperation
rate in the bovine pericardium group (log rank test, p = 0.02). On multi-
variate Cox proportional hazard analysis, greater age (hazard ratio (HR) 1.25, p < 0.0001) and acute rheumatic pancarditis (HR 8.15, p = 0.001) at operation were significant predictors of reoperation.

CONCLUSION: Aortic cusp extension provides adequate valve repair in a large proportion of children with rheumatic aortic regurgitation. Fresh autologous and Photofix bovine pericardium showed better durability, requiring fewer reoperations.
23. Atrioventricular Valve Repair in Patients with Single Ventricle Physiology: Impact of Ventricular Function and Morphology, Valve Morphology and Mechanism of Insufficiency on Outcomes

Osami Honjo, Cori Atlin, Luc Mertens, Osman O. Al-Radi, Andrew N. Redington, Christopher A. Caldarone*, Glen S. Van Arsdell*

The Labatt Family Heart Centre, The Hospital for Sick Children, Toronto, ON, Canada.

Invited Discussant: Jennifer C. Hirsch, MD

OBJECTIVE: We hypothesized that ventricular function and morphology, valve morphology, and mechanism of insufficiency may be the determinants of survival in single ventricle patients undergoing atrioventricular (AV) valve repair.

METHODS: From '98–'08, 58 (13%) of 422 single ventricle patients underwent AV valve repair. Diagnoses included HLHS (n = 32), DORV (n = 8), AVSD (n = 10), and tricuspid atresia/DILV (n = 4). Timing was at stage II in 34 (59%), between stage II and III in 10 (17%), at stage III in 10 (17%) and after the Fontan in 2 (3%). Valve morphology, regurgitation mechanism, ventricular morphology and function were analyzed for impact on survival and repair outcome using Cox regression. A case match control analysis was performed for survival/reoperation/transplantation.

RESULTS: Valve morphology: tricuspid (n = 38 [67%]), common (n = 16 [28%]), and mitral (n = 2 [4%]). Ventricular morphology: RV (n = 48 [83%]) and LV (n = 8 [14%]). Regurgitation mechanism: prolapse (n = 24 [46%]), dysplasia (n = 18 [35%]), annular dilatation (n = 8 [15%]), and restriction/cleft (n = 2 [4%]). Post-repair insufficiency was none/trivial in 14 (26%), mild in 33 (61%), and moderate in 7 (13%). Survival in the valve repair group was lower than case match controls (1 year, 77 vs. 94%, 5 years, 66 vs. 86%, p = 0.004). No differences were found in survival among diagnoses, ventricular or AV valve morphology, mechanism of insufficiency or timing of surgery (p = not significant). Valve and ventricular repair status was divided into 4 groups based on valve and ventricular function. (Figure 1A): Group 1 (n = 42) had significantly higher survival (1 year, 83%, p = 0.02) than other groups: Group 2 (n = 5, 1 year, 60%), Group 3 (n = 9, 1 year, 33%), and Group 4 (n = 2, 1 year, 50%). The competing risk outcomes are shown in Figure 1B. Cox regression showed that predictors for death/transplant included cardiopulmonary bypass (CPB) time and postoperative ventricular function (p < 0.02 for both).

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for all events (death/transplant/reoperations) included body surface area, CPB time, preoperative regurgitation grade, and ventricular dilatation (p < 0.05 for all). No predictor was identified for re-repair/replacement.

**CONCLUSION:** Patients with mild or less residual regurgitation and normal ventricular function had a better survival than other sub groups but not equivalent to case match controls. Less than optimal ventricular function following repair predicted death suggesting that strategies designed to preserve ventricular function are of prime importance.
24. The Kinetics of the Development of Ventricular Dysfunction in Patients with Hypoplastic Left Heart Syndrome

Heather Underkofler¹, Michele Frommelt³, Raymond T. Fedderly³, Pippa M. Simpson², Michael E. Mitchell³, James S. Tweddell⁴*, Kimberly L. Gandy³

¹. Surgery, Medical College of Wisconsin, Milwaukee, WI, USA. ². Pediatrics, Children's Research Institute, Milwaukee, WI, USA. ³. Children's Hospital of Wisconsin, Milwaukee, WI, USA.

Invited Discussant: Shunji Sano, MD, PhD

OBJECTIVE: Although the survival of children with single ventricle physiology has improved, a significant number experience decline in ventricular function over time. The kinetics and incidence, however, of functional decline needs further description.

METHODS: One hundred and four patients born between 1999 and 2005 with a diagnosis of hypoplastic left heart syndrome (HLHS) were evaluated and were the subjects of this retrospective study. Echo data was analyzed to determine functional decline of the single ventricle at a number of time points for each patient. Cardiac function was evaluated with echocardiography by two independent evaluators. Function was classified as normal, mildly depressed, moderately depressed, and severely depressed. Factors that were thought to have potential impact on subsequent cardiac function and were concurrently evaluated included: aortic coarctation, tricuspid regurgitation, aortic insufficiency, creation of a fenestration at time of Fontan, and age at original operation. Additionally, twenty-three patients born between 1999–2005 with a diagnosis of tricuspid atresia were studied retrospectively and used for comparison. Function was evaluated in this patient population by ejection fraction. Gender, age at each operation, weight at initial operation, and saturation at each stage of operation were evaluated in both populations.

RESULTS: At 87 months, 50% of HLHS patients had decline in function. Patients that died had a more rapid rate of functional decline (p < 0.001). The factors affecting decline were fenestration at time of Fontan and aortic coarctation. When the degree of dysfunction was divided into subcategories based on severity of dysfunction, aortic arch obstruction was found more often in patients that progressed to moderate dysfunction than in those whose function remained normal (p = 0.003). Patients that had a fenestration had less dysfunction than patients that did not have a
fenestration (p < 0.001). Neoaortic regurgitation and tricuspid valve regurgitation did not correlate with the development of dysfunction. Patients with tricuspid atresia and a functional left ventricle had less decline than did patients with a hypoplastic left ventricle and a functional right ventricle.

**CONCLUSION:** Cardiac function declined postoperatively in patients with HLHS. Aortic coarctation and absence of a fenestration correlated with an increased incidence of functional decline in the HLHS population.

3:20 p.m. **INTERMISSION – VISIT EXHIBITS/COFFEE BREAK**
*Exhibit Hall A & B*
3:55 p.m.  SIMULTANEOUS SCIENTIFIC SESSION –
CONGENITAL HEART DISEASE
Constitution 105, Metro Toronto Convention Centre

Moderators:  Emile A. Bacha, MD
James S. Tweddell, MD

25. Subsequent Aortic Arch and Left Ventricular Outflow Tract Procedures in Patients after Interrupted Aortic Arch Repair: A Multi-Institutional Study


¹. Surgery, Division of Cardiovascular Surgery, The Hospital for Sick Children, Toronto, ON, Canada. ². Pediatric and Congenital Heart Surgery, Cleveland Clinic Foundation, Cleveland, OH, USA. ³. Cardiothoracic Surgery, Children’s Mercy Hospital, Kansas, MO, USA. ⁴. Surgery, Division of Thoracic and Cardiovascular Surgery, The Congenital Heart Institute of Florida (CHIF), Cardiac Surgical Associates of Florida (CSAoF), University of South Florida (USF), All Children’s Hospital, Children’s Hospital of Tampa, Saint Petersburg and Tampa, FL, USA. ⁵. Division of Cardiac Surgery, Center for Heart, Lung and Kidney Disease, Children’s National Medical Center, Washington, DC, USA. ⁶. Pediatrics, Division of Cardiology, The Hospital for Sick Children, Toronto, ON, Canada. ⁷. Thoracic and Cardiovascular Surgery, Cleveland Clinic Foundation, Cleveland, OH, USA. ⁸. Surgery, Division of Cardiothoracic Surgery, University of Miami Health Systems, Miller School of Medicine, Miami, FL, USA. ⁹. Cardiothoracic Surgery, The Congenital Heart Institute at Arnold Palmer Hospital for Children, Orlando, FL, USA.

Invited Discussant:  Charles D. Fraser, MD

OBJECTIVE:  Surgical management of interrupted aortic arch (IAA) requires aortic arch (AoA) repair and is often associated with multiple subsequent procedures (SPs) directed at the AoA and/or the left ventricular outflow tract (LVOT). We sought to determine time-dependent outcomes and freedom from these SPs after an index AoA repair, and factors associated with these SPs.

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METHODS: 470 patients (pts) with IAA at 33 institutions from 1987–1997 were reviewed. “Time zero” was the date of index AoA repair. SPs were classified by type (catheter-based or surgical) and focus (AoA, LVOT, both, or neither). Competing risks and modulated renewal analysis were used to explore SPs of the AoA and LVOT.

RESULTS: 447 of the 470 pts had an index AoA repair (318 type B IAA), with concomitant repair of the LVOT in 39 pts. 287 pts had no AoA or LVOT SPs. 119 pts had 158 AoA SPs, 70 pts had 101 LVOT SPs, and 231 pts had 436 SPs of any type (136 catheter-based, 300 surgical). 15 years after index repair, competing risks analysis (AoA SPs, n = 447) demonstrated that 32% had died, 29% had an AoA SP, and 39% remained alive without an AoA SP. Factors associated with a first AoA SP included index AoA repair with an unrepaired ventricular septal defect (VSD) or use of the left subclavian artery as a patch/conduit, and worse clinical status at the time of admission for index repair. Modulated renewal analysis demonstrated a lower risk of further repeated AoA SPs after the first AoA SP (Figure). 15 years after index repair, competing risks analysis (LVOT SPs, n = 423) demonstrated that 33% had died, 18% had a LVOT SP, 48% remained alive and at risk, and 1% were alive but no longer at risk for an LVOT SP. Factors associated with a first LVOT SP included initial anatomic features (outlet VSD, smaller aortic valve, bicuspid aortic valve), concomitant procedures at index AoA repair (pulmonary artery (PA) banding, systemic to PA shunt, VSD repair not via right atrium), and undergoing a non-AoA procedure prior to the first LVOT SP. Modulated renewal analysis demonstrated a higher risk of further repeated LVOT SPs after the first LVOT SP (Figure).

CONCLUSION: IAA repair is associated with a high prevalence of subsequent aortic arch and left ventricular outflow tract procedures. Anatomic and procedural factors are associated with increased risk. After the first subsequent procedure, the risk of further aortic arch procedures decreases, while the risk of further left ventricular outflow tract procedures increases.
OBJECTIVE: When mitral and tricuspid leaflets are retracted or tethered, acceptable coaptation can’t always be obtained using standard repair techniques, such as ring annuloplasty, leaflet shaving, resection of retracted secondary or primary chordae tendineae and pericardial patch leaflet augmentation. We assessed the safety and mid-term results using a novel technique used as a salvage procedure before valve replacement, to address leaflet retraction or tethering in children.

METHODS: All patients who required this additional technique after valve repair with an unacceptable result and in whom a conversion to valve replacement was considered were included, between March 2003 and May 2009. A 5–0 polypropylene suture was placed on the free edge of the retracted leaflet, reinforced by an autologous pericardial pledget if the tissue appeared fragile, and then anchored to the atrial side of the contralateral annulus. This allowed the retracted or tethered leaflet to be brought up and rest closer to the natural coaptation plane and improved the repair result in all patients. 40 consecutive children required this additional technique, 36 for the mitral valve (rheumatic 22, congenital 11, functional 1, degenerative 1 and endocarditis 1) and 4 for the tricuspid valve (dysplastic 2, Ebstein 2), with a mean age of 11.4 ± 3.9 years.

RESULTS: Mean aortic cross-clamping and CPB times were 34.3 ± 15.9 and 66.5 ± 24.2 minutes, respectively. There were no early deaths. At discharge, 1 patient in the mitral group had mild regurgitation (the remainder having trivial or no regurgitation) with a transvalvular gradient of 4.4 ± 2.5 mmHg (range 1.3–11, >7 in 3), and all patients had no or trivial tricuspid regurgitation with a transvalvular gradient of 2 ± 1.4. During a follow-up of 32.5 ± 19.2 months, there were no late deaths and no thromboembolic events. Three patients with rheumatic mitral valve repair required reoperation for mitral valve replacement at a median of...
3 months from the repair. At echocardiography of the remaining patients, the repair remained stable; the transvalvular gradient progressed to $5.3 \pm 2.2$ (range 2–11, >7 in 1) for the mitral valve and $2 \pm 1.4$ for the tricuspid valve, with no suspension suture breakage.

**CONCLUSION:** This suspension technique is simple and safe. It creates a large coaptation area that can eliminate regurgitation and avoid or delay valve replacement, with acceptable diastolic transvalvular gradients in most patients, which did not significantly increase with the child's growth.
Pediatric Heart Re-Transplantation: Patterns of Primary Graft Failure Differ Between Recipients Initially Transplanted in Infancy Compared to Older Children

John M. Karamichalis, Shelley Miyamoto, David N. Campbell*, Jilayne Smith, Sydne Clark, Biagio A. Pietra, Max B. Mitchell
Cardiac Surgery and Cardiology, The Children's Hospital, University of Colorado Denver Health Science Center, Denver, CO, USA.

Invited Discussant: Jonathan M. Chen, MD

OBJECTIVE: The goal of this study was to compare patterns of primary graft failure between children undergoing heart transplantation in infancy vs. older children.

METHODS: A retrospective review of all transplants (1988–2009, n = 341) at our institution was performed. Children undergoing cardiac re-transplantation (n = 25) were identified and stratified by age at primary transplant: Group 1 (infants <1 year of age), and Group 2 (≥1 year of age). Immunosuppression in all patients was calcineurin based and steroid-free.

RESULTS: There were 14 Group 1 patients and 11 Group 2 patients. The mean age at primary transplant was 0.4 ± 0.3 years in Group 1 vs. 8.5 ± 5.7 years in Group 2, p < 0.01. Congenital heart disease was the commonest indication for primary transplant in Group 1 (64%) vs. cardiomyopathy in Group 2 (64%). Acute rejection episodes prior to re-transplant were more common in Group 2 (Group 1: 2.2 ± 2.0 per patient, vs. Group 2: 4.6 ± 2.5 per patient, p < 0.05). All patients in Group 2 had 2 or more episodes of acute rejection (range 2–10). Three of 14 Group 1 patients had no acute rejections prior to re-transplantation. Primary graft survival for Group 1 (9.2 ± 3.1 years) was significantly longer than Group 2 (4.2 ± 2.1 years, p < 0.01). No difference in age at re-transplantation was apparent (9.7 ± 3.2, Group 1, vs. 12.8 ± 5.3 years Group 2). Hospital survival for all re-transplants was 100%. Indications for re-transplantation differed between groups: transplant coronary artery disease (TCAD) was the indication in 6 of 14 (43%) Group 1 patients, vs. 1 of 11 (9%) in Group 2. Recurrent or chronic cellular rejection was the indication for re-transplantation in 5 of 14 (36%) Group 1 patients, compared to 10 of 11 (91%) Group 2 patients.

*AATS Member
CONCLUSION: Infant heart recipients have longer primary graft survival and fewer episodes of acute rejection prior to re-transplantation compared to older pediatric heart recipients requiring re-transplantation. Despite an advantage in adaptive immunity in infant heart recipients as evidenced by less acute rejection, graft life is limited by TCAD. Further investigation into the etiology and treatment for TCAD should improve primary graft survival in infant heart recipients.

5:00 p.m.   ADJOURN
AMERICAN ASSOCIATION FOR THORACIC SURGERY

NOTES
OBJECTIVE: Hibernating myocardium may not become normal following successful coronary artery bypass grafting (CABG), potentially because of altered expression of mitochondrial proteins in chronically ischemic, viable tissue. This inability to support electron flow through the electron transport chain (ETC) may lead to insufficient oxygen consumption during high work states, which potentially may have a negative impact on oxygen expenditure and contraction. In a swine model of hibernating myocardium, we hypothesized that persistent myocardial wall motion abnormality in revascularized myocardium by off-pump CABG is due to persistently depressed expression of key mitochondrial proteins involved with the ETC.

METHODS: In a novel animal model, seven pigs were instrumented at age 4 weeks with a constrictor around the proximal left anterior descending (LAD) artery. By 12 weeks, a severe stenosis was demonstrated by multi-detector computer tomography (MDCT) along with an anteroseptal wall motion abnormality by 2D ECHO. Using off-pump CABG, the left internal mammary artery (LIMA) was grafted to the mid-LAD and pigs recovered for 4 weeks. During the terminal study, regional blood flows were determined by colored microspheres at baseline and during stress with...
dobutamine infusion (40 μg/kg/min-iv). Following sacrifice, mitochondria were isolated from myocardial regions and protein expression from the ETC was determined, using proteomic analysis with iTRAQ.

**RESULTS:** Prior to sacrifice, repeat MDCT showed a widely patent LIMA to the LAD and 2D ECHO demonstrated improved but mildly reduced regional function in the LAD region. TTC staining confirmed the absence of necrosis. As shown in the Table, regional myocardial blood flow (MBF) was lower in the LAD region during the high work state at a time that the graft was widely patent. Essential mitochondrial proteins for ATP production within the ETC, including NADH dehydrogenase, ATP synthase, and cytochrome c oxidase were >40% lower in the LAD region compared with remote regions.

**CONCLUSION:** In successfully revascularized hibernating myocardium, resting regional function does not normalize and MBF at a high work state are lower than remote regions. Critical proteins within the mitochondria remain depressed despite successful CABG, and may explain an inability of myocytes to maximize oxygen utilization. Persistent mitochondrial adaptations to chronic ischemia despite CABG may provide insight into failure of complete recovery in hibernating myocardial tissue.

<table>
<thead>
<tr>
<th>Myocardial Region</th>
<th>MBF-Basal Work Load</th>
<th>MBF-Dobutamine Stress</th>
<th>NADH Dehydrogenase</th>
<th>Cytochrome C Oxidase</th>
<th>ATP Synthase</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD</td>
<td>0.58 ± 0.05</td>
<td>2.54 ± 24†</td>
<td>0.58†</td>
<td>0.57†</td>
<td>0.53†</td>
</tr>
<tr>
<td>Remote</td>
<td>0.63 ± 0.07</td>
<td>3.46 ± 0.33</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
</tbody>
</table>

MBF (ml/min/g); †p < 0.05 versus remote (Student’s t-test)
F2. Transforming Growth Factor-β Induces the Expression of Extracellular Matrix Molecules in Myxomatous Mitral Valves

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Invited Discussant: Y. Joseph Woo, MD

OBJECTIVE: The histological hallmark of myxomatous mitral valve disease is abundant and disorganized extracellular matrix. The primary etiology for myxomatous degeneration in sporadic cases is unknown. Transforming growth factor (TGF)-β has been implicated in the pathogenesis of myxomatous mitral valves in fibrillin-1 deficient mice where treatment with TGF-β antagonism in-vivo rescues the phenotype. The purpose of this study was to test the hypothesis that TGF-β signaling is activated in human myxomatous mitral valve disease.

![Graphs showing expression levels of various extracellular matrix molecules and TGF-β isoforms.](image)

*Real-time PCR of respective target mRNA in normal mitral valve tissue and myxomatous mitral valve relative to GAPDH. *p<0.05

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METHODS: Mitral valve tissue (posterior leaflet) was obtained from organ donors, cardiac transplant recipients, and patients undergoing mitral valve repair by an approved institutional protocol. Tissue specimens were paraffin embedded and processed for mRNA. Immunohistochemistry was performed using anti-phosphoSMAD 2/3 antibody. Real-time PCR was performed with for multiple extracellular matrix targets. Tissue cultures were performed in serum free RPMI 1640 with or without TGF-β (12 ng/ml) for 6 hours.

RESULTS: Histological examination confirmed abundant and disorganized expression of collagen and elastin. By real-time PCR, mRNA transcripts for collagen-1A1, collagen-1A2, collagen-3A1, collagen-5A1, and elastin were significantly increased in myxomatous valves compared to normal valves. Expression of TGF-β1, -β2, and -β3 as well as TGF-β receptor 1 and 3 were also up-regulated in diseased valves (see figure). The association between increased extracellular matrix molecules and TGF-β ligands and receptors was further investigated using an organ culture system. TGF-β treatment of normal mitral valve tissue resulted in up-regulation of collagen-1A1, collagen-1A2, collagen-3A1, and elastin as well as TGF-β1 and TGF-β3 transcripts. Conversely, treatment with TGF-β neutralizing antibody resulted in down-regulation of collagen-1A2, collagen-3A1, and TGF-β1 transcripts. Immunohistochemistry using phospho-SMAD 2/3 antibodies demonstrated increased nuclear labeling in myxomatous mitral valve tissue when compared to non-diseased mitral valves indicating activation of the TGF-β signaling pathway.

CONCLUSION: These results support a functional role for TGF-β signaling in the pathogenesis of myxomatous degeneration suggesting that agents with anti-TGF-β activity might be of potential benefit in patients with myxomatous mitral valve disease.
F3. **Less Invasive and Highly Effective Method for Preventing Postoperative Atrial Fibrillation by the Intraoperative Application of a Novel Biodegradable Disc Containing Amiodarone**

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**Invited Discussant:** Ralph J. Damiano, Jr., MD

**OBJECTIVE:** Atrial fibrillation (AF) is a common and undesirable complication after cardiac surgery. Amiodarone is a potent anti-AF agent; however, its systemic administration induces serious side-effects such as interstitial pneumonia. To avoid such systemic side effects, we develop a novel local sustained release system of amiodarone.

**METHODS:** A biodegradable, cross-linkable dextran disc (10 mm of diameter) was developed as a sustained release carrier for amiodarone. Under general anesthesia, Japanese white rabbits underwent median sternotomy and the novel bio-disc containing amiodarone (30 mg) was implanted onto the surface of the right atrium. The disc without amiodarone was used as a control. After implantation, chest was closed. <Study 1> Three days after implantation, tissue concentrations of amiodarone were measured in the atria, ventricles, lungs, liver, thyroid, and the blood of the rabbits (n = 5). <Study 2> Three days after implantation, AF threshold (AFT) and atrial effective refractory period (AERP) of the left atrium were measured and compared.

**RESULTS:** <Study 1> The tissue concentrations of amiodarone in the atria, ventricles, lungs, liver, and thyroid were 827 ± 381, 10.5 ± 8.1, 1.8 ± 1.1, 1.3 ± 0.5, and 7.9 ± 7.3, respectively. The atrial concentration of amiodarone was far higher than those of the other organs (p < 0.001, respectively). The blood concentration of amiodarone was under detection. <Study 2> The bio-disc containing amiodarone significantly increased the AFT (6.9 ± 4.6 mA for the amiodarone group, vs. 0.46 ± 0.63 mA for the control group, p = 0.0007) and ERP (53.9 ± 8.9 ms for the amiodarone group vs. 43.9 ± 9.5 ms for the control group, p = 0.035) of the left atrium, which indicates that bio-disc containing amiodarone had pharmacological effect.
on left atrium, and significantly reduced the threshold of inducing AF. As for the duration and the frequency of AF, in 5mA and 10mA stimulus the amiodarone group significantly kept low and less likely to induce AF compared with the control group. (P < 0.05)

CONCLUSION: A novel biodegradable disc containing amiodarone is easily applicable and maintained effective atrial concentration without the elevation of other tissue or the blood concentration. This approach may be less invasive and highly effective therapeutic option to prevent post-operative AF.
F4. Cardiac Xenotransplantation Technology Provides Materials for Improved Bioprosthetic Heart Valves
Christopher G. McGregor1*, Nermine Lila2, Michal Vlasin1, John S. Logan1, Guerard W. Byrne1

Invited Discussant: R. Duane Davis, MD

OBJECTIVE: Humans and Old World primates have high levels of anti-Gal antibody. Gal knockout pigs (GTKO) were produced by somatic nuclear transfer (cloning) to eliminate this dominant xeno antigen from prospective pig cardiac donors. Today's commercially available bioprosthetic heart valves (BHV), of porcine and bovine origin, retain the Gal antigen despite current processing techniques. Anti-Gal immune responses have been reported in patients after implantation of BHVs. BHV degeneration with calcification may, in part, have an immunological basis. This study tests whether binding of human anti-Gal antibody effects calcification of wild type and GTKO glutaraldehyde-fixed porcine pericardium using a standard rat implantation model.

METHODS: Pericardium was obtained from GTKO and wild type pigs. Expression of α-Gal was characterized by lectin GS1B4 staining. Glutaraldehyde-fixed pericardial disks from Gal-positive and GTKO pigs were implanted into 12-day-old Wistar rats with and without prelabeling with affinity purified human anti-Gal antibody. Calcification of the implants was determined after 3 weeks by inductively coupled plasma spectroscopy.

RESULTS: The α-Gal antigen was detected in wild type but not GTKO porcine pericardium. Wild type pericardial disks prelabeled with human anti-Gal antibody exhibited significantly greater calcification compared to antibody free wild type samples (111 ± 8.4 mg/g and 74 ± 9.6 mg/g respectively, P = 0.01, mean + SEM). In the presence of anti-Gal antibody significantly greater level of calcification was detected in wild type compared to GTKO pericardium (111 ± 8.4 mg/g and 55 ± 11.8 mg/g respectively. P = 0.005). Calcification of GTKO pericardium was not effected by the presence of anti-Gal antibody (51 ± 9.1 mg/g and 55 ± 11.8 mg/g).

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CONCLUSION: In this model, anti-Gal antibody accelerates calcification of wild type but not GTKO glutaraldehyde-fixed pericardium. This study suggests that preformed and induced anti-Gal antibody may contribute to calcification of currently used BHVs. GTKO pigs may become the preferred source for new potentially calcium resistant BHVs allowing greater durability and wider application of this valve type to younger patients.
F5. Cardiac Insulin Resistance as a Risk Factor for Heart Failure
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Invited Discussant: Todd K. Rosengart, MD

OBJECTIVE: Pressure overload and ischemic heart disease are the two most common causes of heart failure (HF). Defects in mitochondrial substrate oxidation (SOX) are thought to play a causal role (Neubauer NEJM 2009). Insulin resistance (IR) affects SOX and mitochondrial function. Clinically, systemic IR is an independent predictor of HF and cardiovascular mortality. However, it is not clear whether IR also affects the heart. Our aims were: First, to assess whether cardiac insulin signaling is impaired in HF. Second, to determine that cardiac IR is associated with diminished SOX and the development of HF.

METHODS: HF was induced in rats by either ligation of the left anterior descending artery (LAD) or transverse aortic constriction (TAC). Two weeks after LAD ligation or 2, 10, and 20 weeks after TAC, cardiac size and function were determined by echocardiography, glucose (GO) and fatty acid (FAO) oxidation rates as well as insulin response were measured in the isolated working heart using radioactive tracers, and the hyperinsulinemic, euglycemic clamp was used to determine whole body insulin sensitivity. Finally, maximal respiratory capacity (state 3) was measured in isolated mitochondria.

RESULTS: The Table summarizes the key results (* = p < 0.05). Echocardiography revealed left ventricular dilation, and impaired contractility two weeks after LAD ligation and 20 weeks after TAC. Animals presented dyspnea and pleural effusions as signs of HF. At 2 and 10 weeks of TAC, hearts were hypertrophied with normal contractile function. Both LAD ligation and TAC caused less power generation and reduced fatty acid and glucose oxidation in the isolated working heart. Importantly, insulin response was dramatically reduced in HF. However, the expected increase in GO (ΔGO) and decrease in FAO (ΔFAO) were already significantly blunted at 10 weeks of TAC, where contractile and mitochondrial function were still preserved. In contrast, the hyperinsuleniemic, euglycemic clamp demonstrated normal whole body glucose utilization (Sham: 25.1 ± 6.2; PO: 27.7 ± 3.9; p = NS), suggesting the presence of cardiac IR in the absence of systemic IR.

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**CONCLUSION:** Cardiac insulin resistance appears to be a risk factor for heart failure (in both ischemic and pressure overload HF), independent of systemic IR. The temporal sequence of events suggests that the onset of cardiac IR may trigger mitochondrial and contractile dysfunction.
F6. Diazoxide Mediated Maintenance of Myocyte Volume Homeostasis During Stress Requires KATP Channel Subunit SUR1

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Invited Discussant: Friedhelm Beyersdorf, MD

OBJECTIVE: Adenosine triphosphate-sensitive potassium (KATP) channel opener diazoxide is cardioprotective and mimics ischemic preconditioning in animal models. Diazoxide also inhibits the detrimental cell swelling and reduced contractility observed in animal and human myocytes during exposure to three stresses (metabolic inhibition, hyposmotic stress, and hyperkalemic cardioplegia) via an unknown mechanism. The sarcolemmal KATP channel in mouse myocytes is composed of SUR2A/Kir6.2 subunits in the ventricle and SUR1/Kir6.2 subunits in the atria. This study evaluated the effect of diazoxide on SUR1 knockout mice in an effort to determine diazoxide’s mechanism of action.

METHODS: Isolated ventricular myocytes from SUR1 knockout (KO) and wild type (WT) mice underwent volume measurement at baseline during exposure to Tyrode’s solution (Ctr) at 37ºC for 5 minutes, after 5 minutes of exposure to test solution (Ctr at 37ºC, St. Thomas hyperkalemic cardioplegia (CPG) at 9ºC, or CPG + 100 uM diazoxide (CPG + DZX) at 9ºC), and after 5 minutes of re-exposure to Ctr.

RESULTS: Data are represented as mean myocyte volume change normalized to baseline ±SEM (Figure 1). Myocyte volume remained unchanged in all groups during exposure to Ctr (time 5 min on Figure 1). Wild type myocytes demonstrated significant volume derangement during exposure to CPG that was prevented by DZX (time 10 min on Figure 1). SUR1 KO myocytes similarly swelled in response to CPG (6%); however, DZX had no effect on swelling. Re-exposure to Ctr is represented at time 15 minutes on Figure 1.

CONCLUSION: Diazoxide provides volume homeostasis via an SUR1-dependent pathway in ventricular myocytes. Because SUR1 is not expressed on the sarcolemma of mouse ventricular myocytes, these data support that the mechanism of action of DZX is via a non-sarcolemmal KATP channel location.

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Figure 1.
F7.  Myocardial Ischemia-Reperfusion Transcriptional Program in Normal and High Fat Diet Pigs

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Invited Discussant: Pedro J. del Nido, MD

OBJECTIVE: We showed that diet with high fat (H) was associated with higher myocardial necrosis after acute ischemia (1h)-reperfusion (2h) injury (I/R) in pigs vs. normal fat diet (N) (Osipov et al Circulation 2009;120 [Suppl 1];S20-S30). To gain insights into the myocardial expression profiling program regulated by dietary fat and I/R we performed microarray studies in no-ischemic area (C) and I/R area (R) of pigs hearts fed either N or H.

METHODS: Pigs were fed N/H (n = 7/group) for 30-days and then subjected to I/R as described (above ref). RNA were extracted and processed for microarray (same lot/time; total = 14) using Pig Genomic 3’ arrays (Affymetrix) containing ~ 24,000 transcripts. Microarrays were quality controlled (Quantile) then normalized (GCRMA). Statistically regulated transcripts (α = 0.05 with false discovery correction) (JMP Genomics) were analyzed for pathways and functions (Ingenuity Systems).

RESULTS: C vs. H showed discrete changes (37/24,000 or [0.15%]) in C hearts transcriptome program. Regulated transcripts were associated with lipid metabolism. ABCA1 (reverse cholesterol transporter important in plaque regression) was up 2-fold, and LXR/RXR (regulates cholesterol metabolism including ABCA1 repression) down 1.5-fold. In R, pigs fed H had 2.8-times more transcriptions regulated (925) than N (334), while 397 were regulated in both. Indicators of increased stress (C-FOS, HO-1, and HSPH1) were high in H animals only. Regulation of leukocyte adhesion molecules and pro-inflammatory pathways (IKKa, selectins L and E, calsequestrin, chemokine C-C and C-X-C [2 and 4]), citrate cycle and Diabetes II signal are also high and likely causal to a less favorable outcome after I/R in H. In contrast, N pigs had exclusively protective (riboflavin (antioxidant) and pro-angiogenic (HIF1α)) pathways up. Common regulated transcripts in R, both N and H pigs, included aquaporin (water channels), lipoprotein lipase, and glycogen synthase.

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CONCLUSION: N and H diet had a discrete effect in gene expression levels in C hearts that was deeply modified by I/R injury. While I/R injury regulates exclusively 1/3 of the number of transcripts in N diet pigs as compared to H diet, it is unclear if these differences have a causal relationship or are just reactive to higher necrosis in H animals. However, in H animals a significant number of regulated transcripts are pro-inflammatory genes indicating that hearts of H animals are primed to a more intense inflammatory response upon I/R injury.

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Invited Discussant: Carmelo A. Milano, MD

OBJECTIVE: Myocardial β-adrenergic receptor (βAR) signaling is severely impaired in chronic heart failure (CHF) due to receptor downregulation and desensitization. We have previously shown that cardiac βAR signaling can be normalized by long-term pulsatile left ventricular assist device (LVAD) support and that the primary mechanism appears to be a decrease in activity of G protein-coupled receptor kinase-2 (GRK2) which phosphorylates and desensitizes agonist-occupied βARs. The objective of this study is to determine if LV βAR signaling can be restored in CHF patients following continuous-flow (HeartMate II) LVAD support.

METHODS: Paired LV biopsies were obtained from patients at the time of HeartMate II LVAD implant (HF group) and at transplant (LVAD group). The mean duration of LVAD support was 6.8 ± 2.5 months. Myocardial βAR signaling was assessed by measuring sarcolemmal membrane adenylyl cyclase (AC) activity, total βAR density (Bmax), and GRK2 protein expression and activity. All patients underwent LVAD implant as a bridge to transplant and were NYHA class IV (n = 5). LV biopsy specimens from non-failing hearts (NF) were used as controls (n = 4).

RESULTS: Basal and isoproterenol (ISO)-stimulated AC activity was significantly lower in HF vs. NF (29.4 ± 1.6 vs. 59.5 ± 2.6 pmol cAMP/mg/min and 45.9 ± 2.5 vs. 122.2 ± 5.1, P < 0.02 for both) indicative of βAR uncoupling. Continuous-flow LVAD support restored basal and ISO-stimulated cyclase activity to levels similar to NF (52.8 ± 4.0 and 115.6 ± 6.8 pmol cAMP/mg/min, P < 0.02 vs. HF and P > 0.05 vs. NF). Bmax was decreased in HF vs. NF (39.4 ± 2.2 vs. 81.2 ± 3.4 fmol/mg, P < 0.01) and increased to near normal in the LVAD group (76.8 ± 4.0 fmol/mg, P < 0.01 vs. HF and P > 0.05 vs. NF). GRK2 expression was increased 2.6-fold in HF vs. NF and was similar to NF following LVAD support. Similarly, GRK2 activity was 3.2-fold greater in HF vs. NF and decreased to NF levels in the LVAD group.

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CONCLUSION: Myocardial βAR signaling can be restored to near normal following HeartMate II continuous-flow LVAD support. This is similar to previous reports for volume displacement, pulsatile LVADs. Decreased GRK2 activity appears to be an important mechanism for improved βAR signaling and indicates that normalization of the neurohormonal milieu associated with CHF is similar with continuous-flow and pulsatile LVADs, despite lesser LV volume unloading. Restoration of this critical signaling pathway has significant implications for myocardial recovery.
F9. Myocellular Maladaptation to Ischemic Reperfusion Injury Increases Propensity of Post-Operative Right Ventricular Dysfunction in Cyanotic Tetralogy of Fallot

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Invited Discussant: James Jaggers, MD

OBJECTIVE: Hypoxia induces cellular adaptive pathway including heat shock protein (HSP) family to tolerate ischemic stress. Despite that, the myocellular vulnerability to ischemic-reperfusion injury during Tetralogy of Fallot (TOF) surgery is increased in the presence of pre-operative cyanosis. We hypothesise that the pathogenesis of post-operative RV dysfunction in cyanotic TOF is related to their myocellular adaptive mechanism to ischemic-reperfusion injury during surgery.

METHODS: We used Western blot to quantify HSP-70i in resected RV tissues from 21 TOFs undergoing corrective repair: 11 cyanotic (Group C, saturation <90%) and 10 non-cyanotic (Group No-C). Systolic annular velocity, as measured by tissue Doppler echocardiography was used to determine pre- and post-operative (day 1) biventricular function and compared with 16 age-matched healthy children. Early outcome measures include post-operative (day 1) lactate, mixed venous oxygen saturation (SvO₂), ventilation and ICU time.

RESULTS: There was no demographic difference between Group C and No-C in term of age (median 23 vs. 16 months; p = 0.23), gender (7,64% vs. 5,50% males; p = 0.7), weight (9.7 vs. 10.5 kg; p = 1.0), and pre-operative BT shunt (7,60% vs. 2,20%; p = 0.08). Group C had higher haematocrit level (48 vs. 38%; p = 0.008), thicker septal wall (0.87 vs. 0.66 cm; p = 0.007), longer cross-clamp time (96 vs. 67.5 mins; p = 0.01), higher rate of transannular/RVOT patch (11,100% vs. 6,60%; p = 0.04), and higher Troponin-I (15.9 vs. 11.1pg/ml; p = 0.048). Pre-operative biventricular function was reduced in TOF compared to healthy children and further impaired on the RV post-operatively (3.78 vs. 4.08 cm/s in No-C; p = 0.97, 95% CI median difference –1.49,1.17) whilst LV function remained unchanged.
There were no difference in HSP-70i between group C and No-C during cross-clamp (median 4.0 vs. 3.2; p = 0.34, 95% CI median difference – 1.8,2.7). However, HSP-70i correlated with post-operative RV function in group C (rho = 0.74 p = 0.01) but not in No-C. There were no correlation between HSP-70i and age, haematocrit, pre-operative saturation, bypass or cross-clamp time, and troponin in either group. In group C, HSP-70i expression within the first 15 minutes of cross-clamp correlated (rho = 0.67, p = 0.036) with better $S_O_2$, but not with lactate or ventilation/ICU time.

CONCLUSION: HSP-70i expression during surgery influences early post-operative RV function and outcome in cyanotic TOF. Cellular malexpression of HSP in cyanotic TOF could lead to RV dysfunction early after corrective repair.
F10. **Lanthanum Carbonate, a Phosphate Binder, Inhibits Calcification of Implanted Aortic Allografts**

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**Invited Discussant:** Richard A. Hopkins, MD

**OBJECTIVE:** Calcification is the most major postoperative problem of implanted aortic allografts (AAs) and tends to appear more frequent and earlier in young recipients. It is reported that this early calcification of AAs in young recipients is partly due to physiological hyperphosphatemia of young recipients. Hyperphosphatemia and ectopic calcification are critical issues for uremic patients, and phosphate binders are used clinically. We hypothesized that phosphate binders, lanthanum carbonate (LC) and calcium carbonate (CC), inhibit calcification of implanted AAs in young recipients, and verified this hypothesis using rat experimental model.

**METHODS:** Aorta was harvested from a 4 week-old BN rat and implanted into subcutaneous space of a 4 week-old LEW rat. Recipient LEW rats were divided into 3 groups, group-N (g-N), group-L (g-L), and group-C (g-C). G-N was fed by normal diets (MF; Oriental Yeast Co., Ltd., Japan), g-L by MF plus 3% by weight of LC, g-C by MF plus 3% by weight of CC. All groups were composed of 9 rats. The implanted AAs were explanted 2 weeks after implantation. Calcification of AAs was evaluated qualitatively by Von Kossa stain and quantitatively by calcium (Ca) content assay using atomic absorption analysis method. Calcification score in Von-Kossa stain was defined as 0 (none), 1 (mild), 2 (moderate), and 3 (severe). Ca content was standardized dividing by dry graft weight. Blood samples were taken by right atrial puncture and centrifuged immediately to obtain serum. Serum Ca and phosphorus (Pi) concentration were measured. Data are expressed as mean ± SEM and comparisons between groups were made using t-test. A value of P < 0.05 was considered significant.

**RESULTS:** Calcification score of g-N, g-L, and g-C were 2.6 ± 0.2, 1.2 ± 0.4, and 0.8 ± 0.4, respectively. Ca content per 1 g dry graft of g-N, g-L, and g-C were 48.9 ± 8.7, 15.8 ± 3.4, and 8.9 ± 3.4 mg, respectively. Calcification was significantly inhibited by LC and CC. Serum Ca of g-N, g-L, and g-C were 11.5 ± 0.3, 12.2 ± 0.2, and 13.5 ± 0.4 mg/dL, respectively. Serum Ca
of g·C was significantly high than that of g·N and g·L. Serum Pi of g·N, g·L, and g·C were 15.4 ± 0.3, 12.5 ± 0.5, and 11.7 ± 0.4 mg/dL, respectively. Both LC and CC reduced serum Pi significantly.

**CONCLUSION:** Phosphate binders, LC and CC, inhibit calcification of implanted AAAs in young rat subdermal implantation model. CC induced significant hypercalcemia and LC has a more potential for clinical use.
F11. Vascular Endothelium Contributes to Tumor Tolerance Induction
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Invited Discussant: David R. Jones, MD

OBJECTIVE: Tumor vasculature is critical for malignant progression but has been considered mainly supportive in nature, providing nutrients and eliminating waist products in the tumor microenvironment. Our laboratory has recently described that antigen presentation by murine vascular endothelium can result in the generation of CD4+FoxP3+ regulatory T cells. This created the possibility that presentation of tumor associated antigen by vascular endothelium in vivo may contribute to tumor tolerance.

METHODS: In order to study this possibility we took advantage of a recently described model of orthotopic left single lung transplantation. This model is ideal for such studies since within several days of transplantation the donor derived hematopoietic cells are replaced by those of the recipient while all non-hematopoietic cells, such as vascular endothelium, remain of donor origin. Thus transplantation of a B6 MHC II- donor to a B6 wild-type recipient could be used to create a “chimeric organ” consisting of MHC II deficient vascular endothelial cells repopulated by wild-type hematopoietic cells. Such an experimental system results in the local disruption of CD4+ T cell-restricted antigen presentation by vascular endothelial cells in the lung. As the native right lung of the recipient remains unaffected it can thus act as an internal control in the same animal. We thus transplanted left B6 MHCII- or wild-type B6 lungs into a B6 recipients and injected B16 melanoma intravenously 1 week after transplantation.

RESULTS: Three weeks after tumor injection the lung tumor burden was calculated by weight and visual inspection. While the transplanted B6 lung supported significant growth of tumor (127.9 ± 41 mg), little tumor...
growth was detected in the transplanted MHCII- lung (13 ± 7.7 mg). Identical tumor growth was detectable in the native right lungs of both groups of recipients (165 ± 30 vs. 172 ± 57 mg). Evaluation of tumor infiltrating T cells demonstrated an increase in both CD4+ and CD8+ T cells with higher level of T cell activation, measured by CD69 expression, in lungs deficient in non-hematopoietic MHC II.

CONCLUSION: Based on our preliminary data we can conclude that expression of MHC Class II in the tumor microenvironment by vascular endothelium and other non-hematopoietic cells may be critical to T cell homeostasis and may play a role in the induction of tumor tolerance. Such mechanisms may need to be taken into account when combining anti-angiogenesis and immune based therapy.
F12. Gene Expression Profiles in Esophageal Adenocarcinoma Predict Survival Following Resection

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Invited Discussant: Chuong D. Hoang, MD

OBJECTIVE: Esophageal adenocarcinoma (EC) is rapidly rising in the western population. Despite aggressive treatment, the survival after esophagectomy is suboptimal. The current TNM classification has also been imprecise in prediction of survival in these patients. We hypothesized that gene expression pattern of EC will predict survival after surgical resection.

METHODS: We conducted a prospective NIH/NCI funded study to evaluate the prognostic significance of gene expression in patients with EC. Esophageal cancer tissues were stored at –80°C until analysis. All tissues were evaluated by a pathologist to ensure >70% tumor representation. Gene expression was analyzed with U-133 plus 2.0 arrays (Affymetrix) and data was filtered to exclude probesets with low variance in expression values. Patients were followed up in the thoracic surgery clinic. The association of gene expression and overall survival was analyzed using the tail strength statistic and Cox regression analysis. Semi-supervised methods using principal components were used to construct a gene signature. Leave-one-out cross-validation was used to develop a prognostic classifier.

RESULTS: We evaluated the results of 69 patients (N1: 34) who underwent esophagectomy for EC. During follow-up (median 34 months), 31 patients are alive. The median overall survival was 27 months (95% CI 22–NR). After filtering, 17,716 probe sets were used for survival analysis. The tail strength statistic for these probesets was 0.23 (95% CI: 0.22–0.24) indicating a significant association of the dataset as a whole with overall survival. Ninety-nine probesets were individually associated with overall survival (P < .001). We applied the semi-supervised methods to 71 of the 99 probesets, representing 70 genes using 3 principal components. Patients were classified into high and low risk groups, based on the gene signature, by leave-one-out cross-validation. High risk patients had a predicted median survival of 13 months while the median was not reached for the low risk groups (Figure, P = 0.000001). We are currently working on refining this gene signature.

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CONCLUSION: In this prospective study of gene expression in EC, global gene expression levels are significantly associated with overall survival after esophagectomy. Furthermore, individual genes can be successfully combined into a strongly predictive internally cross-validated gene signature. If further validated, these results may help direct further clinical trials in neoadjuvant and adjuvant therapies for Esophageal Adenocarcinoma.
F13. Optimized Intrapleural Cisplatin Chemotherapy with a Fibrin Carrier after Extrapleural Pneumonectomy: A Pre-Clinical Study

Isabelle Opitz1, Barbara V. Erne1, Seval Demirbas1, Alexander Jetter2, Burkhardt Seifert4, Rolf Stahel3, Walter Weder1*

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Invited Discussant: W. Roy Smythe, MD

OBJECTIVE: The aim of the study was to evaluate whether platinum concentration in the subpleural tissue and in the blood are optimized by local application of cisplatin loaded to a fibrin carrier compared to cisplatin-solution in a randomized setting of a pig model.

METHODS: After left-sided pneumonectomy plus parietal pleurectomy the pigs were randomly assigned to receive either 90 mg/m² cisplatin intrapleural solution (control, n = 5) or to receive 5mg cisplatin combined to a fibrin sealant applied on a predefined area of the chest wall (study group, n = 5). Platinum concentration in the plasma was determined at several early time points (1–24 h) and at 2 and 5 days following treatment. Chest wall biopsies were assessed for platinum concentration after 2 and 4 hours and at day 2 and 5 after cisplatin application. The level of total platinum was measured by means of inductively coupled plasma sector field mass spectrometric detection with a matrix-matched calibration procedure.

RESULTS: The dose- and surface-corrected mean concentration of cisplatin in the chest wall tissue 2 h after the application was 504.1 g/L in animals treated with cisplatin-fibrin (geometric coefficients of variation, CV, 88%), compared to 249.1 g/L (CV 261%) in the control group. Five days after the application, mean concentrations in the tissue were 72.5 g/L (CV 216%) and 21.8 g/L (CV 427%) in fibrin- and solution treated animals, respectively. In plasma, the dose- and application surface-corrected exposure towards cisplatin (area under the concentration-time curve from 0–5 d after surgery) was clearly and significantly lower with cisplatin-fibrin than with cisplatin-solution: 68.5 g/L*h (CV 28%) versus 755.8 g/L*h (CV 110%). This is also reflected by significantly reduced serum-creatinine
values in the study group in comparison to the control group (p = 0.02) as well as significantly better well-being scores for the animals treated with cisplatin-fibrin at each day of the observation (all p < 0.05).

CONCLUSION: Cisplatin tissue concentration after cisplatin-fibrin treatment was at least two fold higher at 2 h and 5 d while systemic cisplatin concentrations were significantly reduced. This finding offers a clear advantage since rate and severity of systemic adverse events can be reduced while local cytotoxic concentrations are at least maintained, what will be soon evaluated in a phase-I study.
F14. Deletion of Tissue Plasminogen Activator Prevents Lung Ischemia-Reperfusion Injury via Inhibition of Neutrophil Extravasation

Yunge Zhao, Abbas Emaminia, Ashish K. Sharma, John Steidle, Gorav Ailawadi, Irving L. Kron*, Christine L. Lau*
Surgery, University of Virginia, Charlottesville, VA, USA.

Invited Discussant: Marc de Perrot, MD, MSc

OBJECTIVE: Ischemia-reperfusion injury (IRI) continues to be the most common cause for early morbidity following lung transplantation. In acute lung injury processes including IRI, extravascular fibrin has been shown to activate inflammation and promote lung dysfunction. We hypothesized that mice lacking the tissue plasminogen activator gene (tPA KO), which are less efficient at fibrin degradation, would experience increased lung IRI.

METHODS: tPA KO (n = 9) and wild-type (C57BL/6) (n = 9) mice underwent in-situ left lung ischemia for 1 hour followed by reperfusion for 2 hours. Sham group (n = 9) were used as control. Lung function/injury was assessed by an ex vivo buffer-perfused isolated lung system. The cellular infiltration was evaluated by immunohistochemical staining and densitometric analysis. Lung vascular permeability was determined using Evan's Blue injection (n = 12). The activities of matrix metalloproteinase-2 (MMP-2), MMP-9, urokinase plasminogen activator and tPA were detected by gelatin- and fibrin- zymography, respectively.

RESULTS: Compared to wild-type mice, tPA KO mice were significantly protected from IRI with lower pulmonary artery pressures (8.15 ± 1.07 vs. 12.53 ± 1.03, P = 0.006), increased lung compliance (5.75 ± 0.97 vs. 2.55 ± 0.20, P < 0.0001), and lower airway resistance (1.01 ± 0.15 vs. 2.24 ± 0.28, P = 0.005). Compared to sham group, tPA KO mice showed no significant changes in lung compliance (P = 0.419), but significant differences in pulmonary artery pressures (P = 0.001) and airway resistance (P = 0.010). By histology and densitometric analysis, neutrophil (P < 0.001) (but not macrophage) extravasation from blood vessels was clearly blocked in tPA KO compared to wild-type mice (Figure 1). tPA KO blocked neutrophil infiltration by decreasing lung vascular permeability (tPA KO IR vs. C57BL/6 IR, P = 0.040) through inhibition of the expression of MMP-9 and platelet endothelial cell adhesion molecule-1 (CD31) expression in the endothelial cells.

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CONCLUSION: Deletion of tPA resulted in significantly attenuated lung ischemia-reperfusion injury. The mechanism may be explained via inhibition of MMP-9 and endothelial cell adhesion molecules. These results have clinical implications as targeted therapies directed against tPA, or tPA combined with MMP-9 and/or CD31, and may be novel strategies to protect against IRI.
F15. The Chemokine/Chemokine Receptor Pair, CXCL12/CXCR4 Is a Key Pathway in Lung Adenocarcinoma Tumor Cells Tumor Associated Fibroblasts Crosstalk

Ori Wald¹, Uzi Izhar¹, Gail Amir², Sophie Kirshberg³, Zippora Shlomai³, Amnon Peled², Oz M. Shapira¹*

¹. Cardiothoracic Surgery, Hadassah University Hospital, Jerusalem, Israel. 2. Goldyne Savad Gene Therapy Institute, Hadassah University Hospital, Jerusalem, Israel. 3. Laboratory for Surgical Research, Hadassah University Hospital, Jerusalem, Israel. 4. Pathology, Hadassah University Hospital, Jerusalem, Israel.

Invited Discussant: Steven J. Mentzer, MD

OBJECTIVE: Microenvironment-derived signals greatly influence carcinogenesis. Chemokine/chemokine receptor interaction communicates such signals. Cancer associated fibroblasts (CAF) are predominant in lung adenocarcinoma, but their role in tumor growth and metastatic spread is not well defined. Expression of CXCR4 by lung adenocarcinoma tumor cells has been previously linked to accelerated metastatic spread and reduced survival. However, the role of CXCL12/CXCR4 axis in tumor proliferation has not been studied. We sought to characterize the role of CXCL12/CXCR4 axis in the crosstalk between epithelial tumor cell and CAFs.

METHODS: Four fibroblast primary cell cultures and two epithelial primary cell cultures were derived from lung adenocarcinoma tumor tissue removed during surgery. The expression of an array of tumor promoting factors and receptors by CAFs and tumor epithelial cells was studied. Cell cycle and colony assays were performed to assess cancer cell responsiveness.

RESULTS: Epithelial cell and CAF identification was confirmed morphologically and by cytokeratin and fibronectin immuno-fluorescent staining (Panel A, B). PCR and ELISA assays showed expression of the pro-angiogenic chemokines CCL2, CXCL8, CXCL12 and the growth factor VEGF by all CAFs cell lines (Panel C). Immunohistochemistry revealed that CXCL12 expressing CAFs were abundant in lung adenocarcinoma. PCR and flow cytometry analysis confirmed that lung adenocarcinoma epithelial tumor cells strongly express CXCR4 (Panel D). Stimulation of these cells with increasing concentrations of CXCL12 promoted cell cycle propagation as measured by BRDU incorporation. In addition CXCL12 increased the colony forming capacity of cancer cells (Panel E).

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CONCLUSION: Lung-adenocarcinoma-derived CAFs express an array of pro-angiogenetic factors. Among these factors CXCL12 is marked as a proliferation promoting chemokine, acting directly on tumor epithelial cells CXCR4. Our findings suggest that the interaction between CAFs and epithelial tumor cells and the CXCL12/CXCR4 axis have a major role in tumor proliferation and mark CXCL12/CXCR4 axis as a target for immune-intervention in lung adenocarcinoma.
F16. WNT Pathway Activation Predicts Increased Risk of Tumor Recurrence in Patients with Stage I Non-Small Cell Lung Cancer

Mark Shapiro¹, Gal Akiri³, Cynthia S. Chin², Juan P. Wisnivesky⁴, Todd S. Weiser², Scott J. Swanson⁵*, Stuart A. Aaronson³

¹. Surgery, The Mount Sinai Medical Center, New York, NY, USA. ². Cardiothoracic Surgery, The Mount Sinai Medical Center, New York, NY, USA. ³. Oncological Sciences, The Mount Sinai Medical Center, New York, NY, USA. ⁴. Medicine, The Mount Sinai Medical Center, New York, NY, USA. ⁵. Cardiothoracic Surgery, Brigham and Women’s Hospital, Boston, MA, USA.

Invited Discussant: Richard J. Battafarano, MD, PhD

OBJECTIVE: Aberrant WNT signaling activation by genetic alterations of components of this pathway plays an important role in the development of a wide variety of tumor types. Recent studies have shown that WNT pathway activation in several tumor types including non-small cell lung cancer (NSCLC) cell lines occurs at high frequency by an autocrine mechanism. There have been reports that, due to increased WNT signaling, primary NSCLCs can exhibit increased levels of cytosolic or nuclear β-catenin as visualized by increased immunostaining of tumor tissues. We developed a quantitative means of identifying WNT activated primary tumors and utilized this biochemical approach to determine the frequency of WNT pathway activation in patients with stage I NSCLC and assess its effect on lung cancer recurrence.

METHODS: 57 patients treated with surgical resection for stage I NSCLC between June 2006 and May 2008 were selected from an IRB-approved database linked to the cancer tissue biorepository containing fresh frozen tumor as well as a normal lung tissue specimens linked to each patient. A glutathione-S-transferase (GST) pull-down assay combined with immunoblot analysis was used to assess the levels of uncomplexed and total β-catenin in tumors with activated WNT signaling, and we compared cancer recurrence rates in WNT pathway positive and negative tumors.

RESULTS: 38.6% (n = 22) of tumors were scored as WNT positive with only one exhibiting a β-catenin oncogenic mutation. Thus, the great majority of WNT activated primary tumors, as with NSCLC tumor lines, likely exhibit WNT autocrine activation. Patients with WNT positive tumors experienced a significantly higher rate of cancer recurrence than those
whose tumors were WNT negative (27.3%, n = 6 vs. 5.7%, n = 2) (Figure).
Moreover, there were 5 patients with distal tumor recurrence in the WNT positive group compared to 1 in the other group (22.7% vs. 2.9%, p = 0.036).

CONCLUSION: Our study establishes a role for WNT pathway activation in a substantial fraction of primary human NSCLCs. Moreover, increased levels of WNT pathway activation were associated with a higher rate of cancer recurrence in patients with Stage I NSCLC. These findings suggest that WNT activation reflects a more aggressive tumor phenotype and identifies patients who may benefit from more aggressive therapy in addition to resection.
F17. Development of a Navigation System for Segmentectomy Using Infrared Thoracoscopy
Noriyuki Misaki, Sung Soo Chang, Hitoshi Igai, Shintaro Tarumi, Masashi Gotoh, Hiroyasu Yokomise
General Thoracic Surgery, Breast and Endocrinological Surgery, Kagawa University, Kagawa, Japan.
Invited Discussant: Ralph A. Schmid, MD

OBJECTIVE: We have reported the feasibility of identifying adjacent lung segments using infrared thoracoscopy (IRT) after intravenous injection of indocyanine green (ICG) in an animal model (JTCVS 2009;138:613–8). In the present study, we attempted a clinical trial of segmentectomy using the same method, and investigated its applicability and any associated problems.

METHODS: IRT uses two wavelengths of infrared light: 805 and 940 nm. Lung tissue without any modification reflects both wavelengths, and is displayed as white. ICG absorbs light at 805 nm, and therefore lung tissue containing ICG reflects light at only 940 nm, and is displayed as blue. A total of 8 patients with lung lesions were investigated (5 with primary lung cancer, 2 with metastatic lung cancer, and 1 with infection). All were scheduled to undergo segmentectomy, and had been confirmed to have no allergy to iodine or ICG. Informed consent was obtained from all patients. We identified the dominant pulmonary artery (PA) supplying the target segment using three-dimensional computed tomography. The dominant pulmonary artery of the target segment was ligated, and after we had observed the lung using IRT with ICG (3.0mg/kg), and marked the white-to-blue transitional zone by electrocautery, we performed segmentectomy. Approval for this study was provided by the institutional review board of Kagawa University Hospital.

RESULTS: Average operation time was 150 ± 62.1 min, and bleeding volume was 68.8 ± 30.5 cc. Under IRT, the area with a normal blood supply became stained blue 13 s after injection of ICG. Maximum staining intensity was attained 28 s after dye injection, and the observation duration was 3 min 30 s. A well-defined color zonation was observed in all patients. We had enough time to mark it. No complications attributable to IRT with ICG were encountered. In cases of emphysema, the lung tissue tended to stain less deeply than normal, and in cases involving large tumors the feasible staining observation period tended to become shorter.
CONCLUSION: IRT with ICG makes it possible to identify the target lung segment very easily and quickly without the need for inflation. This method will be especially useful for cases associated with severe emphysema or when surgery offers only a limited view, as is the case with video-assisted thoracic surgery.
F18. Endothelin-1 Mediates Bronchiolitis Obliterans in Lung Transplant Recipients
Mohamed Salama², Olena Andrukhova², Peter Jaksch¹, Shahrokh Taghavi¹, Walter Klepetko¹*, Seyedhossien Aharinejad¹
¹. Departments of Cardiothoracic Surgery, Medical University of Vienna, Vienna, Austria. 2. Cardiovascular Research, Center of Anatomy and Cell Biology, Medical University of Vienna, Vienna, Austria.

Invited Discussant: Daniel Kreisel, MD, PhD

OBJECTIVE: Bronchiolitis obliterans (BO) is a severe complication limiting the long-term survival in lung transplantation (LTX) and there exists no cure for this disease. BO is characterized by progressive fibrosis and obliteration of airways; however, the mechanisms leading to BO are not well understood. Endothelin-1 (ET-1) is a potent mitogenic and pro-fibrotic peptide produced by pulmonary vascular endothelial cells that reportedly plays a role in the pathophysiology of lung allograft dysfunction. Whether ET-1 mediates BO is unknown.

METHODS: Serum and brochoalveolar lavage (BAL) were obtained from 30 LTX patients with and 30 without BO. ET-1 concentrations were examined by ELISA in both serum and BAL. BO syndrome was diagnosed and scored based on first second forced expiratory volume (FEV1) values according to International Society for Heart and Lung Transplantation guidelines and verified histologically in transbronchial biopsies.

RESULTS: Serum ET-1 concentrations were significantly elevated in patients who developed BO (p < 0.0001) compared to those who did not. Moreover, BO patients had significantly increased ET-1 concentrations in their BAL (p = 0.04) compared to no-BO patients. Serum (rs = 0.72; P = 0.0015) and BAL (rs = 0.58; P = 0.0035) ET-1 concentrations correlated significantly with the BO diagnosis in the patient cohort examined.

CONCLUSION: This study indicates that elevated ET-1 is strongly associated with BO where it may contribute to the BO-related pathology by induction of airway fibrosis and altering pulmonary vascular resistance. Therefore, assessment of circulating and BAL ET-1 concentrations might be beneficial in BO diagnosis and monitoring. Furthermore, targeted ET-1 antagonism could be a novel treatment strategy in BO patients.

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F19. Trans-Lymphatic Chemotherapy Controls Lymphatic Metastasis and Prolongs Survival in an Orthotopic Lung Cancer Model

Jiang Liu1, Ming Li1, Michael R. Johnston2*
1. Princess Margaret Hospital/University Health Network, Toronto, ON, Canada. 2. Thoracic Surgery, Queen Elizabeth II Health Sciences Centre, Dalhousie University, Halifax, NS, Canada.

Invited Discussant: Michael Liptay, MD

OBJECTIVE: Lymphatic metastasis is a critical prognostic factor in lung cancer and poses a significant challenge to treat. We developed an implantable trans-lymphatic drug delivery system which can effectively control lymphatic tumor metastasis. In the present study we investigated the survival benefit of the system as an adjuvant to surgery in an orthotopic lung cancer model.

METHODS: A gelatin sponge device containing polylactide-co-glicolide paclitaxel (PLGA-PTX) microspheres was synthesized to contain 20 mg PLGA-PTX microparticles (7% drug loading, 1.4 mg PTX/each). A placebo control sponge containing equivalent amounts of blank PLGA microspheres was similarly made. The lung cancer model was established in nude rats by endobronchial implantation of one million NCI-H460 human lung cancer cells into the left lung via a tracheotomy. Fourteen days following tumor implantation, the lung was resected by left pneumonectomy. Animals were treated intraoperatively with either intrapleural implantation of a PLGA-PTX gelatin sponge (n = 16), or a placebo sponge (n = 15). Postoperative survival time and tumor recurrences were measured. The incidence of tumor recurrence at the resection margin, lymph node metastasis and lymph node weight were compared.

RESULTS: Three control animals, but no treated animals, developed tumor at the resection margin. All control animals developed tumor recurrence in mediastinal lymph nodes. 4 of 15 (27%) of treated animals developed mediastinal lymph node metastasis. Mean lymph node weight, reflecting the tumor burden, was 0.35 ± 0.15 g (n = 16) and 0.13 ± 0.16 g (n = 4) for control and treated groups respectively (p < 0.01). PLGA-PTX microspheres were evident microscopically in the lymph nodes. The mean survival time (mean ± SE) for control animals and treated animals was 27.4 ± 0.83 days and 42 ± 2.87 days respectively (p < 0.01).

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CONCLUSION: This novel trans-lymphatic drug delivery system can effectively control lymphatic metastasis and local tumor recurrence. It also prolongs lung cancer survival in an adjuvant orthotopic lung cancer model. The system offers a unique therapeutic modality as an intraoperative adjuvant to lung cancer resection.
OBJECTIVE: The majority of solid malignancies including malignant pleural mesothelioma (MPM) are refractory to TRAIL-mediated cytotoxicity. SAHA has mild antitumor activity against MPM in a phase I clinical trial. This study is designed to evaluate the cytotoxicity of the SAHA+TRAIL combination on a panel of 10 MPM cells and two normal cells (primary fibroblasts and endothelial cells) in vitro.

METHODS: Cytotoxicity and apoptosis mediated by TRAIL (0–100 ng/ml), SAHA (0.5–5.0 microM) or concurrent SAHA+TRAIL combinations are evaluated by MTT assay and AnnexinV-FITC. NF-κB transcriptional activity is measured by luciferase-based reporter plasmid and lucinometer. Protein expression is evaluated by western blot.

RESULTS: MPM cells exhibit a wide range of intrinsic susceptibility to TRAIL (2/10 sensitive: IC50 <50 ng/ml, 5/10 mildly sensitive: IC50: 150 to 2500 ng/ml, 3/10 resistant: IC50: infinity). Profound enhancement of cytotoxicity is observed following treatment with SAHA+TRAIL combinations in 7 MPM cells that exhibit some degrees of TRAIL sensitivity as indicated by 2-fold to >100-fold reduction of TRAIL IC50's (Figure 1a). Supra-additive induction of apoptosis is observed in representative MPM cells treated with SAHA+TRAIL (Figure 1b). Combination-induced cytotoxicity and apoptosis is equally abrogated by either selective caspase 8 or caspase 9 inhibitor and thus implicating the essential role of the mitochondria-driven death signaling cascade. SAHA-dependent strong activation of NF-κB is completely abrogated by TRAIL and this is paralleled with caspase 8-mediated cleavage of RIP (receptor-interacting protein) to form the NF-κB dominant negative cRIP fragment in combination-treated cells. Overexpression of the caspase 8 resistant/uncleavable RIP mutant results

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in maintenance of high NF-κB transcriptional activity and less cytotoxicity in the H513 MPM cells treated with SAHA+TRAIL. Most importantly, this combination is not toxic to normal cells.

**CONCLUSION:** SAHA+TRAIL combination induces profound cytotoxic effect in MPM cells that is dependent on the intrinsic death signaling pathway, caspase 8-mediated RIP processing and down-modulation of NF-κB transcriptional activity. Further investigation is ongoing to develop molecular strategy to promote SAHA+TRAIL cytotoxicity in TRAIL-resistant MPM cells.
8:45 a.m.  

PLENARY SCIENTIFIC SESSION  

Hall C, Metro Toronto Convention Centre  

(8 minutes presentation, 12 minutes discussion)  

Moderators:  

G. Alec Patterson, MD  
Thoralf M. Sundt, III, MD  

28. Surgical Management and Outcome of Patients with Chronic Thromboembolic Pulmonary Hypertension: Results from a European Prospective Registry  

Eckhard Mayer¹, Jaroslav Lindner², Andrea M. D’Armini³, David Jenkins⁴, Walter Klepetko⁵*, Philippe G. Dartevelle⁶*  

¹. Department of Thoracic Surgery, Kerckhoff Lung Center, Bad Nauheim, Germany. ². Department of Cardiovascular Surgery, Charles University, Prague, Czech Republic. ³. Division of Cardiac Surgery, San Matteo Hospital, University of Pavia, Pavia, Italy. ⁴. Department of Cardiothoracic Surgery, Papworth Hospital, Cambridge, United Kingdom. ⁵. Department of Cardiothoracic Surgery, Medical University of Vienna, Vienna, Austria. ⁶. Department of Thoracic and Vascular Surgery, Marie Lannelongue Hospital, Le Plessis Robinson, Paris-Sud University, Le Plessis Robinson, France.  

Invited Discussant:  
Thoralf M. Sundt, III, MD  

OBJECTIVE: Pulmonary endarterectomy (PEA) is the treatment of choice for CTEPH patients who are considered operable by an experienced surgeon and interdisciplinary team. Normal or near-normal cardiopulmonary function and exercise capacity can be restored by surgery. The current real-world management and early postoperative outcome of patients undergoing PEA are described from the European CTEPH registry.  

METHODS: The prospective registry was designed to include newly diagnosed (≤ 6 months) consecutive patients with CTEPH, from February 2007 until January 2009. Diagnosis was confirmed by right heart catheterization, ventilation-perfusion lung scintigraphy, computerized tomography and/or pulmonary angiography.  

RESULTS: 679 patients with CTEPH were registered from 26 European and 1 Canadian centers. Based on surgeon’s assessment, 428 patients (63.5%) were considered operable and 246 (36.5%) non-operable (5 patients missing data). Eventually, 381 patients (56.1%) underwent surgery, 38 refused, 7 died prior to surgery; the median [range] time from CTEPH diagnosis to surgery was 78 days [0–588 days].

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Prior to PEA, 99 (26.0%) of the patients were on PH-specific mono therapy (phosphodiesterase type V inhibitor, endothelin receptor antagonist or prostacyclin analogue), and 6 (1.6%) were on dual therapy. PEA was considered “complete” by the operating surgeon in 347 out of 371 evaluable patients (93.5%). The median [range] duration of circulatory arrest was 36 minutes [0–108 min] and median duration of postoperative mechanical ventilation was 1.4 days [0–45 days].

Operative mortality was 4.5%. Perioperative complications occurred in 182 patients (48.7%): persistent PH (mPAP >25 mmHg or echocardiographic systolic PAP >40 mmHg) at the end of intensive care (n = 63, 16.8%), pulmonary reperfusion edema (n = 37, 9.9%), neurological (n = 41 (2 irreversible), 11.0%), bleeding (n = 38, 10.2%) problems or pericardial effusion (n = 31, 8.3%).

Surgical results are summarized in the table for all operated patients, patients with persistent PH and patients who died perioperatively.

**CONCLUSION:** The management of CTEPH patients in the participating specialized centers suggests high quality of care as indicated by low operative mortality and good early results. Future follow-up data will support critical decision-making regarding operability and treatment options for these patients.
29. What Is the Role of Complex Coronary Anatomy in Modern Bypass Surgery? Lessons Learned from The SYNTAX Trial after Two Years

Friedrich W. Mohr1,*, Ardawan J. Rastan1, Patrick W. Serruys², A. Pieter Kappetein2, Elisabeth Stahle3, David R. Holmes4, Jose L. Pomer5, Stephen Westaby6,*, Katrin Leadley7, Keith D. Dawkins7, Michael J. Mack8,*

1. Cardiac Surgery, Heart Center Leipzig, Leipzig, Germany. 2. Erasmus University Medical Center Rotterdam, Rotterdam, Netherlands. 3. University Hospital Uppsala, Uppsala, Sweden. 4. Mayo Clinic, Rochester, MN, USA. 5. Hospital Clinico Y Provincial, Barcelona, Spain. 6. John Radcliffe Infirmary Oxford II, Oxford, United Kingdom. 7. Boston Scientific Corporation, Natick, MA, USA. 8. Medical City Hospital, Dallas, TX, USA.

Invited Discussant: Sheng-shou Hu, MD

OBJECTIVE: SYNTAX is a prospective, multicenter trial comparing the outcome of CABG with PCI in patients with 3-vessel and/or left main disease. Complexity of coronary arterial anatomy and disease was analyzed by a scoring system, the SYNTAX Score. The SYNTAX Score is a composite of 9 characteristics (e.g., number and location of lesions, tortuosity, calcification, etc.) of each significant lesion. This study investigates whether the complexity of coronary disease as defined by the SYNTAX Score impacts CABG outcomes.

METHODS: Patients from the CABG randomized (RCT) and registry arms of the trial were included in the analysis. Patients were stratified according to their SYNTAX Score into 3 groups: low (0–22), medium (23–32), and high (33 and above) complexity. Clinical outcomes at 12–24 months post allocation were determined for each group.

RESULTS: Of the 3075 patients enrolled in SYNTAX, 1541 underwent CABG (897 and 644 in the randomized and registry cohorts, respectively). Patients enrolled in the registry had more complex disease than those in the RCT (mean total SYNTAX Score ± SD: registry, 37.8 ± 13.3; RCT, 29.1 ± 11.4). At 30 days, mortality was 0.9% (combined groups: 14/1507; RCT: 1.2% [10/866]; registry: 0.6% [4/641]). Major adverse cardiac and cerebrovascular events (MACCE) at 30 days was 4.4% (combined groups: 67/1507; RCT: 5.2% [45/866]; registry: 3.4% [22/641]). At 12 months, compared with RCT patients, patients enrolled in the registry had lower mortality (2.5% [16/633] vs. 3.5% [30/849], registry vs. RCT) and symptomatic post procedure graft occlusion (2.0% [12/612] vs. 3.4% [27/787], registry vs. RCT); however, cerebrovascular accident rates were comparable (2.2% [14/633] vs. 2.2 [19/849], registry vs. RCT). The 24-month results of the pooled

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group (RCT and registry arms) are contained in Table 1. Patients with low, medium, and high SYNTAX Scores had similar overall MACCE outcomes and no significant differences in safety outcomes.

Table 1: CABG Outcomes According to SYNTAX Score

<table>
<thead>
<tr>
<th>Syntax score</th>
<th>0–22</th>
<th>23–32</th>
<th>≥33</th>
</tr>
</thead>
<tbody>
<tr>
<td>MACCE, % (n)</td>
<td>14.8 (222)</td>
<td>15.4 (51)</td>
<td>13.9 (62)</td>
</tr>
<tr>
<td>Death, % (n)</td>
<td>4.9 (73)</td>
<td>4.5 (15)</td>
<td>4.7 (21)</td>
</tr>
<tr>
<td>CVA, % (n)</td>
<td>2.9 (43)</td>
<td>3.1 (10)</td>
<td>2.9 (13)</td>
</tr>
<tr>
<td>MI, % (n)</td>
<td>3.2 (48)</td>
<td>2.7 (9)</td>
<td>2.7 (12)</td>
</tr>
<tr>
<td>Revascularization, % (n)</td>
<td>7 (102)</td>
<td>7.4 (24)</td>
<td>7.1 (31)</td>
</tr>
</tbody>
</table>

CVA = cerebrovascular accident; MACCE = major adverse cardiovascular and cerebrovascular events; MI = myocardial infarction.

**CONCLUSION:** Outcomes of CABG were excellent independent of the SYNTAX Score. Higher degrees of coronary disease complexity did not impact CABG outcomes, in contrast to PCI. Repeat revascularisation, CVA, and MI rarely occurred in the second year.
OBJECTIVE: To question the validity of surgical aortic valvotomy for congenital bicuspid aortic valve stenosis (CBAS), a retrospective study was undertaken to determine the long-term survival, the incidence of valve restenosis (AS) or insufficiency (AI), and freedom of reoperation or valve replacement.

METHODS: From January 1990 through 2009, 85 consecutive children diagnosed with CBAS underwent open aortic valvotomy at our institution. Ages at operation ranged from 2 month to 18 years (mean 7.1 ± 5.7 years). The mean peak aortic valve gradient was 82 ± 21 mm Hg. The mean follow-up of these patients has been 9.9 ± 5.9 years (range, 6 months to 18 years).

RESULTS: There was no hospital mortality, but two late deaths occurred. Kaplan-Meier 18-year survival was 97%. Twenty-nine patients (34%) developed moderate or severe AI and 26 (31%) of them required aortic valve replacement. Thirty-six patients required reoperation (mean time, 8.6 years) after initial surgical valvotomy (10 for restenosis, 4 for severe late AI, and 22 for combined lesions). In these 36 pts, Ross AVR (n = 26), mechanical prosthesis (n = 7), and repeat open commissurotomy (n = 3) are performed. Kaplan-Meier 10- and 18-year freedom from any aortic reoperation or reintervention is 48 and 33%, respectively; 10- and 18-year freedom from aortic valve replacement is 55 and 40%, respectively. Echocardiography in survivors who have not required reintervention (n = 43) revealed a Doppler peak instantaneous systolic gradient of 41.2 ± 15.5 mm Hg and mild or less AI in 40 patients and moderate AI in 3 patients.

CONCLUSION: Surgical aortic valvotomy is a simple and effective procedure for CBAS with excellent long-term results. Reinterventions are frequent, but AVR can be delayed until the implantation of an adult-sized prosthesis is frequently possible. Late survival is excellent. Open surgical valvotomy continues to be the treatment of choice for children with CBAS.
31. Robotic-Assisted Thymectomy – Lessons Learned from 160 Cases

Jens C. Ruckert, Marc Swierzy, Mahmoud Ismail
Department of General, Visceral, Vascular and Thoracic Surgery,
Universitätsmedizin Berlin – Charité Campus Mitte, Berlin, Germany.

Invited Discussant: Franca M.A. Melfi, MD

OBJECTIVE: Mediastinal surgery is the most important part of treatment for mediastinal tumors especially for thymic tumors in the anterior mediastinum. A radical thymectomy (Thx) is crucial for comprehensive treatment of myasthenia gravis (MG). Minimally-invasive operation techniques are increasingly used for this indication. The latest development of robotic thoracoscopic surgery has been described to be successful for mediastinal surgery. To establish robotic technique as a standard, the results of high volume centers and comparision with traditional surgery is mandatory. The largest series of robotic thymectomy is presented.

METHODS: All mediastinal robotic approaches were prospectively evaluated and compared to the conventional or thoracoscopic techniques. A prospective study analyzed 160 consecutive robotic-assisted thoracoscopic thymectomies (rThx) (1/2003–01/2009, 90 female, mean age 40.7 ± 17.3 years, range 4–83) by a 3-trocar left-sided technique using the da Vinci robotic system. Results of rThx were compared to the previous consecutive series of 80 thoracoscopic non-robotic Thx (tThx). All patients with MG (141/160, 88%) were analyzed for quantification of improvement of MG and postoperative morbidity according to the Myasthenia Gravis Foundation of America classification. For patients with thymoma survival data were compared to median sternotomy and analyzed as to revised operation technique.

RESULTS: 160 robotic mediastinal procedures were performed (116 rThx for non-thymoma MG). With zero mortality the overall postoperative morbidity rate was 1.2% (2/160). Complete rThx required 181.4 ± 49 minutes (90–360). The conversion rate was 1.8% (3/160, because of thymoma). The dominant histological finding was follicular hyperplasia of the thymus (67/141, 47.5%). The cumulative complete stable remission rate of MG was 52% with a median follow up of 22 months (0–70). This was significantly better compared to tThx. For thymoma there was no difference of rThx as compared to sternotomy.
CONCLUSION: There is an improved outcome for MG after 160 rThx as compared to 80 tThx. Mediastinal dissection due to robotic technology allows for adequate radicality in different anatomical mediastinal configurations. The results of the largest single series of MG and thymoma are promising.

10:05 a.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK

Exhibit Hall A & B
10:40 a.m.  PLENARY SCIENTIFIC SESSION
Hall C, Metro Toronto Convention Centre
(8 minutes presentation, 12 minutes discussion)

Moderators:  G. Alec Patterson, MD
Thoralf M. Sundt, III, MD

32. Moderate Control of Hyperglycemia Is Superior to Tight Control in Patients Undergoing Coronary Artery Bypass Grafting
Castigliano M. Bhamidipati, Damien J. LaPar*, John A. Kern*, James J. Gangemi, Irving L. Kron*, Gorav Ailawadi
Division of Thoracic and Cardiovascular Surgery, Department of Surgery, University of Virginia School of Medicine, Charlottesville, VA, USA.

Invited Discussant: Anthony P. Furnary, MD

OBJECTIVE: Hyperglycemia has been associated with detrimental outcomes in surgery. Although there has been a consensus in cardiac surgery that tight control of perioperative hyperglycemia (glucose <120mg/dl) is beneficial, recent studies in sepsis have suggested that permissive hyperglycemia may be superior. We sought to determine if tight control or moderate control of hyperglycemia is optimal in patients undergoing coronary artery bypass grafting (CABG).

METHODS: From 1995–2008, a total of 4658 patients (Primary = 4433 [95.17%], Re-operative = 225 [4.83%]) with known diabetes or peri-operative hyperglycemia (perioperative serum glucose >126 mg/dl and/or preoperative HbA1c ≥8) underwent isolated CABG at our institution. Patients were stratified into three postoperative glycemic control groups: Group I (Tight control, ≤126mg/dl), Group II (Moderate control, 127–179mg/dl), and Group III (Liberal control, ≥180 mg/dl). Pre-operative risk factors, glycemic management, and post-operative outcomes were analyzed.

RESULTS: Overall operative mortality was 2.5% (119/465) and major complication rate was 12.5% (581/4658). Compared to the tight and liberal glycemic control groups, the moderate control group had the lowest mortality (p = 0.016, Figure 1). In addition, the moderate control group had a lower incidence of major complications, prolonged ventilation (p < 0.001), and postoperative sepsis (I: 3.73% [5/134], II: 0.97% [27/2785], and III: 1.44% [25/1739], p = 0.01). Sternal wound infection and stroke were equivalent across the groups (p = 0.618 and p = 0.6, respectively). More patients in the tight control group had preoperative renal failure (I: 16.42%...
Figure 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Tight (n=134)</th>
<th>Moderate (n=2785)</th>
<th>Liberal (n=1739)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>STS Predicted Risk Mortality</td>
<td>0.22 ± 1.127</td>
<td>0.86 ± 1.082</td>
<td>2.33 ± 3.234</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Emergent</td>
<td>7 (5.22%)</td>
<td>52 (1.87%)</td>
<td>55 (3.10%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Prolonged Ventilation</td>
<td>22 (16.42%)</td>
<td>166 (5.90%)</td>
<td>108 (6.21%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Major Complications</td>
<td>26 (19.4%)</td>
<td>308 (11.1%)</td>
<td>247 (14.2%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Operative Mortality</td>
<td>4 (2.99%)</td>
<td>56 (2.01%)</td>
<td>59 (3.39%)</td>
<td>0.016</td>
</tr>
</tbody>
</table>

[22/134], and II: 8.33% [232/2785], p = 0.001, and underwent emergent operations (I: 5.22% [7/134], and II: 1.87% [52/2785], p = 0.007) compared to the moderate control group. Despite these apparent differences, the STS predicted risk of mortality was lower in the tight compared to the moderate control group (p < 0.001).

CONCLUSION: Moderate glycemic control or permissive hyperglycemia is superior to tight glycemic control with decreased mortality and major complications, and may be the ideal strategy in patients undergoing coronary artery bypass operations.
33. Does Pre-Operative Sildenafil Protect Against Pulmonary Related Complications Following Cardiopulmonary Bypass? A Randomized Trial in Children Undergoing Cardiac Surgical Repair
Antony Vassalos¹, Edward W. Peng¹, David Young³, Kenneth J. MacArthur¹, James Pollock¹, Fiona Lyall², Mark Danton¹
1. Department of Cardiac Surgery, Royal Hospital for Sick Children, Glasgow, United Kingdom. 2. Maternal & Fetal Medicine, Institute of Medical Genetics Yorkhill Hospitals, Glasgow, United Kingdom. 3. Department of Statistics and Modelling Science, University of Strathclyde, Glasgow, United Kingdom.

Invited Discussant: Paul M. Kirshbom, MD

OBJECTIVE: To investigate the acute effects of pre-operative sildenafil on haemodynamics, contractility, oxygenation and serum cyclic guanosine monophosphate (cGMP) in children at risk of pulmonary hypertension early after corrective cardiac surgery utilising cardio-pulmonary bypass (CPB).

METHODS: 24 children (median age, weight: 0.4 years, 5.4kg) undergoing elective repair (AVSD n = 13, VSD n = 11) were randomised to receive oral sildenafil (SIL), 0.5mg/kg or placebo (PB), 4 times the day before surgery. Blood samples were collected peri-operatively to determine serum cGMP. Haemodynamic data and echocardiography were acquired under standardised conditions (FiO₂ = 0.65) at 2 and 24 hrs post-operatively: (1) Mean PA and LA pressures from indwelling monitoring lines. (2) Pulmonary vascular resistance index (PVRI) = transpulmonary gradient/pulmonary blood flow determined by spectral Doppler (3) LV and RV systolic contractility by tissue Doppler imaging (TDI) including peak velocities (SaLV, SaRV, SaS) and isovolumic acceleration (IVARV). Post-operative oxygenation was assessed using calculated oxygen delivery (arterial oxygen content × derived aortic blood flow by spectral Doppler; DO₂) and oxygenation index (OI) on admission to PICU and day 1.

RESULTS: cGMP levels (pmol/l) pre- and post-CPB were higher in the sildenafil group (SIL vs. PB; pre-CPB; 72.8 ± 135.2 vs. 41.4 ± 56.1, p = 0.44 and post-CPB; 108.1 ± 146.8 vs. 82 ± 172.8, p = 0.32). PVRI (WU.m²) remained unchanged between sildenafil and placebo groups at 2 hrs (2.67 ± 0.91 vs. 2.07 ± 1.98 respectively, p = 0.43) and 24 hrs (2.64 ± 2.28 vs. 1.9 ± 1.12 respectively, p = 0.43) post-operatively. Bi-ventricular systolic TDI parameters were significantly reduced in the sildenafil group both pre- and post-operatively (SIL vs. PB: pre-op; SaLV 3.78 ± 0.94 vs. 0.44 ± 0.19, p = 0.04 and post-op; SaRV 3.60 ± 0.93 vs. 1.35 ± 0.81, p = 0.01).
AMERICAN ASSOCIATION FOR THORACIC SURGERY

4.55 ± 1.08, SaRV 6.93 ± 1.47 vs. 8.09 ± 2.25, p = 0.002 and post-op; SaLV 2.54 ± 1.74 vs. 3.29 ± 1.16, SaRV 2.32 ± 0.77 vs. 3.20 ± 1.53, p = 0.002). Post-operative DO₂ (ml/min/m²) was significantly reduced in the sildenafil group (SIL vs. PB: 57.18 ± 21.24 vs. 74.13 ± 35.46, p = 0.04). OI was higher in the sildenafil group post-operatively (SIL vs. PB: 5.29 ± 4.6 vs. 3.38 ± 2.54, p = 0.26).

CONCLUSION: Although pre-operative Sildenafil administered at 0.5mg/kg increased serum cGMP there was minimal effect on pulmonary vascular resistance. The impact of sildenafil on peri-operative systolic function and post-operative oxygenation raises significant clinical concerns over the routine use of this drug.
34. Does Earlier Surgery Improve Left Ventricular Mass Regression Following Mitral Valve Repair for Leaflet Prolapse?
John M. Stulak, Rakesh M. Suri, Joseph A. Dearani*, Harold M. Burkhart, Thoralf M. Sundt*, Maurice Sarano, Hartzell V. Schaff*
Cardiovascular Surgery, Mayo Clinic College of Medicine, Rochester, MN, USA.
Invited Discussant: David H. Adams, MD

OBJECTIVE: Left ventricular hypertrophy in valvular heart disease is associated with poor long-term survival. Although reverse remodeling of left ventricular dimensions has been shown occur following mitral valve repair, it is unclear whether significant mass regression follows, and if so, which factors predict improved long-term normalization of left ventricular hypertrophy.

METHODS: Between March 1995 to December 2005, 2,584 patients had mitral valve repair. Of these, 463 pt (324 male) underwent repair for leaflet prolapse and had echocardiographic data available from which left ventricular mass index (LVMI) could be calculated. Concomitant preoperative tricuspid valve regurgitation (TR) was more than mild in 81 pt (17%). Patients with preoperative atrial fibrillation and other cardiac pathology necessitating concomitant intracardiac repair were not included.

RESULTS: Significant regression of LVMI occurred during the first 3 years (–28 g/m² from baseline, P < 0.001) and was maintained during follow-up longer than 3 years (–26 g/m² from baseline, P < 0.001) (Figure 1). In a multivariable model, higher preoperative left ventricular ejection fraction and greater preoperative LVMI were associated with significantly greater LVMI regression at 3 years. During follow-up later than 3 years, greater preoperative LVMI persisted in predicting improved mass regression (P < 0.001), while more than mild preoperative tricuspid valve regurgitation was independently associated with less late mass regression (P < 0.001).
CONCLUSION: Performing mitral valve repair before a fall in preoperative left ventricular ejection fraction and the development of significant TR predicts a greater likelihood of significant regression of left ventricular hypertrophy. These data provide additional support for early valve repair in patients with degenerative mitral valve disease.

11:40 a.m.  HONORED SPEAKER LECTURE
“Too Big to Fail? Healthcare Reform in the U.S. and Canada”
David Naylor, MD
University of Toronto

Introduced By: G. Alec Patterson, MD

12:30 p.m.  ADJOURN FOR LUNCH – VISIT EXHIBITS
Exhibit Hall A & B
NOTES
OBJECTIVE: Acute renal failure (ARF) after valve surgery carries a significant morbidity and mortality. Preoperative cardiac catheterization is a standard of care; however, it is unclear whether timing of radiocontrast administration for catheterization significantly affects renal function after valve surgery. We hypothesized that preoperative cardiac catheterization within 24 hours of valve surgery would be associated with the development of acute renal failure.

METHODS: A large retrospective review of all patients undergoing heart valve surgery between 2003 and 2008 was performed at our institution. ARF was defined according to the Society of Thoracic Surgery Database criteria and patients with preoperative renal dysfunction were excluded. Patients with postoperative ARF were matched to those without postoperative ARF according to their age, sex, date of surgery, NYHA class, elective versus urgent/emergent status, concomitant CABG and type of valve surgery. A logistic regression model was constructed to examine the effects of risk factors on the development of acute renal failure.
RESULTS: Out of 1,287 heart valve surgery patients, 61 patients with ARF were matched to 136 patients without ARF. The two groups were equivalent based on matching criteria, including emergent/urgent status. Cardiac catheterization within 24 hours of surgery was significantly greater in patients with ARF than in those without ARF (31.2% versus 8.8%, p = 0.013). The risk of ARF was more than 5 times higher (OR 5.2) for patients with cardiac catheterization within 24 hours of surgery as compared to patients with cardiac catheterization more than 72 hours before surgery. As demonstrated in Figure 1 the incidence of ARF decreases as time from cardiac catheterization to surgery increases. Interestingly, the number of vasopressors given within the first 6 hours after surgery was significantly associated with postoperative ARF (p = 0.0135). The risk of ARF increases 2 fold for every additional vasopressor given (OR 2.08).

CONCLUSION: Though it is often performed for patient convenience, cardiac catheterization within 24 hours of valve surgery is significantly associated with the development of acute renal failure. Current practices may need to be adjusted to ensure that more than 24 hours have passed from the time of cardiac catheterization to time of valve surgery.
36. An Analysis of Open Aortic Arch Reconstruction in the Endovascular Era

Himanshu J. Patel, Christopher Nguyen, Amy C. Diener, Mary C. Passow, G. Michael Deeb*

Surgery, University of Michigan Cardiovascular Center, Ann Arbor, MI, USA.

Invited Discussant: Alberto Pochettino, MD

OBJECTIVE: Recent advancements in endovascular repair (TEVAR), such as branched endografts or hybrid debranching/TEVAR, have extended the option of endoluminal therapy into the realm of the aortic arch. A contemporary assessment of open arch repair to provide long-term data for comparative analysis for these newer therapies is timely, warranted, and is presented here.

METHODS: Since the inception of our TEVAR program in 1993, 732 patients (mean age 59.4 yrs, 68.9% male) have undergone median sternotomy and open arch reconstruction with hypothermic circulatory arrest (HCA). Extended arch repair was performed in 42.8% with construction of bypasses to the innominate (282), left carotid (209) and subclavian (91) arteries or elephant trunk procedures (38). Concommitant AVR or aortic root replacement was needed in 388; root reconstruction in 208. Retrograde and/or antegrade cerebral perfusion were used for neuroprotection during HCA in 638 (87.2%). The operative procedure was urgent or emergent in 308 (42.1%), and included repair of Type A dissection in 263 (35.9%). 110 patients (15%) had undergone prior cardiac surgery. Primary outcomes in this study were early and late mortality. Followup was 100% complete (mean 42.1 months).

RESULTS: In-hospital or 30-day morbidity included death (39, 5.3%), stroke (35, 4.8%), or permanent dialysis (13, 1.8%). Independent predictors of early mortality included advancing age, prolonged bypass times, and operation for either type A dissection or isolated arch pathology (all p < 0.01). Actuarial survival at 10 years was 71.9%. Independent predictors of late mortality included advancing age, prolonged cerebral ischemic or lower body circulatory arrest times, and operation for isolated arch pathology (all p < 0.01). By Kaplan-Meier analysis, 10 year survival was significantly reduced following operative procedures for type A dissection (non-type A 77.3% vs. type A 66.5%, p = 0.003) or isolated arch pathology (non-isolated arch 73.5% vs. isolated arch 57.4%, p = 0.002). Freedom from aortic reoperation (any segment) was 71% at 10 years, and was reduced by the presence of dissection (no dissection 79.2% vs. dissection 61.6%, p = 0.06).

*AATS Member
CONCLUSION: Open aortic arch repair can be accomplished with excellent early and late results. These outcomes provide objective data for comparison, and suggest that newer endovascular therapies should first be evaluated in high risk groups such as those with advanced age or with isolated arch pathology prior to broader application in all.
37. Mid-Term Results of Hybrid Aortic Arch Repair Procedures

Kazuo Shimamura¹, Toru Kuratani¹, Yukitoshi Shirakawa¹, Keiwa Kin¹,
Masaaki Kato², Yoshiki Sawa¹
¹. Department of Cardiovascular Surgery, Osaka University Graduate School of Medicine, Osaka, Japan. ². Department of Cardiovascular Surgery, Morinomiya Hospital, Osaka, Japan.

Invited Discussant: Anthony L. Estrera, MD

OBJECTIVE: Various kind of hybrid aortic arch repair is reported as a candidate of a good alternative to conventional aortic arch surgery, however there still is no consensus about the indication for these procedures. In this study, we evaluated the early and mid-term results of hybrid aortic arch repair procedures.

METHODS: 353 hybrid aortic arch repairs performed from January 1994 to August 2009 was retrospectively examined. Open stent grafting was the first line procedure, however, when adequate proximal landing could be achieved with cervical branch coverage, endovascular aortic repair (with cervical bypassing) was selected. Operative procedures included 126 simple open stent grafting, 135 branched open stent grafting, and 92 endovascular aortic repair with cervical branch coverage (bypass). The average age was 67.7 ± 10.9 years, and the aortic pathology included 199 non-dissection aneurysms and 154 aortic dissections. Multivariable analysis with logistic regression analysis and Cox’s proportional hazard model was used to assess the risk factors.

RESULTS: Hospital mortality was 19/353 (5.4%). Postoperative stroke (p = 0.0054, OR 5.13) and preoperative renal failure (p = 0.031, OR 3.50) was significant risk factor for hospital death. Postoperative complications included 24 strokes (6.7%), 13 spinal cord ischemia (SCI) (3.7%). There was no significant risk factor for stroke nor SCI, however incidence of SCI was significantly reduced in endovascular aortic repair (p = 0.049). With average 36.2 ± 33.6 (1–153) months (1064 patents-year) follow up, overall survival was 86.2%, 70.8%, 62.3% at 1, 5 and 8 year respectively. Postoperative stroke (p = 0.0004, OR 3.22), preoperative renal failure (p = 0.031, OR 1.86), age (p = 0.0013, OR 1.05) and early term operation (before 2000) (p = 0.041, OR 1.75) were significant risk factors for late mortality. Freedom from aorta related death was 92.2%, 88.9%, and 85.4% at 1, 5 and 8 year respectively. Freedom from aortic event was 94.4%, 75.2% and 64.3% at 1,
5 and 8 years respectively, with male (p = 0.042, OR 2.00) being significant risk factors of aortic event.

**CONCLUSION:** Hybrid aortic arch repair procedures provided satisfactory early and mid-term results with good avoidance of aorta related death. Endovascular aortic repair with cervical branch coverage (bypassing) provided a satisfactory results even in high risk patients, which suggests the possibility of expanding the indication of this procedure.

**3:00 p.m.** INTERMISSION – VISIT EXHIBITS/COFFEE BREAK

*Exhibit Hall A & B*
OBJECTIVE: It is unknown whether avoidance of cardiopulmonary bypass (CPB) during CABG impacts intraoperative cerebral injury or long-term postoperative neuropsychological function.

METHODS: 200 unselected patients with multi-vessel coronary artery disease were randomized to off-pump (OPCAB) or on-pump (CPB) CABG between March 2000 and August 2001. 168 patients had postoperative brain MRI (1.5 T) prior to hospital discharge. Of 148 long-term survivors, 87 returned after a mean 7.5 yrs (range 6.8–8.4 yrs) for clinical follow-up; 67 had repeat MRI and 76 had neuropsychological tests covering domains including attention, memory and executive functions, administered by a neuropsychologist. MRI images were scored by a neuroradiologist. Atrophy in the temporal and frontal lobes, subcortical/periventricular white matter lesions (T2-weighted or FLAIR images indicating any cerebral injury) and diffusion-restricted lesions (early cerebral infarctions) were rated on ordinal scales and compared between groups using Wilcoxon tests.

RESULTS: There were 22 deaths from all causes among OPCAB patients and 27 among CPB patients as of March 30, 2009. 76 patients (41 CPB; 35 OPCAB) had neuropsychological testing at late follow-up. Groups were similar with respect to potential confounders such as age, gender, depression and IQ. OPCAB patients showed better attention, performing better at simultaneously tracking and mentally manipulating visual information (p = 0.011). OPCAB patients showed a trend towards better verbal learning (p = 0.064). There were no significant differences between
groups in visuospatial memory. OPCAB patients showed better cognitive reasoning and made fewer errors in reasoning (p = 0.05). There were no domains in which CPB patients outperformed OPCAB patients. Early MRI in 168 patients showed no significant differences between groups in atrophy, subcortical white matter lesions or acute infarctions. Among 59 patients who had both early and late MRI, there were no significant differences between groups with respect to atrophy over time or new subcortical white matter lesions or infarctions.

CONCLUSION: After mean 7.5 yr follow-up in this randomized trial, OPCAB was associated with superior neuropsychological function compared to CPB. Early brain MRI showed no significant differences in acute cerebral infarctions between the OPCAB and CPB groups. Repeat MRI at late follow-up showed similar atrophic changes in both groups.
39. Administration of Recombinant Activated Factor VII in the Intensive Care Unit after Complex Cardiovascular Surgery: Clinical and Economic Outcomes

John M. Toole¹, Jason S. Haney², Martha R. Stroud¹, John Lazarchick³, Fred A. Crawford¹*, Walt Uber², John S. Ikonomidis¹*

¹. Cardiothoracic Surgery, Medical University of South Carolina, Charleston, SC, USA. ². Pharmacy Services, Medical University of South Carolina, Charleston, SC, USA. ³. Pathology and Laboratory Medicine, Medical University of South Carolina, Charleston, SC, USA.

**Invited Discussant:** Arvind K. Agnihotri, MD

**OBJECTIVE:** Reoperation for bleeding following complex cardiovascular surgery is associated with increased morbidity and mortality. Recombinant activated factor VII (rFVIIa) can reverse intraoperative coagulopathy in patients undergoing cardiovascular surgery. However, its high cost and potential thrombotic risk are concerning. This study evaluated the clinical and economic impact of rFVIIa administration in the intensive care unit (ICU) to avoid reoperation for bleeding following high-risk cardiovascular procedures.

**METHODS:** From December 2003 to September 2007, 421 patients undergoing high-risk procedures (cardiac transplant, aortic surgery, redo operations, multiple cardiac procedures) were evaluated for post-operative bleeding defined as chest tube output >3 mL/kg/hr for >2 consecutive hours and their need for rFVIIa in the ICU following the primary surgery for refractory bleeding or reoperation for refractory bleeding in the first 24 hours. Patients with post-operative bleeding but who did not receive rFVIIa or undergo reoperation were included to determine post-operative bleeding incidence. Patients who received rFVIIa in the ICU were compared to patients who did not receive rFVIIa but underwent reoperation for bleeding with regard to demographics, risk assessment, blood product use, reoperation rates, OR time, ventilator time, ICU and hospital length of stay, mediastinitis, thrombotic events, renal failure, survival, and hospital cost/reimbursement.

**RESULTS:** The incidence of post-operative bleeding, indication for reoperation, and reoperation were 5.9% (25/421), 5.7% (24/421), and 3.6% (15/421), respectively. In this cohort, 12 patients, who all met criteria for reoperation, received rFVIIa in the ICU after their primary operation and 12 patients underwent reoperation for bleeding without receiving rFVIIa.

*AATS Member*
In the rFVIIa group, hemostasis was achieved in 9 (75%) patients with a significant decrease in chest tube output (5.3 vs. 1.8 mL/kg/hr, p = 0.002). Reoperation for bleeding was required in 3 patients in the rFVIIa group. Total OR time was significantly decreased in the rFVIIa group (444 vs. 599 min, p = 0.001). Hospital survival was 100% in both groups. There were no discernable differences for efficacy, safety, and cost endpoints (Table).

**CONCLUSION:** In high-risk cardiovascular surgery patients, rFVIIa can control refractory non-surgical post-operative hemorrhage and may reduce reoperation for bleeding with no appreciable increase in cost.
40. Diagnosis and Repair of Aortic Valve Cusp Prolapse: Implications for Valve Sparing Procedures
Munir Boodhwani, Laurent de Kerchove, David Glineur, Christine Watremez, Jean Rubay, Jean-Louis Vanoverschelde, Phillipe Noirhomme, Gebrine El Khoury
Cardiovascular and Thoracic Surgery, Cliniques Universitaires Saint Luc, Brussels, Belgium.

Invited Discussant: Marc R. Moon, MD

OBJECTIVE: Cusp prolapse causing aortic insufficiency (AI) is a distinct clinical entity with unique echocardiographic and surgical features. Recognition and appropriate repair of this pathology can not only treat affected patients, but can also improve results of aortic valve (AV) sparing procedures, for which pre-existing or induced cusp prolapse is a major cause of failure. We sought to examine clinical, echocardiographic and surgical characteristics of cusp prolapse in trileaflet AVs, with or without aortic disease, to enable better diagnosis and to evaluate outcomes following repair.

METHODS: Of 348 patients undergoing AV repair, 184 (53%) were treated for cusp prolapse, and 88 (48%) of those had trileaflet AV and were included in this cohort. Cusp disease was the sole mechanism for AI (Isolated Group) in 39 (44%) whereas aortic dilatation (Root–38 [43%], Supracoronary aorta–11 [13%]) was contributory in the rest (Associated Group). Mean age was 57 ± 17 years and 80 (91%) were male. In total, 104 cusps were repaired in 88 patients using central free margin plication (32%), free margin resuspension using PTFE suture (36%), or both (32%). Annular stabilization was systematically performed using sub-commissural annuloplasty or valve-sparing root replacement.

RESULTS: On preoperative echocardiography, presence of an eccentric AI jet, regardless of severity, had a 92% sensitivity and 98% specificity for the detection of single cusp prolapse. Furthermore, a transverse fibrous band (Figure 1A and 1B, arrows) was characteristically identified on the prolapsing cusp, and had a sensitivity and specificity of 59% and 93% respectively. The fibrous band correctly localized the prolapsing cusp in 100% of cases. There were no hospital mortalities and 4 late deaths (2 cardiac), all in the associated group. Freedom from AV reoperation at 8 years was 100% in the isolated group and 93 ± 5% in the associated group (p = 0.21). Freedom from recurrent AI (>2+) at 5 years was 90 ± 5% in the isolated...
and 85 ± 8% in the associated group (p = 0.68, Figure 1C). The choice of surgical technique did not affect AV reoperation or AI recurrence at follow-up (p = 0.6).

**CONCLUSION:** Recognition and surgical repair of isolated aortic cusp prolapse provides durable mid-term outcome. The findings of an eccentric AI jet and a fibrous band can aid in the diagnosis and localization of cusp prolapse associated with ascending aortic disease and may help to improve results of AV sparing procedures.

**5:00 p.m.  EXECUTIVE SESSION**  
(AATS Members Only)  
*Constitution 107, Metro Toronto Convention Centre*
2:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION – GENERAL THORACIC SURGERY
Constitution 106, Metro Toronto Convention Centre
(8 Minutes Presentation, 12 Minutes Discussion)

Moderators: Joseph B. Shrager, MD
David J. Sugarbaker, MD

41. Predictors of Recurrence, Time to Progression and Disease-Free Survival in Patients with Completely Resected Esophageal Carcinoma

Paul C. Lee1*, Jeffrey L. Port1*, Subroto Paul1, Brendon M. Stiles1, James Saunders1, Paul Christos2, Nasser K. Altorki1*
1. Cardiothoracic Surgery, Weill Cornell Medical College, New York, NY, USA.
2. Division of Biostatistics and Epidemiology, Department of Public Health, Weill Medical College of Cornell University, New York, NY, USA.

Invited Discussant: David C. Rice, MD

OBJECTIVE: The goal of this study was to determine the clinical and pathological predictors of recurrence, time to progression and long-term disease-free survival in patients with completely resected esophageal carcinoma.

METHODS: We conducted a retrospective review of a prospective database to identify patients with surgically resected esophageal carcinoma. Medical records were reviewed, and disease-free survival (DFS) was analyzed. Multivariate analysis was performed to determine independent predictors of recurrence, time to progression and DFS.

RESULTS: A total of 483 patients had esophagectomy for cancer (median age of 64, 79% male, 29% squamous). Of those, 169 patients received induction therapy. Hospital mortality was 2.5%. Median length of follow-up was 44 months. Ninety-two percent of patients had a R0 resection. The median number of lymph nodes resected was 25. Five and 10-year DFS for the entire cohort were 41% and 34%. Local recurrence rate was stage dependent and occurred in 11.2% of patients. Median time to disease progression was 10 months. Multivariate analysis demonstrated that good performance status, induction chemotherapy and en bloc surgical resection were significant independent factors predicting improved DFS (See Table). Multivariate analysis also showed that poor performance status and non-en bloc resection were significant predictors for increased...
local recurrence (p = 0.016 and p = 0.001, respectively). Local recurrence occurred in 9.7% of patients after en bloc resection and 14.3% after non-en bloc resection (p = 0.08). Time to progression was significantly prolonged in the en bloc group compared to the non-en bloc group, median of 11.3 months vs. 7.2 months (p < 0.004). Five and 10-year DFS were significantly improved in the en bloc group compared to non-en bloc group (44% and 37% vs. 29% and 8% respectively, p = 0.001).

CONCLUSION: For patients with completely resected esophageal cancer, good performance status, induction chemotherapy and en bloc resection were significant independent predictors of improved disease-free survival. Local recurrence rate was reduced and time to progression was significantly prolonged after en bloc resection.
42. The Safety of Thoracic Surgery in Patients Taking Clopidogrel (Plavix)
Robert J. Cerfolio*, Ayesha S. Bryant, Douglas Minnich
Cardiothoracic Surgery, UAB, Birmingham, AL, USA.
Invited Discussant: David P. Mason, MD

OBJECTIVE: The objective of this study is to assess the safety of thoracotomy and video-assisted surgery (VATS) in patients who are taking anti-platelet therapy, clopidogrel (Plavix).

METHODS: An observational study of a consecutive series of patients who were taking clopidogrel sulfate (Plavix) or ticagrelor (Brilinta) on the day of surgery and each day postoperatively and who underwent general thoracic surgery. Post-operative outcomes were compared to historical controls (20 controls to 1 patient) that were matched for procedure and age.

RESULTS: There were 231 patients (220 controls) between 1/2009 and 8/2009 who met the inclusion criteria, 11 patients underwent surgery while on clopidogrel. The procedures performed were: open thoracotomy with lymph node resection and lobectomy in 6 patients (including en bloc chest wall resection in one), Ivor Lewis esophagogastrectomy in two and a median sternotomy with resection of an 8 cm thymic carcinoma in one. Epidurals were not used. Two patients had video-assisted thoracoscopic wedge resections. There was no intra-operative morbidity or significant bleeding. No patients were transfused intra-operatively or post-operatively. The average hospital length of stay was 4.5 days. Only one patient experienced a 30-day major adverse cardiac event (MACE). There were no significant differences in morbidity or mortality between the cases and the matched controls.

CONCLUSION: The widely held belief that thoracotomy, esophagectomy, median sternotomy and VATS cannot be performed on patients taking anti-platelet therapy (clopidogrel sulfate, Plavix) is not true. Therefore, patients who have undergone recent coronary artery stenting do not have to come off of their anti-platelet therapy prior to surgery. This new finding may reduce the risk of post-operative cardiac events and it eases the pre-operative conversation between the cardiologist, anesthesiologist and surgeon prior to surgery.
43. Single Center Experience of 1000 Adult Lung Transplants

Daniel Kreisel¹, Sasha A. Krupnick², Varun Puri³, Tracey J. Guthrie¹,
Elbert Trulock², Bryan F. Meyers¹*, G. Alec Patterson¹*

¹. Surgery, Washington University School of Medicine in St. Louis, St. Louis,
MO, USA. ². Medicine, Washington University in St. Louis, St. Louis, MO, USA.

Invited Discussant: Shaf Keshavjee, MD, MSc

OBJECTIVE: Lung transplantation has become accepted therapy for end stage pulmonary disease. The objective of this study was to review a single institution experience of adult lung transplants.

METHODS: We reviewed 1000 adult lung transplants that were performed at a single center between 7/1988 and 1/2009.

RESULTS: Indications for transplantation were emphysema in 52% of recipients, cystic fibrosis in 18.2%, pulmonary fibrosis in 16.1% and pulmonary vascular disease in 7.2%. There was a decrease in transplants for emphysema (54% from 7/88–11/93 vs. 39.5% from 6/05–1/09) and vascular disease (14.5% vs. 1.5%) and an increase for fibrosis (9.5% vs. 31.5%). Overall recipient age was 48 ± 13 years with an increase from 43 ± 12 (7/88–11/93) to 50 ± 14 years (6/05–1/09). We performed bilateral transplants in 80.5% and single transplants in 18.6% of recipients. Lengths of ICU and hospital stays were 3 and 15 days, respectively. Overall incidence of primary graft dysfunction (PGD) was 22.1%. There was no decline in the incidence of PGD during the study period (23% for patients transplanted between 7/88–11/93 and 23.5% for transplants between 6/05–1/09). Overall hospital mortality was 6.1%. Hospital mortality was significantly higher for patients, who suffered from PGD compared to those, who did not experience PGD (13.6% vs. 4%, p < 0.001). 20 patients (2%) required retransplantation for PGD (11) or bronchiolitis obliterans (BO) (9). Freedom from BO syndrome was 84% at 1 year, 38.2% at 5 years and 12.2% at 10 years after transplantation. Freedom from BO syndrome at 5 years has improved from 24.3% for transplants performed between 7/88 and 11/93 to 43.5% for patients transplanted between 10/01 and 6/05. 1-, 5-, 10- and 15-year survival rates were 84%, 56.4%, 32.2% and 17.8%, respectively. 5-year survival rates improved from 49.6% for patients transplanted between 7/88 and 11/93 to 62.1% for transplants between 10/01 and 6/05. Notably, PGD was associated with lower survival rates at 1, 5 and 10 years (PGD: 72.8%, 43.9%, 18.7% vs. No PGD: 87.1%, 59.8%, 35.7%, p < 0.001) and lower rates of freedom from BO syndrome (PGD: 78%, 27.5%, 8.5% vs. No PGD: 85.4%, 40.7%, 13.1%, p = 0.007).

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CONCLUSION: While 5-year survival rates have improved over the study period, long term outcomes continue to be limited by the development of BO. PGD is associated with higher rates of BO syndrome and impaired short and long term survival. A better understanding of PGD and BO are critical to improve outcomes after lung transplantation.

3:00 p.m. INTERMISSION – VISIT EXHIBITS/COFFEE BREAK
Exhibit Hall A & B
44. Retrospective Analysis of Two Endoscopic Thoracic Sympathectomy Techniques for Palmar Hyperhidrosis: Clamping versus Cutting of the Sympathetic Chain

Ali N. Ibrahimiyev, Ted Yanagihara, Alan Weinberg, Joy Hirsch, Catherine R. Harris, Lyall Gorenstein

Invited Discussant: M. Blair Marshall, MD

OBJECTIVE: Endoscopic Thoracic Sympathectomy (ETS) at the T3 level is one of the surgical treatment options that can eliminate the disabling symptoms of palmar hyperhidrosis. To date, several studies have shown good results when clamping at various levels and some have contrasted the effects of cutting and clamping at multiple thoracic levels, but to our knowledge no single institution studies have directly compared the effects of cutting versus clamping when the sympathectomy is restricted to T3. We quantified and compared pre- and post-surgical subjective sweat production in different areas of the body and changes in quality of life, accounting for both benefits and side effects with clamping versus cutting of the sympathetic chain at T3 level.

METHODS: Patients seen between June 30, 2003 and March 16, 2007 were asked to quantify the severity of their symptoms before and after endoscopic thoracic sympathectomy. Interviews were conducted approximately one year following the procedure and only patients receiving sympathectomy at level T3 for a chief complaint of palmar hyperhidrosis were included in the analysis (n = 153). In 45% of these, clamping of the sympathetic chain was performed while the remaining 55% had the chain ablated.
**RESULTS:** Following surgery, 77% of patients experienced no hand sweating, 20% reported mild or normal sweating. No patients had continued excessive sweating of the hands. Ninety-six percent of patients were satisfied with the results. Coincidental decrease in sweating also occurred in other area of the body: feet (63%), axilla (60%). Compensatory sweating was experienced on at least one part of the body by 87% of patients and was severe in 4% of patients. There was no difference in outcome between patients having clamping versus cutting of the sympathetic chain (Table 1).

**Table 1: Comparison of cutting versus Clamping of Sympathetic Chain**

<table>
<thead>
<tr>
<th></th>
<th>Cutting</th>
<th>Clamping</th>
<th>P-value</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hands</td>
<td>-2.58 (0.64)</td>
<td>-2.71 (0.52)</td>
<td>0.148</td>
<td>Greatly Improved</td>
</tr>
<tr>
<td>Face</td>
<td>0.06 (0.78)</td>
<td>-0.19 (0.88)</td>
<td>0.089</td>
<td>No Change</td>
</tr>
<tr>
<td>Blushing</td>
<td>-0.15 (0.88)</td>
<td>-0.34 (0.79)</td>
<td>0.220</td>
<td>No Change</td>
</tr>
<tr>
<td>Armpits</td>
<td>-0.97 (1.17)</td>
<td>-0.88 (1.18)</td>
<td>0.777</td>
<td>Mildly Improved</td>
</tr>
<tr>
<td>Feet</td>
<td>-0.73 (0.93)</td>
<td>-0.86 (0.98)</td>
<td>0.319</td>
<td>Mildly Improved</td>
</tr>
<tr>
<td>Trunk</td>
<td>1.09 (0.86)</td>
<td>1.03 (0.97)</td>
<td>0.728</td>
<td>Moderately Worse</td>
</tr>
<tr>
<td>Thighs</td>
<td>0.91 (0.8)</td>
<td>0.69 (0.97)</td>
<td>0.229</td>
<td>Mildly Worse</td>
</tr>
<tr>
<td>QOL</td>
<td>2.18 (0.83)</td>
<td>2.03 (0.92)</td>
<td>0.150</td>
<td>Greatly Improved</td>
</tr>
<tr>
<td>Satisfied</td>
<td>% Yes 95</td>
<td>% Yes 97</td>
<td>0.741</td>
<td></td>
</tr>
<tr>
<td></td>
<td># Yes 74</td>
<td># Yes 61</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The data indicate no significant difference between cutting and clamping of the sympathetic chain across all parameters, including the resolution of hand sweating (p = 0.148), the appearance of CS on the trunk (p = 0.728) or thighs (p = 0.229) and rates of patient satisfaction (p = 0.741).

**CONCLUSION:** We found high rates of success and patient satisfaction when T3 sympathectomy was performed for palmar hyperhidrosis. In all of the same outcome measures we found no differences between cutting and clamping techniques.
45. Expanding the Indications for Laparoscopic Wedge Gastroplasty with Fundoplication for the Shortened Esophagus

Daniel C. Wiener1, Jon O. Wee1, Abraham Lebenthal2, David J. Sugarbaker1, Raphael Bueno1*
1. Thoracic Surgery, Brigham and Women’s Hospital, Boston, MA, USA.
2. Surgical Oncology, Fox Chase Cancer Center, Philadelphia, PA, USA.

Invited Discussant: Michael A. Maddaus, MD

OBJECTIVE: Laparoscopic wedge gastroplasty (LWG) has been described for esophageal lengthening in patients undergoing surgery for giant paraesophageal hernias (GPH). We sought to examine expanded indications for LWG in patients with shortened esophagus presenting with other disorders arising from gastroesophageal reflux.

METHODS: We performed a retrospective review of a prospective database with supplemental chart review at an academic tertiary care hospital. Consecutive patients undergoing laparoscopic fundoplication with concurrent esophageal lengthening procedure were identified. All lengthening procedures were performed as a wedge gastroplasty calibrated over a Maloney dilator (48–60F) utilizing multiple firings of an articulating endostapler (Covidien® 45 mm, 3.8 mm). Clinical and post-operative data were reviewed for all patients.

RESULTS: 571 consecutive patients underwent laparoscopic fundoplication from November, 2002 to December, 2008. 87 (15%) underwent LWG as part of their procedure. Mean age was 61 year and 56 (64%) were women. Median follow up time was 33 months. Primary indications for surgery were either GPH (43, 49%) or symptomatic reflux (44, 51%). Six of 87 (6.8%) were reoperations for failed anti-reflux procedures. The mean endoscopically measured distance between the incisors and the GE junction in patients requiring LWG was 35.6 cm. All patients had a 360 degree Nissen wrap, 52 patients (59.7%) had biologic mesh reinforcement of crural closure. The majority of patients with GPH also had a gastrostomy tube placement (26, 60.5%). Early post-operative complications occurred in 3 patients (3.4%) including: splenectomy for bleeding (n = 1) exploratory laparotomy for missed colon injury (n = 1), trochar site hernia (n = 1). There were no peri-operative deaths or leaks. Fifteen patients (17.2%) had late complications of dysphagia after discharge requiring dilation. Mean

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time to dilation was 4.5 months (range 1–23 months). Two patients (2.2%) ultimately required conversion to a partial fundoplication due to recurrent dysphagia despite multiple dilatations.

**CONCLUSION:** (1) LWG and 360 degree fundoplication can be performed safely for shortened esophagus associated with GPH as well as reflux. (2) Dysphagia is a significant late complication that often resolves with dilatation. (3) LWG should be considered in the surgical management of any reflux disease associated with esophageal shortening.
46. A Prospective European Multicenter Randomized Trial to Evaluate Pleuraseal for Control of Air Leaks after Elective Pulmonary Resection

Paul De Leyn1*, Michael R. Mueller2, Jan W. Oosterhuis3, Thomas Schmid4, Cliff K. Choong5, Walter Weder6*, Youri Sokolov7, Peter De Rooij8
1. Department of Thoracic Surgery, University Hospitals Leuven, Leuven, Belgium. 2. Otto Wagner Hospital, Vienna, Austria. 3. VU Medisch Centrum, Amsterdam, Netherlands. 4. Universitaetsklinik Landeskrankenhaus Innsbruck, Innsbruck, Austria. 5. Papworth Hospital, Cambridge, United Kingdom. 6. Division of Thoracic Surgery, University Hospital, Zurich, Switzerland. 7. Service de Chirurgie Thoracique, Hopital Erasme, Bruxelles, Belgium. 8. Medical Centre Rotterdam Zuid, Rotterdam, Netherlands.

Invited Discussant: Alessandro Brunelli, MD

OBJECTIVE: Postoperative air leaks are a major cause of morbidity after lung resections. This study was designed to evaluate the efficacy and safety of a synthetic bio-resorbable surgical sealant (PleuraSeal) in treating air leaks after pulmonary resection.

METHODS: In a European multicenter trial, patients with intraoperative air leaks after lung resection (lobectomy or segmentectomy by open thoraecotomy) were randomized in a 1:1 ratio to receive PleuraSeal applied to sites of air leak after standard method of lung closure [PleuraSeal (P) group] or to have standard lung closure only [Control (C) group]. Air leaks were graded intra-operatively on a scale from 0–3.

The primary outcome variable was the percentage of patients remaining air leak free until discharge. The secondary outcome variables were the proportion of patients for whom intra-operative air leak sealing success was achieved, the time to last observable air leak, the duration of chest tube drainage and the duration of hospitalization. Morbidity and safety outcomes were also evaluated.

RESULTS: In 8 European investigative sites 161 patients consented for potential study participation and 121 of them were randomized. Sixty-two patients were intraoperatively randomized in the P group and 59 patients in the C group. Most of the patients (98.3%) underwent an open lobectomy for bronchogenic carcinoma. Overall intra-operative air leak sealing success was 71.0% for P group and 23.7% for C group, (p < 0.001). In patients with grade 2 and 3 air leaks (n = 77) sealing was successful in 71.7% (P group) versus 9.1% (C group), (p < 0.001). More patients with

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grade 2 and grade 3 air leak remained air leak free in the P group (43.5% vs. 15.2%, p = 0.013). In these patients the median time from skin closure to last observable air leak was 6 hours (P group) versus 42 hours (C group), p = 0.718. There was no statistically significant difference in the duration of chest tube drainage and hospitalization in the whole cohort. There were no deaths. Incidence and nature of the serious adverse events were comparable between groups and none of the complications were reported as treatment related.

CONCLUSION: Surgical sealants are safe and reduces significantly post-operative air leaks. The application of this surgical sealant is safe and effective in treating intra-operative air leaks after lung resection. In this prospective study, significantly less patients with surgically relevant air leaks (grade 2 or 3) had postoperative air leaks when PleuraSeal was applied.

5:00 p.m. EXECUTIVE SESSION
(AATS Members Only)
Constitution 107, Metro Toronto Convention Centre
47. **In-Situ Pericardial Extracardiac Lateral Tunnel Fontan Operation: Fifteen-Year Experience**

Nahidh W. Hasaniya, Anees J. Razzouk*, Leonard L. Bailey*

*Cardiothoracic Surgery, Loma Linda University Medical Center, Loma Linda, CA, USA.

**Invited Discussant:** Erle H. Austin, III, MD

**OBJECTIVE:** To evaluate the long term outcome of in-situ pericardial extra-cardiac lateral tunnel (ECLT) Fontan operation in a single institution.

**METHODS:** From June 1994 to August 2009, 160 patients (n = 96, 60% males, median age = 39 months, mean weight 15.5 K gm) underwent completion Fontan using pedicled pericardial ECLT. Patients charts were reviewed for peri-operative and long term follow up data and outcome. The potential growth of these tunnels were evaluated with echocardiography.

**RESULTS:** The main primary diagnoses were: Tricuspid atresia (n = 44, 27%); Double outlet right ventricle (n = 29, 18%); hypoplastic left heart syndrome (n = 26, 16%) and Double inlet left ventricle (n = 20, 12.5%). All operations used cardiopulmonary bypass (median 126 min). Concomitant procedures included: atrial septectomy (n = 24, 15%), pulmonary arterioplasty (n = 6, 3.7%), and repair of atrioventricular valve (n = 2, 1.2%). Fenestration was performed in (3, 1.8%) patients. All patients were placed on aspirin post-operatively and no anticoagulants were used. The mean follow up was 6.5 ± 3.7 (range 0.1 to 15) years.

There were 2 operative (1.2%) and 6 (3.7%) late deaths. Actuarial survival at 14 years was 90%. Average hospital stay was 4.2 days.

Early complications included: prolonged effusions (n = 49, 31%); chylothorax (n = 7, 4.4%); re-admissions (n = 35, 22%); cerebrovascular accidents (n = 8, 5%); left phrenic nerve palsy (n = 1, 0.8%); transient arrhythmias (n = 5, 3.1%). No patient required pace-maker secondary to this operation.

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Late complications included: Tunnel stenosis (n = 3, 1.8%) managed successfully with balloon dilatation and senting in 2 and surgical revision in 1 patient; tunnel thrombosis (n = 2, 1.2%) both patients died; protein losing enteropathy (n = 4, 2.5%).

Follow up echocardiography of 10 patients showed laminar flow with no turbulence or gradient at the inferior vena cava and mid-tunnel levels. The diameter indexed to body surface area showed growth, reduction or no change depending on the flow demands.

**CONCLUSION:** We believe that ECLT Fontan operation using in-situ pericardial flap should be the procedure of choice in most single ventricle patients. This operation is technically feasible with minimal intermediate and late complications and mortality. It is the most cost effective live conduit that has a low incidence of arrhythmias and thrombosis, and has potential to adjust dimensions to flow requirements.
48. Primary Sutureless Procedure for “Simple” Total Anomalous Pulmonary Venous Connection: Mid-Term Results in a Single Institution

Bobby Yanagawa, Abdullah A. Alghamdi, Christopher A. Caldarone*, John G. Coles*, Osman O. Al-Radi, Glen S. Van Arsdell*

Surgery, Division of Cardiac Surgery, University of Toronto, Toronto, ON, Canada.

Invited Discussant: Francois Lacour-Gayet, MD

OBJECTIVE: The outcomes for the repair of total anomalous pulmonary venous connection (TAPVC) continue to be complicated by pulmonary vein stenosis (PVS), particularly with young age at initial surgery, infracardiac connection type and existing PVS. We have previously reported the use of a sutureless repair for surgical management of iatrogenic PVS. Because of potential benefits of achieving maximal performance from small pulmonary veins by not having associated suture lines and the potential for precluding post repair development of pulmonary vein stenosis, we have been evolving to utilizing a primary sutureless repair technique for TAPVC–particularly for those at increased risk for PVS.

METHODS: Fifty-seven patients (median age 15 days [1 to 1157 days] and median weight 3.4 kg [1.7 to 11.7 kg]) underwent a sutureless or classical TAPVC repair from 1/97 to 7/09. Median follow-up time was 2.9 years (5 days to 11.7 years). Comparisons were made for preoperative characteristic and outcomes between sutureless and classical repair.

RESULTS: Types of TAPVC included 31 (54%) supracardiac, 15 (26%) cardiac and 11 (19%) infracardiac. Preoperative PVS was seen in 8 (14.0%) and vertical vein obstruction in 35 (61.4%) patients. A primary sutureless repair with in situ pericardium was carried out successfully in 21 (36.8%; supracardiac n = 12, cardiac n = 4, infracardiac n = 5) patients. There was a trend for greater preoperative PVS (23.8% vs. 5.3%) and significantly higher vertical vein obstruction (81.0 vs. 50.0%; P < 0.02) in the sutureless repair as compared to the classical repair. Primary outcomes of reoperation for PVS [n = 1 (classical repair) 1.9%] or death [n = 2; 3.8%] were not different. Furthermore, post-operative PVS scores were similar between two repair groups.

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CONCLUSION: Primary sutureless repair has equivalent outcomes to classical repair of TAPVC in the presence of higher levels of preoperative stenosis. The lack of late development of PVS in 21 infants including 5 infracardiac type suggests that continued evaluation of primary sutureless repair is warranted.
49. Repair of Major Cardiac Defects in Low Birth Weight Infants: Is Delayed Surgical Intervention Warranted?

Charles Sheppard², James H. Moller², Roosevelt Bryant¹, Ronald M. Rosengart², James D. St. Louis¹
1. Department of Surgery, University of Minnesota, Minneapolis, MN, USA.
2. Department of Pediatrics, University of Minnesota, Minneapolis, MN, USA.

Invited Discussant: Peter J. Gruber, MD, PhD

OBJECTIVE: Few studies have accurately described survival in neonates weighing less than 1.5 kg at the time of surgical correction for a major congenital cardiac defect. The goal of this study was to confirm the hypothesis that early intervention in this population has comparable survival to individual in which repair is delayed.

METHODS: A mutli-institutional retrospective review of patients that underwent correction of a major cardiac surgical anomaly with cohorts divided into individuals either weighing less than 1.5 kg or between 1.5 to 2.5 kg. Operative ligation of a PDA or other minor surgical procedures were excluded from this analysis. Survival was defined as either discharge from the hospital or alive at the next major cardiac operation.

RESULTS: Four hundred forty one patients who underwent operative repair of a major cardiac anomaly were included in this review. Cohort 1 consisted of 172 patients weighing less than 1.5 kg, while cohort 2 included 269 patients weighing between 1.5 and 2.5 kg. The number of patients categorized to RACHS-1 scores for cohorts 1 and 2 were: 3 and 7 (1); 79 and 114 (2); 47 and 84 (3); 11 and 35 (4); 11 and 9 (5) respectively. In patients weighing less than 1.2 kg, overall survival was 65%. In those between 1.2–1.5 kg, survival was 79%. In infants weighing less than 1.5 kg at birth, but were between 1.5 and 2.5 kg at operation, survival was 77%. Operative procedures not requiring cardiopulmonary bypass, survival was 79% for less than 1.5 kg and 87% between 1.5–2.5 kg. For patient requiring cardiopulmonary bypass, survival was 62% in the group less than 1.5 kg and 59% in the 1.5 to 2.5 kg group. Weight was not an independent risk factor for mortality. Operations were performed on a greater number of low weight infants each year over the period of the study. No notable improvement in outcome over time was found for those individuals weighing more than 1.2 kg. There was a trend toward improved outcome over time for those weighing less than 1.2 kg, but not statistically significant.
CONCLUSION: Survival in infants weighing less than 1.5 kg is comparable to infants that undergo a major cardiac procedure at a greater weight. These data suggest that operative delay secondary to size is not warranted.

3:00 p.m.  INTERMISSION – VISIT EXHIBITS/COFFEE BREAK

Exhibit Hall A & B
50. Precise Evaluation of Bilateral Pulmonary Artery Banding for Initial Palliation for High-Risk Hypoplastic Left Heart Syndrome

Kazuo Kitahori1, Arata Murakami1, Tetsuhiro Takaoka1, Shinichi Takamoto2, Minoru Ono1
1. Cardiothoracic Surgery, The University of Tokyo, Hospital, Tokyo, Japan.
2. Cardiothoracic Surgery, Mitsui Memorial Hospital, Tokyo, Japan.

Invited Discussant: Christopher A. Caldarone, MD

OBJECTIVE: In patients with high-risk hypoplastic left heart syndrome (HLHS), the Norwood operation (NW) in the neonatal period still has a high mortality compared with other cardiac surgery, so bilateral pulmonary artery banding (bPAB), a very effective initial operation for HLHS, was performed, but precise evaluation of bPAB has not been done sufficiently. We present our findings here.

METHODS: We have performed bPAB since 2006. Seventeen patients with HLHS or a variant underwent bPAB before NW. Echocardiography was performed between bPAB and NW, and the flow acceleration just after bPAB and before NW was evaluated. Before NW, a catheter examination was also performed.

RESULTS: The bPAB was performed at 6.6 ± 0.6 days of age, and NW, at 130 ± 88 days. Body weight (BW) was 2.5 ± 0.4 kg at bPAB and 4.0 ± 1.1 kg at NW. The length of the tape for bPAB was 9.9 ± 0.6 mm in the right PA (RPA) and 9.4 ± 0.6 mm in the left PA (LPA) because RPA was usually wider than LPA. The width of the tape was 2 mm in all cases. Catheter examination was performed at 95 ± 85 days after bPAB. SaO2 was 71 ± 8.6%.

Multi-regression analysis revealed that SaO2 was estimated well by 4 factors; banding size of RPA, BW at bPAB, BW at NW and period between bPAB and catheter examination (R2 = 0.79). The echocardiography just after bPAB showed the blood flow at bPAB was accelerated to 3.0 ± 0.8 m/s in RPA and 3.3 ± 0.8 m/s in LPA. The estimated pressure gradient was 39.2 ± 17.6 mmHg in RPA and 46.1 ± 23.0 mmHg in LPA. The blood flow at bPAB was accelerated to 3.7 ± 0.7 m/s in RPA and 4.0 ± 0.6 m/s in LPA.
before NW. The estimated pressure gradient was 62.6 ± 27.6 mmHg in RPA and 56.1 ± 19.6 mmHg in LPA before NW. Catheter examination revealed mean wedge pressures of 18.0 ± 7.2 mmHg for RPA and 16.2 ± 4.3 mmHg for LPA. Operative mortality was zero. One patient needed reoperation to adjust PAB. Prolonged pleural effusion was observed in 1 case.

**CONCLUSION:** Post operative SaO2 after bPAB correlated closely with banding size, BW at PAB and NW and period after bPAB. Since the mean PA pressure before NW was low enough for single ventricular circulation, the bPAB in this study was an effective option for high-risk patents with HLHS or a variant. We consider that our size of bPAB was suitable; BW + 7 mm in LPA and BW + 7.5 mm in RPA.
51. Determinants of Outcome after Surgical Treatment of Pulmonary Atresia with Ventricular Septal Defect and Major Aortopulmonary Collateral Arteries

Adriano Carotti1, Sonia B. Albanese1, Sergio Filippelli1, Lucilla Rava2, Paolo Guccione1, Giacomo Pongiglione1, Roberto M. Di Donato1*

1. Department of Pediatric Cardiology and Cardiac Surgery, Bambino Gesu’ Children’s Hospital, Rome, Italy. 2. Epidemiology Unit, Bambino Gesu’ Children’s Hospital, Rome, Italy.

Invited Discussant: Frank L. Hanley, MD

OBJECTIVE: Analysis of results and identification of variables influencing surgical outcome in a 15-year series of patients treated for pulmonary atresia, ventricular septal defect, and multiple aorto-pulmonary collateral arteries.

METHODS: Ninety consecutive patients aged 37 ± 61 months (range: 20 days–35 years) primarily underwent either one-stage complete unifocalization (n = 69) or palliation to promote native pulmonary arterial development (n = 21), the latter limited to patients with hypoplastic dominant pulmonary arteries. Chromosome 22q11.2 microdeletion occurred in 37% of the cases. Ventricular septal defect closure was accomplished in 70 patients (78%), with a mean postoperative pRV/pLV ratio of 0.48 ± 0.14 (range: 0.2–0.8). End-points of univariate and multivariate analysis were suitability to one-stage unifocalization, suitability to simultaneous VSD closure, postoperative pRV/pLV ratio, and survival.

RESULTS: Fourteen-year survival, freedom from conduit reintervention, and freedom from percutaneous intervention on pulmonary arteries were 75%, 46%, and 52% respectively. At a median follow-up of 46 months pRV/pLV ratio did not significantly change compared to early postoperative value (paired t test on 35 observations: early postoperative, 0.51 ± 0.14 vs. follow-up, 0.53 ± 0.18: p = ns). Patients with confluent intrapericardial pulmonary arteries had a worse postoperative pRV/pLV ratio after ventricular septal defect closure than those without (p = 0.04). Kaplan-Meier survival estimates showed age below 30 days (p = 0.0004), weight below 3 kgs (p = 0.0004), and 22q11.2 microdeletion (p = 0.001) significantly affecting survival. Microdeletion was significantly associated with mortality even at Cox regression model (p = 0.006). Finally, simultaneous ventricular septal defect closure at the time of unifocalization significantly correlated with both early (p = 0.0013) and overall survival (p = 0.013).

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CONCLUSION: Results of surgery are satisfactory and durable, in spite of the need of repeated percutaneous or surgical interventions on pulmonary arteries or conduit. Avoiding neonatal age and body weight below 3 kg may increase the chance of positive outcome in terms of survival, as it does the simultaneous closure of the ventricular septal defect, when feasible, whereas presence of intrapericardial pulmonary arteries does not improve the hemodynamic result after ventricular septal defect closure. Finally, chromosome 22q11.2 microdeletion remains a variable significantly affecting survival.
52. The Cone Reconstruction of the Tricuspid Valve in Ebstein Anomaly with or without 1.5-Ventricle Repair
Jinfen Liu*, Lisheng Qiu, Zhongqun Zhu, Huiwen Chen, Haifa Hong
Shanghai Children’s Medical Center, Shanghai, China.

Invited Discussant: Pedro J. del Nido, MD

OBJECTIVE: The cone reconstruction of the tricuspid valve is more and more utilized in the surgical treatment for Ebstein anomaly. The bidirectional cavopulmonary shunt (BCPS), or “one and one-half (1.5) ventricle repair” has been used for the operation of severe anatomic and functional Ebstein anomaly. We sought to review our institutional experience and midterm results of the tricuspid valve cone reconstruction in Ebstein anomaly with or without 1.5-ventricle repair.

METHODS: From Jan 2004 to Oct 2009, 30 consecutive patients with Ebstein anomaly underwent the tricuspid valve cone reconstruction. All patients had the cone reconstruction of the tricuspid valve and twenty patients with severe Ebstein anomaly had a BCPS constructed. The median age at operation was 60 months (2 months–192 months). Our criteria to use BCPS as an adjunctive surgical way for severe Ebstein anomaly includes: a severely enlarged right-sided chambers, significant abnormality of the septal leaflet of tricuspid valve from morphology and hemodynamic instability after separation from cardiopulmonary bypass. Clinical or echocardiographic characteristics were studied both preoperatively and postoperatively.

RESULTS: There was 1 (3.3%) hospital death. Before operation, tricuspid incompetence was moderate in 8 and severe in 22. Postoperative early echocardiography showed that tricuspid incompetence was mild in 26, moderate in 3 and severe in 1. After median follow-up time of 22 months, tricuspid incompetence of twenty patients with BCPS was mild in 16 and moderate in 4. Tricuspid incompetence of ten patients without BCPS from the latest echocardiogram (median follow-up time is 32 months) was mild in 4, moderate in 4 and severe in 2. For patients whose postoperative tricuspid incompetence was beyond mild among ten patients without BCPS, preoperative echocardiogram showed severely dilated right ventricle. 20 patients with BCPS reported no functional limitations at the time of their last follow-up. 4 patients with BCPS reported facial swelling, but this was not problematic.

*AATS Member
CONCLUSION: Satisfactory early results can be achieved with the cone reconstruction of the tricuspid valve in the surgical management for Ebstein anomaly. However, for patients with severe Ebstein anamoly, the cone reconstruction of tricuspid valve alone may not be enough to produce ideal midterm results. 1.5-ventricle repair should be added as a planned procedure to the cone reconstruction of the tricuspid valve for patients with severe Ebstein anomaly.

5:00 p.m. EXECUTIVE SESSION
(AATS Members Only)
Constitution 107, Metro Toronto Convention Centre
OBJECTIVE: Transcatheter Valve Replacement is a rapidly emerging alternative for patients at high risk for open surgical repair. The Direct Flow Medical (Santa Rosa, CA) Percutaneous Aortic Valve is a non-metallic tri-leaflet bovine tissue valve that is repositionable and retrievable. The purpose of this study is to assess its safety and function in patients one year post implant and beyond.

METHODS: Thirty-one patients with severe aortic stenosis demonstrated by baseline mean gradient of 49.1 ± 13.8 mmHg and EOA of 0.54 ± 0.16 cm² were enrolled in the study at 2 centers in Germany. Patients were considered non-surgical candidates with a mean Logistic EuroSCORE of 26.9%, mean age 82.2 years, and increased NYHA Functional Class (71% NYHA Class III). The study valve was successfully implanted with the 22 Fr Direct Flow Medical Delivery System in 22 patients via a transfemoral approach. Of those patients who did not receive a study valve, the catheter could not be inserted (n = 2), a successful balloon valvuloplasty was not possible (n = 2), or the valve was successfully retrieved (n = 5).

* AATS Member
RESULTS: At one year post implant, mean gradients remain improved at 20.7 ± 9 mmHg. EOA has decreased slightly from 30 days to 1.22 ± 0.29 cm², however NYHA functional class has remained greatly improved with all patients reporting NYHA Class I or II. Aortic regurgitation and paravalvular leak also continues to be negligible with all patients exhibiting no or low grade insufficiency. One year risk adjusted survival for the intent to treat population was 72% and early patients are already reaching the 2 year post implant time point. One late death occurred between the 6 month and 1 year time points in an 89 year old patient who suffered from ventricular arrhythmia following a coronary stenting procedure. This death was adjudicated by the CEC as unrelated to the device. All other patients previously reported surviving at 6 months completed a 1 year follow-up visit.

CONCLUSION: Despite multiple comorbidities and a high surgical risk profile, patients implanted with the Direct Flow Valve continue to have positive outcomes at one year post implant. Extended follow-up appears promising as well with 2 year data collection ongoing. Further, the development of an enhanced 18 Fr delivery system will increase patient inclusion and ease of use making this novel technology a formidable option for future patients.
T2. **Sutureless Aortic Valve Replacement with the Trilogy Trilobal Aortic Valve System-Multicenter Experience**

Ingo Breitenbach¹, Jerzy Sadowski³, Gerhard Wimmer-Greinecker⁵, Christoph Schmitz², Leo A. Bockeria⁴*, Krzysztof Bartus³, Ravil M. Muratov⁴, Wolfgang Harringer¹

¹. Department of Thoracic and Cardiovascular Surgery, Klinikum Braunschweig, Braunschweig, Germany. ². Department of Cardiac Surgery, University of Munich, Munich, Germany. ³. Department of Cardiovascular Surgery and Transplantology, Jagiellonian University, Krakow, Poland. ⁴. Bakoulev Scientific Center for Cardiovascular Surgery, Moscow, Russia. ⁵. Department of Thoracic and Cardiovascular Surgery, Herz und Gefässzentrum Bad Bevensen, Bad Bevensen, Germany.

**OBJECTIVE:** There is a need for fast sutureless and simplified implantation of valve prostheses due to minimal invasive aortic valve surgery. The novel modular sutureless Trilogy Aortic Valve System has the potential to fulfill these requirements. In this study we report the multicenter experience with this system in 32 patients.

**METHODS:** Between November 2006 and November 2008, 32 patients (18 females; mean age 71.7 ± 6.5 years) with severe aortic valve stenosis underwent Aortic Valve Replacement with the Trilogy System. In 6 cases concomitant CABG was performed. Hemodynamic parameters (Effective Orifice Area (EOA), mean and peak transvalvular gradients) were measured by transthoracic echocardiography at discharge, 4–6 months, 11–14 months and annually thereafter and confirmed by an independent core lab.

**RESULTS:** Pump time was 111 ± 41 min and cross clamping time was 70 ± 23 min. Valve deployment time was 21 ± 6 min. The transvalvular gradients at discharge were 10.2 ± 3.5 mmHg (mean) and 20.0 ± 6.8 mmHg (peak) and the EOA was 1.9 ± 0.4 sqcm, at 2 year follow up gradients were 7.2 ± 2.7 mmHg (mean) and 13.5 ± 4.7 mmHg (peak) and the Effective Orifice Area was 1.9 ± 0.3 sqcm. There was no early postoperative mortality however two patients died on follow up of non cardiac reasons. There was one redo AVR at 22 months postoperatively due to prosthetic valve endocarditis.

*AATS Member*
CONCLUSION: Sutureless AVR is feasible and safe with the Trilogy Aortic Valve System. Following an initial learning curve, the unique Trilogy Attachment Clips (TACs) and modular valve design facilitate a more rapid, secure and simpler implantation procedure when compared to conventional tissue valves. The simplicity of this technique may also enable a greater adoption of minimally invasive AVR by a broader spectrum of surgeons. The trilobal valve shape, which matches the contours of the Sinuses of Valsalva, allows for an extreme supra-annular implantation and results in a larger EOA for a given patient annulus. Further, the independently suspended leaflets and proprietary anticalcification method create a low stress condition which is expected to positively impact long term tissue durability.
OBJECTIVE: Closed chest intra-cardiac mitral valve repair with the MitraClip system is undergoing continued evaluation in the EVEREST trials, and until recently was performed exclusively by the interventional cardiologist (IC). At a single institution, the intra-procedural outcomes from a joint cardiology and cardiac surgery approach are reported.

METHODS: 24 patients (age 73.6 ± 11.3 years, range 40–88 years) with 3 or 4+ mitral regurgitation (MR) were enrolled under the EVEREST II Continued Access REALISM protocol-12 with low to moderate risk, (STS 4.7 ± 2.5%) and 12 with high risk (STS 18.8 ± 13.2%). Procedures were performed either by the principal investigator (PI is an IC with previous MitraClip experience), or by a non-PI IC or a cardiac surgeon (CS) under supervision by the PI. The IC and CS had previously undergone didactic and in vitro simulator training for the MitraClip procedure. Assessments of MR severity at baseline and post procedure were made in the catheterization laboratory by the on-site echocardiologist. Comparisons of procedure times were made by 1-way ANOVA with Kruskal-Wallis statistic.

RESULTS: Overall MR severity was 3.6 ± 0.5 pre, and 1.0 ± 0.7 post. Ten of 24 MitraClip procedures were performed by the PI with an overall procedure time of 135 ± 64 minutes, and transseptal time of 11.2 ± 11.8 minutes requiring 1.4 ± 0.7 transseptal punctures. Procedure or transseptal times by IC (5/24) or CS (9/24) were not significantly different when compared to the PI, and were shorter over time (Figure). Fluoroscopy time was shorter for the PI (22.2 ± 7.6 minutes, P = 0.01), but not different between the IC and the CS (36.2 ± 9.6 and 37.5 ± 17.9 minutes, p = NS). No patient had an intra-procedural complication.

CONCLUSION: With increasing experience, MitraClip procedural times decrease regardless of specialty. Mitral valve repair with the MitraClip can be performed by interventional cardiologists or cardiac surgeons with good outcomes and low complication rates.

*AATS Member
A Fluid Diode for Control of Pulmonary Insufficiency
Tain-Yen Hsia¹, Tiffany Camp², Tim McQuinn³, Richard S. Figliola²

OBJECTIVE: Current options for pulmonary valve replacement remain imperfect. Bioprostheses are limited by eventual failure, while mechanical valves are designed for left heart circulation and require systemic anticoagulation due to moving parts. A fluid diode is a motionless device that offers low resistance to antegrade flow, but much higher resistance to regurgitation. We present the hemodynamic efficacy of a novel, motionless fluid diode device specifically designed to control pulmonary regurgitation.

METHODS: A 25 mm momentum-nozzle fluid diode valve design made of nonthrombogenic materials was tested in a right heart-pulmonary artery circulatory simulator. The simulator allows control of pulmonary vascular resistance (PVR) and compliance (PVC) over a range of human physiological values using a blood analog. Instantaneous flow rates and pressures, along with detailed velocity fields using particle image velocimetry (PIV), were measured. Data were obtained over a range of PVR values, at a simulated heart rate of 75 beats/min and cardiac output of 6 L/min. Outcomes were regurgitant fraction (RF), peak systolic gradient (PPG), instantaneous velocity fields, and shear stresses.

RESULTS: In the simulator, higher PVR and lower PVC increase fluid diode RF and PPG. Trivial regurgitation (RF < 10%) was obtained at PVR less than 4.0 Wood’s units. For PVR between 4 and 5 Wood’s units, RF was less than 20%. Maximum PPG was 10 mm Hg at PVR of 6 Wood’s units. PIV velocity fields reveal a developing jet-like flow structure through systole. Peak hydrodynamic shear stresses remained below 50 dynes/cm².

CONCLUSION: A motionless fluid diode device limits pulmonary regurgitation in an experimental right heart-pulmonary circulation simulator, with low pressure gradients and shear stresses. Moreover, the fluid diode provides a large undisturbed central flow during systole, without rigid moving leaflets, and can be constructed with single nonthrombogenic material. The fluid diode design may be a permanent pulmonary valve replacement solution with minimal thrombogenic risk.
Photograph of fluid diode, and cross section view of the device.
Truly Stentless Autologous Pericardial AVR: An Alternative to Standard AVR

K.M. John Chan, Jemyrr Therese A. Gavino, Gilles D. Dreyfus*
Cardiothoracic Surgery, Royal Brompton and Harefield NHS Foundation Trust, Harefield, United Kingdom.

OBJECTIVE: The aim of this prospective pilot study was to determine the feasibility and durability of truly stentless AVR using autologous pericardium sutured directly onto the aortic wall. The use of autologous pericardium eliminates any immune reaction between the host and implanted valve avoiding leaflet calcification, and direct suture of the valve onto the aortic wall eliminates the need for a valve stent or sewing ring preserving the normal dynamics of the aorta, and avoids mechanical shear stress from contact of valve leaflets with valve struts.

METHODS: 11 patients (mean age 55.9 years, range 22 to 75 years) requiring AVR were recruited. A circular piece of pericardium about 8 cm in diameter was harvested and prepared. The aortic valve was excised and using specially designed instruments (CardioMend) the sino-tubular junction was sized, the pericardium tailored to the required size and shape, and then sutured directly onto the aortic wall close to the sino-tubular junction. The reconstructed valve geometry and competency was assessed directly and by echo at the end of the operation, and before discharge, and by echo and cardiac MRI at 6 months, and yearly.

Table 1: Comparison of Baseline and Last Follow-Up Data

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Baseline</th>
<th>Last Follow-Up</th>
<th>Change</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA class (1–4)</td>
<td>2.4 (0.5)</td>
<td>1.3 (0.8)</td>
<td>-0.9</td>
<td>0.0047</td>
</tr>
<tr>
<td>LVESD (mm)</td>
<td>27.4 (2.9)</td>
<td>30.2 (2.6)</td>
<td>2.8</td>
<td>0.31</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>44.4 (3.1)</td>
<td>45.2 (3.6)</td>
<td>0.8</td>
<td>0.76</td>
</tr>
<tr>
<td>Peak AV gradient (mmHg)</td>
<td>90.7 (25.2)</td>
<td>10.3 (8.2)</td>
<td>-80.4</td>
<td>0.0005</td>
</tr>
<tr>
<td>Peak AV velocity (m/s)</td>
<td>4.9 (0.8)</td>
<td>1.4 (0.8)</td>
<td>-3.5</td>
<td>0.0005</td>
</tr>
<tr>
<td>Mean AR grade (0–4)</td>
<td>0.7 (0.8)</td>
<td>0.4 (0.5)</td>
<td>-0.3</td>
<td>0.36</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>231 (49.9)</td>
<td>198.6 (49.8)</td>
<td>-32.4</td>
<td>0.14</td>
</tr>
</tbody>
</table>

Data given as mean (S.D.). Mean follow-up 60.2 months (range 51–73 months). *paired t-test.
RESULTS: Hospital mortality was 0%. There were 4 re-operations at 4, 13, 15 and 46 months, 3 of them due to endocarditis (2 of which had endocarditis pre-operatively) and 1 due to technical failure noted at the time of surgery. Histology of the explanted leaflets showed no disruption of the collagen matrix or calcification. The remaining 7 patients are all alive and well. Follow-up was 100% complete with a mean duration of 60.2 months (range 51–73 months). Mean NYHA class was 1.3, all had normally functioning aortic valves with no valve calcification. Freedom from structural valve deterioration excluding endocarditis was 100% at 6 years. Freedom from thromboembolism and endocarditis was 100% and 73% respectively at 6 years.

CONCLUSION: Truely stentless AVR using autologous pericardium sutured directly onto the aortic wall without supporting stents is feasible and has excellent durability excluding endocarditis up to 6 years, with no leaflet calcification. The high rate of endocarditis should indicate caution in the presence of endocarditis. Preservation of the normal physiological dynamics of the aorta without a valve stent or sewing ring, and using autologous pericardium, may offer increased valve durability free of calcification, and may be useful particularly for younger patients who do not want anticoagulation.
T6. Effect of Transapical Aortic Valve Implantation for Aortic Stenosis on Severity of Mitral Regurgitation

Robert L. Smith², Arnaud Van Linden¹, Joerg Kempfert¹, Ines Schimpke¹, Gerhard Schuler¹, Friedrich W. Mohr¹**, Thomas Walther¹

¹. Department of Cardiac Surgery, Heart Center, University of Leipzig, Leipzig, Germany. ². Cardiopulmonary Research Science and Technology Institute, Dallas, TX, USA.

OBJECTIVE: Patients with aortic stenosis (AS) often present with mitral regurgitation (MR). For patients with non-severe MR, treatment of AS with conventional aortic valve replacement will often result in decreased severity of the MR. Transapical aortic valve implanta- tion (TAAVI) using catheter-based technologies is emerging as an effective treatment for AS. It is unknown whether similar improvements in MR severity can be conferred with this therapeutic approach. The objective of this study is to determine if TAAVI will result in decrease severity of regurgitation in patients presenting with non-severe MR. The hypothesis is that TAAVI will reduce the severity of non-severe MR.

METHODS: This is a single center study. Patients identified as having non-severe MR at the time of TAAVI for severe AS and with a one-year follow up echocardiography were included in the study. MR severity was characterized echocardiographically by intra-atrial jet area, vena contracta measurement, proximal isovelocity surface area, and/or effective regurgitation orifice and was categorized as trace (1), mild (2), or moderate (3). A decrease in category by one or more category was considered improvement. Comparisons were by univariate analysis and significance was determined by p value <0.05.

RESULTS: Of the 250 patients who have undergone TAAVI at our institution, 40 patients were identified with relevant MR and appropriate one year follow up. These 40 patients form the study cohort. Except for one case, the same surgical team performed cases. The cohort had an average STS score of 15.4 ± 8.66 and a Euro log-mortality % of 28.8 ± 12.5. Preoperatively, 17.5% (7/40) patients had trace MR, 37.5% (15/40) mild MR, and 45% (18/40) moderate MR. At one year follow up, 10% (4/40) patients had no residual MR, 40% (16/40) mild, and 20% (8/40) moderate. No patient developed severe MR. The average level of MR decreased significantly at one year: 2.3 pre-TAAVI to 1.6 post-TAAVI, p value = 0.001. 22 patients showed decreased severity in MR with a change in one class or more, 13 patients were unchanged, and 5 patients
had worsened MR. There was no statistical difference in ejection fraction in the pre- and post-TAAVI, nor was there a difference in ejection fraction noted within the categories of MR improvement.

**CONCLUSION:** In patients undergoing TAAVI, conservative treatment of non-severe MR is a viable option for this high-risk cohort.
Transapical Transcatheter Mitral Valve in Valve Implantation: A Case Series

Anson Cheung, Jian Ye, John Webb, David A. Wood, Ronald G. Carere, Christopher Thompson, Samuel V. Lichtenstein
University of British Columbia, Vancouver, BC, Canada.

OBJECTIVE: There are a few single case reports on transapical transcatheter valve-in-valve aortic valve implantation (AVI) into failed aortic bioprosthesis in humans. We are now reporting our early experience in transapical transcatheter valve-in-valve AVI without cardiopulmonary bypass in 7 patients.

METHODS: Between April 2006 and August 2009, 7 patients (2 females) underwent transapical transcatheter valve-in-valve AVI with either 23 or 26 mm Edwards-SAPIEN™ balloon-expandable bioprostheses. All patients were declined for conventional reoperative aortic valve replacement (AVR) due to unacceptable operative risks. Two patients underwent rescue valve-in-valve implantation of 23 mm SAPIEN valves into the first unsuccessful 23 mm SAPIEN valves (rescue AVI), and 5 patients underwent planned valve-in-valve implantation of 23 or 26 mm SAPIEN valves into failed previous surgically-implanted aortic tissue valves (21–25 mm valves) (planned AVI). Clinical and echocardiographic follow-ups were performed before discharge, at 1 and 6 months, and then yearly. The mean follow-up was 10.3 ± 9.9 months (1–29 months) with a total of 72 months of follow-up.

RESULTS: Mean age was 80.1 ± 7.8 years and predicted operative mortality was 36.9 ± 9.9% by logistic EuroSCORE and 15.9 ± 10.7% by STS Risk Calculator. The valve-in-valve AVI was successful in all patients. Of two patients with rescue AVI, one had intraoperative apical bleeding and another one died from acute ischemic colitis on postoperative day 10. Of 5 patients with planned AVI, there were no intraoperative complications, and no valve-related complications. No stroke or valve embolization/migration was observed in any patient. Six patients were discharged home with mean hospital stay of 14.3 ± 11.4 days, who have been doing well at the time of last follow-up. NYHA class improved from preoperative III-IV to postoperative I-II in all patients. Echocardiographic follow-up demonstrated stable valve position and valve function, no aortic regurgitation in 5 patients, and mild AI in 1 patient.

CONCLUSION: Transcatheter transapical valve-in-valve AVI of balloon-expandable valves into failed bioprostheses is feasible with acceptable mortality and morbidity and could be a viable approach for selected high risk patients with failed bioprostheses. Further clinical assessments are required.

Zachary N. Kon, Amod Tendulkar, Zhongjun Wu, Aldo T. Iacono, Brian McCormick, Bartley P. Griffith*, Jose P. Garcia

*University of Maryland School of Medicine, Baltimore, MD, USA.

**OBJECTIVE:** End-stage lung disease is a complex entity that remains a challenge to manage. Successful therapies include early use of mechanical ventilation, complex fluid regimens, and pulmonary vasodilators. Despite these advances, some patients remain with insufficient pulmonary gas exchange. We investigated the use of extra corporeal membrane oxygenation (ECMO) via cannulation of the internal jugular (IJ) vein with a novel dual-lumen single catheter system as a treatment strategy for this group of patients.

**METHODS:** In this pilot study, we enrolled 10 patients (mean age of 45.3, 80% male) with severe end-stage lung disease (idiopathic pulmonary fibrosis 40%, pneumonia/ARDS 40%, COPD 10%, and pulmonary hypertension 10%), who were unresponsive to conventional treatment with intensive mechanical ventilation and adjunct therapies. ECMO was initiated and minimal mechanical ventilatory support was used in an effort to “rest” the injured lungs. The patients were either intended to be weaned from respiratory support or bridged to transplantation.

**RESULTS:** The mean length of time on ECMO was 20 (9–59) days, with a mean blood flow of 3.5 (1.6–4.9) L/min, and mean CO2 removal and oxygen transfer of 228 (54–570) mL/min and 127 (36–529) mL/min, respectively. 60% of patients were either weaned from all respiratory support (N = 4) or transplanted (N = 2) and survived to discharge from the hospital. Cause of death for the remaining four patients was sepsis (N = 2), renal failure (N = 1), and stroke (N = 1). Four of the six surviving patients were able to be extubated and ambulatory while still on ECMO. During that time, three of the four patients exercised at the bedside, with the remaining patient able to undergo full cardiopulmonary rehab, including treadmill walking.

* AATS Member
CONCLUSION: Although the use of ECMO for end-stage lung disease is not a new idea, this study illustrates a novel approach of its use. Because of the single IJ cannulation, early ambulation and the avoidance of further deconditioning can be achieved. Pending further studies, perhaps this approach could be utilized even earlier in severe lung disease.
T9. Preliminary Results of Anatomic Lung Resection Utilizing the Liga Sure Energy Based Tissue and Vessel Coagulative Fusion Technology

Matthew J. Schuchert, Ghulam Abbas, Brian L. Pettiford, James D. Luketich*, Rodney J. Landreneau*

Heart, Lung and Esophageal Surgery Institute, University of Pittsburgh Medical Center, Pittsburgh, PA, USA.

OBJECTIVE: Mechanical stapling devices are established as the mainstay of therapy in the selective isolation and division of vascular structures during anatomic lung resection. Little data currently exists regarding the application of energy-based tissue and vessel fusion technology during anatomic lung resection. We evaluated the use of the LigaSure™ (Valley Lab, Boulder, Co) device for vascular division during anatomic lung resection.

METHODS: Anatomic lung resection was performed initially utilizing the LigaSure Impact™ (n = 12) device, and subsequently with the Atlas™ (n = 187) hand-controlled instruments. The Impact™ device working jaw achieves a seal 36 mm in length and a variable width from tip to base of 3.3–4.7 mm. The jaw also has a 14-degree curvature, facilitating passage around vascular structures. In contrast, the LigaSure Atlas™ instrument has a uniform coagulating surface of 22 mm in length and 6 mm in width. Two energy applications are applied prior to vessel division. We initially chose the Impact™ device because of the increased working length, allowing for apposition across main pulmonary veins and large basilar arterial trunks. The LigaSure device is currently the only FDA-approved device for vascular fusion and division during pulmonary surgery.

RESULTS: The LigaSure system was utilized in 199 patients from 2008–2009 (104 lobectomies and 95 anatomic segmentectomies). Initially, the LigaSure Impact™ device was employed (n = 12 patients, 15 vascular fusions). There were two of five basilar partial arterial dehiscences, and seven partial pulmonary venous dehiscences that were recognized and controlled intra-operatively with local suture ligation. Due to the noted vascular dehiscences, we converted to the shorter but wider Atlas™ device. We have found that two serial applications of the energy to the first order pulmonary venous branches prior to peripheral division appears safer than dividing the main pulmonary venous trunks.

*AATS Member
CONCLUSION: The LigaSure Atlas™ system provides safe and reliable control of pulmonary arterial and venous branches during anatomic lung resection. The use of energy-based tissue fusion technology represents a reasonable alternative to mechanical stapling devices during anatomic lung resection.
T10. The Novel Use of Coil Spring Fiducials Placed via Navigation Bronchoscopy in Inoperable Patients Allows for the Safe and Effective Delivery of Cyberknife Stereotactic Radiation

Carsten Schroeder\(^1\), Rana Hejal\(^2\), Philip Linden\(^1\)

\(^1\) Thoracic & Esophageal Surgery, Case Medical Center, Cleveland, OH, USA. \(^2\) Pulmonary Critical Care & Sleep Medicine, Case Medical Center, Cleveland, OH, USA.

OBJECTIVE: Stereotactic radiosurgery (Cyberknife) is a treatment option for patients who are medically unfit to undergo lung tumor resection. For precise tumor ablation, the Cyberknife requires fiducial marker placement in or near the target tumor. Fiducial placement under transthoracic CT guidance is associated with a high risk of iatrogenic pneumothorax. Electromagnetic navigation bronchoscopy (ENB) offers a less morbid alternative to accurately deploy fiducials to bronchoscopically invisible peripheral lung lesions. Prior studies, in which linear markers were used, showed at least a 10% dislocation rate and required general anesthesia for placement. We propose the use of coil-spring fiducials placed under moderate sedation in an outpatient bronchoscopy suite setting to decrease these complications.

METHODS: 27 Consecutive non-surgical patients with isolated lung tumors underwent fiducial placement using ENB under moderate sedation in an outpatient bronchoscopy suite. Four patients received 17 linear fiducials and 23 patients with 28 tumors received 104 coil-spring fiducials. The procedures were considered successful if fiducials were placed in or near the tumors and remained in place without migration allowing radiosurgery to proceed. The need for alternative or additional intrathoracic fiducial placement was documented as procedure failure.

RESULTS: A total of 121 fiducials markers were successfully deployed in 27 patients with 32 tumor locations (mean diameter 21.6 mm). 17 tumors (53%) were adjacent to the pleura. 13 patients (48%) underwent concomitant transbronchial biopsy. At Cyberknife planning a week after placement, 8 of 17 linear fiducial markers (47%) and 100 of 104 coil-spring fiducials (96%) were still in place. 2/4 patients with linear fiducials required additional CT guided fiducials; none of the coil fiducial patients required additional procedures. Two pneumothoraces (6%) occurred after transbronchial biopsy (one treated with a pig-tail chest tube and one with observation only).
CONCLUSION: ENB can be used to deploy fiducial markers for Cyberknife radiosurgery of lung tumors safely and accurately with fewer complications than via CT guided placement. Transbronchial biopsies can be performed in the same setting. Coil-spring fiducials rarely dislocate and therefore reduce the re-procedure rate and/or Cyberknife tracking errors. The procedure can be performed safely in an outpatient bronchoscopy suite setting under moderate sedation.
9:00 a.m.  
CONTROVERSIES IN CARDIOTHORACIC SURGERY

Hall C, Metro Toronto Convention Centre
Moderator: Irving L. Kron, MD

Randomized Controlled Clinical Trials are Necessary to Evaluate New Surgical Operations

Pro: Timothy J. Gardner, MD
Con: Joel D. Cooper, MD
10:00 a.m.  

**ADULT CARDIAC: SURGICAL THERAPIES FOR CONGESTIVE HEART FAILURE**  
*Constitution 107, Metro Toronto Convention Centre*  
**Chairs:** Vivek Rao, MD, PhD  
University of Toronto  
Thoralf M. Sundt, III, MD  
Mayo Clinic

10:00 a.m. – 10:20 a.m.  
**Medical Management of CHF: “What the Surgeon Needs to Know”**  
Heather J. Ross, MD, MHSc  
University of Toronto

**Patients at Risk of Developing Heart Failure**

- Clinical assessment is recommended in all patients to identify known or potential risk factors for heart failure:
  - Hypertension
  - Ischemic Heart Disease
  - Diabetes Mellitus
  - Hyperlipidemia
  - Smoking
  - Obesity-independent risk factor due to changes in LV structure and function

All modifiable risk factors for heart failure, including those for coronary artery disease, such as hypertension, diabetes and hyperlipidemia, should be treated in accordance to current national guidelines.

**Diagnosis**

- The diagnosis of clinical heart failure is made when symptoms and signs of impaired cardiac output and/or volume overload are documented in the setting of abnormal systolic and/or diastolic cardiac function.
Pathogenesis

Suspected heart failure

Clinical history
- Symptoms
- Functional limitation
- Prior cardiac diseases
- Risk factors
- Exacerbating factors
- Comorbidities
- Drugs

Physical examination
- Vital signs
- Weight
- Volume status
- Cardiac
- Pulmonary
- Abdominal
- Vascular

Initial investigations
- Chest radiograph
- Electrocardiogram
- Natriuretic peptides
- Other blood work

Diagnosis excluded
- Normal
- Abnormal

Pathology excluded
- Normal
- Abnormal

Assessment of ventricular function
- Echocardiogram
- Inconclusive

Additional diagnostic investigations
- Radionuclide imaging
- Cardiac catheterization
- Cardiopulmonary exercise testing
- Others

No heart failure

Heart failure
Initial investigations should be targeted to confirm or exclude heart failure as the diagnosis and to identify systemic disorders that may affect its development or progression.

Two-dimensional and Doppler transthoracic echocardiography are the initial imaging modalities of choice in patients suspected to have heart failure because they assess systolic and diastolic ventricular function, wall thickness, chamber sizes, valvular function and pericardial disease.

**Clinical History & Physical Examination**

Relevant clinical history, physical examination should be performed on all patients to assess their current status and response to treatment, as well as identify modifiable factors that may affect the progression of heart failure. These risk factors can be targeted for potential goal setting and patient self-management.

**Laboratory Testing & Monitoring**

When initiating or significantly adjusting the dosage of ACEI, ARBs, Spironolactone and diuretics, blood work should be checked for electrolyte status and renal function with close monitoring as required.

**Treatment**

[Diagram of treatment flowchart]
References


Introduction

Both implantable cardioverter defibrillators (ICD) and cardiac resynchronization therapy (CRT) devices have important roles in the management of left ventricular systolic dysfunction (LVSD). A multitude of well designed, randomized clinical trials have demonstrated the beneficial role of these devices, and they are accepted therapy for patients with LVSD and heart failure. Although the devices are often used in combination (that is, as a CRT-D configuration), ICD may be implanted as a single or double chamber device, and CRT may be implanted as a two- or three-lead pacemaker system.

As a general overview, ICD is utilized for both at risk patients with LVSD as a primary prevention, prophylactic device, and as a secondary prevention device for those with aborted sudden cardiac death or ventricular tachycardia but it does not provide any benefit to mechanical function or structural remodelling. In contrast, CRT, which can be coupled with an ICD or function solely as a pacemaker, has mechanical benefits and can lead to structural reverse remodelling for optimally managed stage C patients with LVSD who have class 3 symptoms, a broad QRS on ECG (greater than 120 ms) and a left ventricular ejection fraction of less than 35%.

In considering ICD and/or CRT after cardiac surgery, little data exists comparing outcomes for patients who have had surgery (be it revascularization, valvular, or other) or not. Differences exist certainly between non-ischemic and ischemic cardiomyopathies and their responses to these therapies, along with varying incidences of sudden cardiac death and variable disease progression because of the underlying disease processes. Ultimately, both devices, in appropriate patients, are indicated in those who have had cardiac surgery although there are nuances specific to both.

ICD after Cardiac Surgery

The major randomized clinical trials have included a proportion of patients with prior cardiac surgery, though not usually stated in the baseline
characteristics of the patient cohort. Exclusion criteria do not exclude typically patients with prior cardiac surgery except for transplant recipients and significant valvular heart disease. Compared with CRT, one randomized clinical trial has specifically evaluated the role of prophylactic ICD after cardiac surgery. The CABG-PATCH trial, published in 1997, evaluated the efficacy for reducing overall mortality of an epicardial ICD lead implanted at the time of CABG in 900 patients with severe coronary artery disease undergoing revascularization and with ejection fraction less than 35% and a positive signal averaged ECG, as a marker of arrhythmia risk, but no prior history of ventricular tachyarrhythmias. This trial was important for being a negative trial, even though ICD therapy reduced arrhythmic death in a trial with predominately non-arrhythmic deaths. The cause was likely the positive effect of revascularization to decrease the incidence of sudden cardiac death and increase ejection fraction. Accordingly, prophylactic ICD is not indicated in the immediate post-CABG period. A similar finding occurred in trials of post-MI prophylactic ICD, with no benefit in the immediate post-MI period, although ongoing LVSD remote from the MI and/or revascularization remains an indication. Of course, there is the need to individualize care and to ensure that at risk patients are not exposed to a window period of no coverage when other factors are suggesting high risk. Another consideration is the role of ICD after surgical ventricular restoration procedures, however further data is required.

**CRT after Cardiac Surgery**

Regarding CRT after cardiac surgery, this therapy can be considered in both the chronic, implantable form, plus acute management post-cardiotomy and cardiopulmonary bypass. The benefit of CRT is based on the concept that an intraventricular conduction delay, such as left bundle branch block which is seen in 30–50% of HF patients, can lead to ventricular dyssynchrony and resultant contractile inefficiencies, and accordingly can worsen HF in patients with LVSD and lead to progressive structural remodelling. Similar changes are seen with RV apical pacing in patients with LVSD. There is a wealth of clinical data, and increasing basic data, establishing the benefit of atrio-biventricular pacing (or CRT).

In most institutions, the majority of devices are implanted endocardially, either as a de novo procedure or as an upgrade from an existing system. Typically, a bipolar or unipolar lead is implanted into a lateral or posterior venous branch of the coronary sinus accessed via the right atrium utilizing
a variety of standard and specialist catheters, interventional guidewires and over-the-wire leads. Further, left ventricular leads can be implanted epicardially via a variety of surgical techniques, but for the most part this remains a technique when technical issues identified prior to attempted implantation or following failure require the lead to be implanted epicardially. It does remain institution dependent to an extent, however. In a subgroup of patients undergoing cardiac surgery with LVSD, consideration to “prophylactic” implantation of an epicardial LV lead can be made, although there is little data that such an option is clinically indicated. Further, it raises concerns since epicardial leads do not necessarily have the same durability as endocardial leads, particularly if they are not utilized immediately from implantation.

A significant proportion of patients with prior cardiac surgery have presumably been enrolled in the major CRT trials, but little data exists in the published literature, and specifically the COMPANION and CARE-HF trials did not include these patients. There is a paucity of data comparing the impact of cardiac surgery on the success, immediate and delayed, of implantable CRT. Also, an important consideration of CRT after cardiac surgery is the likelihood of technical issues arising in relation to anatomical or structural changes following surgery. Although there is little available published data, it is not unusual to find absent veins corresponding to grafted arteries, and similarly the coronary venous branches can be entrapped by sutures in the vicinity of the mitral annulus. In these circumstances, it is often prudent to perform imaging studies prior to CRT implantation with cardiac CT, cardiac MRI or levophase coronary angiography to evaluate coronary venous anatomy. Again, anatomical issues precluding a transvenous approach may require epicardial implantation. Alternative surgical approaches to LV lead implantation, such as transapically or via a subxiphoid intrapericardial approach, are being evaluated.

Mentioned here for completeness is the role of CRT following cardiac transplant. Little published data exists of the role of CRT in transplant recipients, and reflects the differing pathophysiology of LVSD in transplant recipients, and the fact that a right, and not left, bundle branch block is more common. Further, only a small proportion of patients require pacing for standard indications, which minimizes the incidence of LV dysfunction due to right ventricular apical pacing.

Increasing interest has turned to epicardial post-cardiotomy atrio-biventricular pacing compared with standard atrio-right ventricular pacing in patients
requiring pacing. There have been several hemodynamic studies and single centre clinical trials. The premise for routine clinical use is founded upon the fact the right ventricular pacing has a detrimental effect on hemodynamic function of normal and compromised ventricles, and that this can be partially reversed or even protected by left ventricular or biventricular pacing. The results have been conflicting amongst those studies evaluating hemodynamic improvements and those evaluating clinical outcomes, and it may reflect the heterogeneous populations evaluated. A clear difference between endocardial and epicardial pacing of the right ventricle is the positioning of the leads, since there is a more physiological electrical activation with pacing over the RVOT compared with the RV apical septum. Similarly, the studies have demonstrated no real consistency on placement of the LV lead. Regardless, with the acute hemodynamic benefits seen in non-cardiotomy patients, further evaluation is required and presumably a specific subgroup will be identified that benefit, such as those with low LVEF and broad QRS.

Summary

ICD and CRT are indicated for patients with severe LVSD. ICD is indicated with prior MI and LVEF less than 30% (MADIT-II criteria) and those with EF less than 35% and NYHA class 2 or 3 HF symptoms (SCD-HeFT criteria). For patients with recent MI (less than 40 days) or within 3 months of PCI or CABG, prophylactic ICD is not indicated, and in these circumstances additional assessment may be required such as with electrophysiology testing. For CRT, this treatment is indicated for those with LVEF less than 35%, QRSd greater than 120 ms and NYHA class 3 or ambulatory class 4 symptoms, although there is likely to be extension of indication soon. Implantation immediately post-surgery is unclear and clinical trials are indicated. Additionally, the utilization atrio-biventricular temporary pacing wires post-cardiotomy remains an area of conflicting studies and a multi-centre trial with tightening of patient selection along with pre-determined pacing lead positions is indicated.
References


10:40 a.m. – 11:00 a.m. Life after STICH: When Do We Repair the Dysfunctional LV?
Robert E. Michler, MD
Albert Einstein Medical College

BACKGROUND: Surgical ventricular reconstruction is a specific procedure designed to reduce left ventricular volume in patients with heart failure caused by coronary artery disease. We conducted a trial to address the question of whether surgical ventricular reconstruction added to coronary-artery bypass grafting (CABG) would decrease the rate of death or hospitalization for cardiac causes, as compared with CABG alone.

METHODS: Between September 2002 and January 2006, a total of 1000 patients with an ejection fraction of 35% or less, coronary artery disease that was amenable to CABG, and dominant anterior left ventricular dysfunction that was amenable to surgical ventricular reconstruction were randomly assigned to undergo either CABG alone (499 patients) or CABG with surgical ventricular reconstruction (501 patients). The primary outcome was a composite of death from any cause and hospitalization for cardiac causes. The median follow-up was 48 months.
RESULTS: Surgical ventricular reconstruction reduced the end-systolic volume index by 19%, as compared with a reduction of 6% with CABG alone. Cardiac symptoms and exercise tolerance improved from baseline to a similar degree in the two study groups. However, no significant difference was observed in the primary outcome, which occurred in 292 patients (59%) who were assigned to undergo CABG alone and in 289 patients (58%) who were assigned to undergo CABG with surgical ventricular reconstruction (hazard ratio for the combined approach, 0.99; 95% confidence interval, 0.84 to 1.17; P = 0.90).

CONCLUSIONS: Adding surgical ventricular reconstruction to CABG reduced the left ventricular volume, as compared with CABG alone. However, this anatomical change was not associated with a greater improvement in symptoms or exercise tolerance or with a reduction in the rate of death or hospitalization for cardiac causes. (ClinicalTrials.gov number, NCT00023595.)

Coronary artery disease is the pre-dominant cause of heart failure, which is a major cause of death and disability throughout the world. Evidence-based medical therapy has been shown to reduce symptoms and increase survival in patients with heart failure and coronary artery disease. In addition, selected patients may benefit from surgical revascularization by means of coronary-artery bypass grafting (CABG), especially if the coronary anatomy is suitable for such surgery and if there is evidence of myocardial viability.

The reduction in left ventricular function that can occur after myocardial infarction is typically accompanied by left ventricular enlargement and changes in chamber geometry. Left ventricular remodeling is correlated with progression of heart failure and a poor prognosis, and the beneficial effects of therapeutic agents such as angiotensin-converting–enzyme (ACE) inhibitors and beta-blockers are associated with their effect on remodeling. These findings have generated considerable interest in the possibility that a surgical approach to remodeling through left ventricular volume reduction could improve outcomes for patients with coronary artery disease and heart failure.

Surgical ventricular reconstruction is a specific surgical procedure developed for the management of heart failure with left ventricular remodeling caused by coronary artery disease. This operation has been shown
to reduce the left ventricular volume, increase the ejection fraction, and improve ventricular function.\textsuperscript{12,13} On the basis of a small, nonrandomized, case–control study,\textsuperscript{14} it has been suggested that surgical ventricular reconstruction that is performed together with CABG may reduce the rate of hospitalization and improve ventricular function to a greater degree than CABG alone.

The Surgical Treatment for Ischemic Heart Failure (STICH) trial was designed to define the role of cardiac surgery in the treatment of patients with heart failure and coronary artery disease.\textsuperscript{15,16} One of the two major hypotheses of this trial (Hypothesis 2) was that surgical ventricular reconstruction, when added to CABG, would decrease the rate of death or hospitalization for a cardiac event, as compared with CABG alone.

**Methods**

**Study Design**

We conducted a multicenter, nonblinded, randomized trial at 127 clinical sites in 26 countries.\textsuperscript{15} The trial was sponsored by the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health. Additional support was provided by Abbott Laboratories, Chase Medical, and CV Therapeutics, which had no role in the design, conduct, or reporting of the trial. The trial protocol was designed by the authors in collaboration with the NHLBI and was approved by the appropriate institutional review board or ethics committee at each study center. Trial operations, site management and monitoring, and data collection and analysis were coordinated by the Duke Clinical Research Institute. Oversight was provided by an independent data and safety monitoring board. A clinical events committee whose members were unaware of study-group assignments adjudicated primary outcome events. The authors wrote the manuscript and vouch for the completeness and accuracy of the data and the analyses.

**Selection of Patients and Randomization**

Patients were eligible for enrollment if they had coronary artery disease that was amenable to CABG and if they had a left ventricular ejection fraction of 35% or less (Fig. 1). Exclusion criteria were a recent myocardial infarction, a need for aortic-valve replacement, a planned percutaneous coronary intervention (PCI), and coexisting noncardiac disease resulting in a life expectancy of less than 3 years. All patients provided written informed consent.
After initial determination of overall eligibility for the trial, patients were evaluated to determine which component of the STICH program was appropriate for them on the basis of suitable therapeutic options for that patient (medical therapy alone, CABG alone, or CABG plus surgical ventricular reconstruction) (Fig. 1). Patients who had stenosis of the left main coronary artery of 50% or more or who had angina of Canadian Cardiovascular Society (CCS) class III or IV while receiving medical therapy were not eligible for medical therapy alone. All patients underwent cardiac imaging for assessment of left ventricular function and wall motion.
Patients who were found to have dominant anterior akinesia or dyskinesia of the left ventricle were considered to have disease that was amenable to surgical ventricular reconstruction.

Using these guidelines, physicians who were responsible for the conduct of the trial selected the randomization stratum for each patient that appeared to offer treatment possibilities with equivalent potential risks and benefits. Permutated-block randomization was used, with stratification according to clinical site and according to whether the patient was a candidate for SVR, for medical therapy alone, or for both (Fig. 1).

The resulting trial included two major components. Patients in the Hypothesis 1 component were randomly assigned to receive either medical therapy alone or medical therapy plus CABG. The Hypothesis 1 component of the trial is ongoing. Patients in the Hypothesis 2 component were randomly assigned to receive either medical therapy plus CABG or medical therapy plus CABG and surgical ventricular reconstruction. The results of the Hypothesis 2 component are the subject of this report.

**Treatment**

Guideline-based recommendations for drug and device use were emphasized for all patients. The lead cardiologist at each site was responsible for monitoring to ensure that ACE inhibitors, angiotensin-receptor blockers, beta-blockers, aldosterone antagonists, antiplatelet agents, statins, diuretics, digitalis, pacemakers (for bradyarrhythmias or for cardiac resynchronization), and implantable cardioverter–defibrillators were used properly throughout the study.

Cardiac surgeons were individually certified to participate in the trial if they met prespecified performance criteria. For CABG certification, surgeons were required to provide data on at least 25 patients with a left ventricular ejection fraction of 40% or less who underwent CABG, with a death rate of 5% or less. Education in the operative technique for surgical ventricular reconstruction and perioperative management was made available before patient enrollment and during investigator meetings. Certification of individual surgeons for performing surgical ventricular reconstruction required evidence of a consistent postoperative decrease in left ventricular volume in five consecutive patients who survived the operation.

During CABG, arterial grafting for stenosis of the left anterior descending coronary artery was required for all patients without specific contraindications.
The use of additional arterial conduits supplemented by vein grafts was recommended for revascularization of all major vessels with clinically significant stenoses. Concurrent mitral-valve surgery for regurgitation was performed at the discretion of the surgeon.

The technique of surgical ventricular reconstruction has been described previously.\textsuperscript{11,12,17} For patients who were assigned to undergo surgical ventricular reconstruction, this component of the operation was most commonly performed during a single period of cardioplegic arrest after construction of bypass grafts. However, the procedure could also be performed with the heart beating in order to facilitate identification of the noncontractile zone of scarring. In this procedure, after an anterior left ventriculotomy is centered in the zone of anterior asynergy, a suture is placed in the interior of the ventricle to encircle the scar at the boundary between the akinetic and viable tissue. Tightening of this suture brings the healthy portions of the ventricular walls together. Visual inspection and palpation facilitate the judgment of whether a patch is needed to optimize the chamber size without deforming the left ventricle during closure of the ventriculotomy.

**Primary and Secondary Outcomes**

Major perioperative events and specified end points were recorded at discharge or at 30 days for patients remaining in the hospital. Patients were evaluated at 4-month intervals after randomization during the first year and thereafter at 6-month intervals.

Symptoms of angina and heart failure were assessed at each follow-up visit. All patients who were able to do so performed a 6-minute walk test at baseline, at 4 months, and annually thereafter. Left ventricular volumes and function were assessed with the use of echocardiography, cardiac magnetic resonance imaging, or single photon emission computed tomography at baseline, at 4 months, and at 2 years.

The primary outcome was the time to death from any cause or hospitalization for cardiac causes. Secondary outcomes included death from any cause at 30 days, hospitalization for any cause and for cardiovascular causes, myocardial infarction, and stroke.

**Statistical Analysis**

We calculated that we would need to enroll 1000 patients in the trial for a power of 90% to detect a 20% reduction in the relative risk of death or...
hospitalization for cardiac causes, assuming a 3-year event rate in the CABG-only group of 45% or more and allowing for a crossover rate of up to 20%.

All major study-group comparisons were performed according to the intention-to-treat principle. Supplementary analyses tabulated postoperative complications and clinical events occurring within 30 days, according to the type of operation that was performed. All statistical tests were two-tailed. Cumulative event rates from the time of randomization were calculated with the use of the Kaplan–Meier method. The log-rank test for time-to-event data was used for the statistical comparison of study groups with respect to the primary outcome and overall mortality. Hazard ratios with associated 95% confidence intervals were derived with the use of the Cox proportional hazards model. The Cox model was also used to assess the consistency of treatment effects by testing for interactions between the type of surgery and prespecified baseline characteristics.

Eight interim analyses of the data were performed and reviewed by the data and safety monitoring board. Interim comparisons between study groups were monitored with the use of two-sided, symmetric O’Brien–Fleming boundaries generated with the alpha-spending-function approach to group-sequential testing. A P value of 0.05 or less was considered to indicate statistical significance. Because of the sequential monitoring, the level of significance that was required for the primary analysis at the completion of the study was 0.04.

Results

Study Population

Between July 24, 2002, and May 5, 2007, 2136 patients were enrolled in the overall STICH trial (Fig. 1). Of these patients, 1000 were enrolled in the Hypothesis 2 component at 96 clinical sites between September 12, 2002, and January 24, 2006, and were randomly assigned to undergo either CABG alone (499 patients) or CABG with surgical ventricular reconstruction (501 patients), with follow-up continued through December 31, 2008. No significant differences between the two study groups were observed in baseline demographic or clinical characteristics (Table 1). The median age was 62 years, and 147 of the 1000 patients were women. The median left ventricular ejection fraction was 28%. The median end-systolic volume index was 82 ml per square meter of body-surface area. Multivessel coronary artery disease was present in 913 patients; 197 patients had stenosis of the left main coronary artery.
# Table 1. Baseline Characteristics of the Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>CABG Alone (N=499)</th>
<th>CABG with Surgical Ventricular Reconstruction (N=500)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age — yr</td>
<td>Median 62</td>
<td>62</td>
</tr>
<tr>
<td></td>
<td>Interquartile range 54–69</td>
<td>55–69</td>
</tr>
<tr>
<td>Female sex — no. (%)</td>
<td>78 (16)</td>
<td>69 (14)</td>
</tr>
<tr>
<td>Race — no. (%)</td>
<td>White 452 (90)</td>
<td>466 (92)</td>
</tr>
<tr>
<td></td>
<td>Black or other</td>
<td>48 (10)</td>
</tr>
<tr>
<td></td>
<td>41 (8)</td>
<td></td>
</tr>
<tr>
<td>Body-mass index</td>
<td>Median 27</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Interquartile range 25–30</td>
<td>24–30</td>
</tr>
<tr>
<td><strong>Medical history</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction — no. (%)</td>
<td>435 (87)</td>
<td>437 (87)</td>
</tr>
<tr>
<td>Hyperlipidemia — no. (%)</td>
<td>367 (74)</td>
<td>331 (70)</td>
</tr>
<tr>
<td>Hypertension — no. (%)</td>
<td>289 (58)</td>
<td>296 (59)</td>
</tr>
<tr>
<td>Diabetes — no. (%)</td>
<td>173 (35)</td>
<td>171 (34)</td>
</tr>
<tr>
<td>Current smoker — no. (%)</td>
<td>117 (23)</td>
<td>100 (20)</td>
</tr>
<tr>
<td>Previous percutaneous coronary intervention — no. (%)</td>
<td>100 (20)</td>
<td>95 (19)</td>
</tr>
<tr>
<td>Chronic renal insufficiency — no. (%)</td>
<td>42 (8)</td>
<td>43 (9)</td>
</tr>
<tr>
<td>Stroke — no. (%)</td>
<td>28 (6)</td>
<td>28 (6)</td>
</tr>
<tr>
<td>Current Canadian Cardiovascular Society angina class — no. (%)</td>
<td>15 (3)</td>
<td>9 (2)</td>
</tr>
<tr>
<td>No angina</td>
<td>121 (24)</td>
<td>128 (26)</td>
</tr>
<tr>
<td>I</td>
<td>36 (7)</td>
<td>35 (7)</td>
</tr>
<tr>
<td>II</td>
<td>94 (19)</td>
<td>94 (19)</td>
</tr>
<tr>
<td>III</td>
<td>203 (41)</td>
<td>205 (41)</td>
</tr>
<tr>
<td>IV</td>
<td>45 (9)</td>
<td>39 (8)</td>
</tr>
<tr>
<td>Current New York Heart Association heart failure class — no. (%)</td>
<td>31 (6)</td>
<td>26 (5)</td>
</tr>
<tr>
<td>I</td>
<td>16 (7)</td>
<td>16 (10)</td>
</tr>
<tr>
<td>II</td>
<td>222 (44)</td>
<td>207 (41)</td>
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<tr>
<td>III</td>
<td>210 (42)</td>
<td>218 (44)</td>
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<tr>
<td>IV</td>
<td>32 (6)</td>
<td>32 (6)</td>
</tr>
<tr>
<td>Blood pressure — mm Hg</td>
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<td></td>
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<tr>
<td>Systolic</td>
<td>Median 120</td>
<td>120</td>
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<tr>
<td></td>
<td>Interquartile range 110–130</td>
<td>110–130</td>
</tr>
<tr>
<td>Diastolic</td>
<td>Median 71</td>
<td>71</td>
</tr>
<tr>
<td></td>
<td>Interquartile range 65–80</td>
<td>66–80</td>
</tr>
<tr>
<td>Pulse — beats/min</td>
<td>Median 70</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td>Interquartile range 64–80</td>
<td>64–80</td>
</tr>
<tr>
<td>Variable</td>
<td>CABG Alone (N=499)</td>
<td>CABG with Surgical Ventricular Reconstruction (N=508)</td>
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<tr>
<td>-----------------------------------------------</td>
<td>--------------------</td>
<td>-------------------------------------------------------</td>
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<tr>
<td><strong>Baseline laboratory measure</strong></td>
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<td></td>
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<tr>
<td>Creatinine — mg/dl</td>
<td>Median: 1.1</td>
<td>Median: 1.1</td>
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<tr>
<td></td>
<td>Interquartile range: 0.9–1.3</td>
<td>Interquartile range: 0.9–1.3</td>
</tr>
<tr>
<td><strong>Left ventricular function</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular ejection fraction — %</td>
<td>Median: 28</td>
<td>Median: 28</td>
</tr>
<tr>
<td></td>
<td>Interquartile range: 21–31</td>
<td>Interquartile range: 24–31</td>
</tr>
<tr>
<td><strong>End-systolic volume index — ml/m²</strong></td>
<td>Median: 82</td>
<td>Median: 82</td>
</tr>
<tr>
<td></td>
<td>Interquartile range: 65–102</td>
<td>Interquartile range: 66–102</td>
</tr>
<tr>
<td><strong>Aloisits or dyskinesis of anterior wall — %</strong></td>
<td>Median: 56</td>
<td>Median: 50</td>
</tr>
<tr>
<td></td>
<td>Interquartile range: 40–60</td>
<td>Interquartile range: 40–60</td>
</tr>
<tr>
<td><strong>Mitral regurgitation — no. (%)†</strong></td>
<td>None or trace: 173 (25)</td>
<td>190 (30)</td>
</tr>
<tr>
<td></td>
<td>Mild (1+) 233 (47)</td>
<td>216 (43)</td>
</tr>
<tr>
<td></td>
<td>Moderate (3+) 72 (14)</td>
<td>70 (14)</td>
</tr>
<tr>
<td></td>
<td>Severe (4+) 16 (3)</td>
<td>20 (4)</td>
</tr>
<tr>
<td></td>
<td>Not assessed 5 (1)</td>
<td>5 (1)</td>
</tr>
<tr>
<td><strong>Coronary anatomy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of vessels with stenosis of ≥50% — no. (%)</td>
<td>One: 36 (7)</td>
<td>51 (10)</td>
</tr>
<tr>
<td></td>
<td>Two: 144 (29)</td>
<td>131 (26)</td>
</tr>
<tr>
<td></td>
<td>Three: 319 (44)</td>
<td>319 (44)</td>
</tr>
<tr>
<td>Stenosis of left main coronary artery — no. (%)</td>
<td>50-74%: 72 (14)</td>
<td>61 (12)</td>
</tr>
<tr>
<td></td>
<td>≥75%: 31 (6)</td>
<td>33 (7)</td>
</tr>
<tr>
<td>≥75% Stenosis of proximal left anterior descending coronary artery — no. (%)</td>
<td>388 (78)</td>
<td>369 (74)</td>
</tr>
<tr>
<td><strong>Duke Coronary Artery Disease Severity Index</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median — %</td>
<td>65</td>
<td>65</td>
</tr>
<tr>
<td>Interquartile range — %</td>
<td>43–91</td>
<td>39–91</td>
</tr>
<tr>
<td><strong>Medication at baseline — %</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>85</td>
<td>87</td>
</tr>
<tr>
<td>ACE inhibitor or angiotensin receptor blocker</td>
<td>87</td>
<td>89</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>80</td>
<td>82</td>
</tr>
<tr>
<td>Diuretic</td>
<td>37</td>
<td>34</td>
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<tr>
<td>Aspirin</td>
<td>69</td>
<td>66</td>
</tr>
<tr>
<td>Aspirin or warfarin</td>
<td>81</td>
<td>83</td>
</tr>
<tr>
<td>Statin</td>
<td>79</td>
<td>75</td>
</tr>
</tbody>
</table>

* P<0.05 for all between-group comparisons. The body-mass index is the weight in kilograms divided by the square of the
Surgical Procedures

Of the 499 patients who were assigned to undergo CABG alone, 463 (93%) underwent the assigned procedure; 9 did not undergo any surgery, and 27 underwent CABG with surgical ventricular reconstruction (Table 1 in the Supplementary Appendix, available with the full text of this article at NEJM.org). Of the 501 patients who were assigned to undergo CABG with surgical ventricular reconstruction, 454 (91%) underwent the assigned procedure; 12 patients did not undergo any surgery, and 35 patients underwent CABG without surgical ventricular reconstruction. Of the 979 patients who underwent surgery, the procedure was elective in 819 patients (84%), urgent in 127 (13%), performed for ongoing ischemia in 21 (2%), and performed under emergency conditions in 11 (1%) (Table 2 in #insert#).
the Supplementary Appendix). Mitral-valve surgery was performed in 178 patients (18%) undergoing surgery. More arterial conduits were used in patients undergoing CABG alone than in patients undergoing CABG with surgical ventricular reconstruction (P = 0.008). Surgical ventricular reconstruction added a median of 27 minutes of cardiopulmonary bypass time to the CABG procedure (P < 0.001). The duration of aortic cross-clamping, the time to endotracheal extubation, and the duration of postoperative hospitalization were longer for patients undergoing CABG with surgical ventricular reconstruction (P < 0.001 for all comparisons).

Follow-Up

The median follow-up for all surviving patients was 48 months (minimum, 30). Only four patients withdrew consent, and six patients were lost to follow-up before the last visit. Of the 1000 patients, 990 (99%) underwent complete follow-up that began at randomization and concluded between August 1 and December 31, 2008.

Left Ventricular Volume

A core-laboratory quantitative assessment of the end-systolic volume index on echocardiography was performed at baseline and at 4 months in a total of 373 patients (212 patients who were assigned to undergo CABG alone and 161 who were assigned to undergo CABG with surgical ventricular reconstruction). The mean end-systolic volume index in patients assigned to undergo CABG alone decreased by an average of 5 ml per square meter, from 82 to 77 ml per square meter (a reduction of 6%). For patients who were assigned to undergo CABG with surgical ventricular reconstruction, the average decrease was 16 ml per square meter, from 83 to 67 ml per square meter (a reduction of 19%) (Fig. 1 in the Supplementary Appendix). The difference between the two groups in the change from baseline was significant (P < 0.001).

Symptoms

Among patients in both study groups, the proportion with no angina increased and the proportion with CCS class III or IV angina decreased during the interval from baseline to the last follow-up visit (Fig. 2A). The symptoms of patients in both study groups improved by an average of 1.7 classes (P=0.84 for the difference between the two groups in the change from baseline). Likewise, in the two groups, the proportion with New York Heart Association (NYHA) class I heart failure (no symptoms)
increased and the proportion with class III or IV heart failure decreased during the interval from baseline to the last follow-up visit (Fig. 2B). The symptoms in the two study groups improved by an average of one NYHA class (P = 0.70 for the difference between the two groups in the change from baseline).

6-Minute Walk Test

In the two study groups, approximately three quarters of patients performed the 6-minute walk test at baseline, and a similar proportion did so
at 4 months (Fig. 2 in the Supplementary Appendix). The median distance walked was 350 m at baseline and 350 m at 4 months for patients assigned to undergo CABG and 358 m at baseline and 410 m at 4 months for those assigned to undergo CABG with surgical ventricular reconstruction. The increase in the median distance walked was similar in the two groups (48 m among patients who were assigned to undergo CABG and 52 m among patients assigned to undergo CABG with surgical ventricular reconstruction, P = 0.80). Among patients assigned to undergo CABG who performed the 6-minute walk test and were assessed for symptoms, 34% were symptomatic during the baseline test and 9% were symptomatic at 4 months. The corresponding rates among patients assigned to undergo CABG with surgical ventricular reconstruction were 33% and 11%.

**Primary Outcome**

The primary outcome of death from any cause or hospitalization for cardiac causes occurred in 292 of 499 patients (59%) assigned to undergo CABG and in 289 of 501 patients (58%) assigned to undergo CABG with surgical ventricular reconstruction (hazard ratio for the combined approach, 0.99; 95% confidence interval [CI], 0.84 to 1.17; P = 0.90) (Table 2 and Fig. 3A). Fatal events of any cause occurred in 141 patients (28%) assigned to undergo CABG and in 138 patients (28%) assigned to undergo CABG with surgical ventricular reconstruction (hazard ratio, 1.00; 95% CI, 0.79 to 1.26; P = 0.98) (Table 2 and Fig. 3B). Hospitalization for cardiac causes occurred in 211 patients (42%) and in 204 patients (41%), respectively (P = 0.73). Hazard-ratio plots showed no interaction for the primary outcome between study-group assignment and baseline characteristics of interest (Fig. 4).

**Secondary Outcomes and Events**

Operative rates of death (i.e., deaths occurring within 30 days after the procedure) did not differ significantly between the two study groups, according to either the intention-to-treat analysis or the as-treated analysis (Table 2). The rates of seven secondary procedures, of acute myocardial infarction (P = 0.96), and of stroke (P = 0.35) were low and similar in the two groups.
Figure 3. Kaplan–Meier Estimates of Outcomes.
Panel A shows the probability of the primary outcome (death from any cause or hospitalization for cardiac causes), which did not differ significantly between the two groups. The primary outcome occurred in 292 patients (59%) assigned to undergo coronary-artery bypass grafting (CABG) alone and in 289 patients (58%) assigned to undergo CABG with surgical ventricular reconstruction (SVR) (hazard ratio, 0.99; 95% CI, 0.84 to 1.17).
Panel B shows the probability of death from any cause, which occurred in 141 patients (28%) assigned to undergo CABG and in 138 patients (28%) assigned to undergo CABG with SVR (hazard ratio, 1.00; 95% CI, 0.79 to 1.26).
**Discussion**

We compared the efficacy of CABG alone with that of CABG combined with surgical ventricular reconstruction in patients with coronary artery disease and left ventricular systolic dysfunction. As anticipated, the addition of surgical ventricular reconstruction resulted in a significantly greater
reduction in left ventricular volume than was achieved with CABG alone. However, this improvement in ventricular volume did not translate into a measurable benefit for the patients. Symptomatic improvement after surgery was similar in the two study groups. There was no significant between-group difference in the primary outcome of death or hospitalization for cardiac causes or in any other clinical outcome. Operative, intubation, and initial hospitalization times were longer in patients treated with the combined procedure. The findings of this study do not support the use of surgical ventricular reconstruction in the population studied.

Two reasons might be offered for the negative outcome of this trial. Perhaps experienced surgeons decided to enroll patients for whom they recognized that surgical ventricular reconstruction would prove unnecessary but offered this procedure directly, instead of enrollment in the trial, to all concurrently evaluated patients for whom they were confident the procedure would be beneficial. Making such precise decisions about patient selection is not consistent with the diverse opinions about the eligibility of specific patients for randomized assignment to surgical ventricular reconstruction, as discussed at STICH investigator meetings. Participating investigators appeared to make randomization decisions from a broad spectrum of positions of equipoise on the basis of differences in judgment in weighing baseline clinical and imaging characteristics of potential participants in the trial.

The more plausible reason for the lack of benefit seen with surgical ventricular reconstruction is that benefits anticipated from surgical reduction of left ventricular volume (reduced wall stress and improvement in systolic function) are counter-balanced by a reduction in diastolic distensibility. Previous work has shown that an important predictor of survival in patients with systolic dysfunction is the left ventricular ejection fraction during exercise. The most favorable dynamic response of the ventricle to the demand for increased cardiac output is associated with both an increase in end-diastolic volume and a decrease in endsystolic volume. Surgical ventricular reconstruction may impede this enhanced filling response.

One limitation of the design of the STICH trial was that physicians and surgeons caring for patients were aware of the treatment received. We sought to mitigate this limitation as much as possible by seeking complete follow-up for all patients and by selecting trial end points that were not
primarily subjective ones. It is conceivable that the decision to hospitalize a patient during the follow-up period could have been influenced by the physician’s knowledge of the specific operation performed. However, rates of death, myocardial infarction, and stroke were similar in the two study groups.

A total of 83 of 1000 patients (8%) did not receive the assigned treatment. Patients who were identified before randomization as eligible for either treatment could be expected to have a higher preoperative crossover rate than patients for whom one operative strategy was preferred. Moreover, physician-directed crossovers were similar in the two study groups. Analyses that were performed on the intention-to-treat principle and according to the surgery received had similar results.

In summary, our trial compared the efficacy of CABG alone with that of CABG with surgical ventricular reconstruction in patients with coronary artery disease and left ventricular systolic dysfunction. There was no significant difference between the two study groups in the degree of symptom improvement or in the rate of death or hospitalization for cardiac causes.

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References


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Surgical Ventricular Reconstruction

- Specific population of patients considered for SVR:
  - Dilated ventricle
  - Anterior wall dyskinesia
  - LVEF
  - Rationale
  - Relieves ischemia with CABG
  - Reduces LV volume with SVR

General Indications for SVR

- CAD
- EDVI >100mL/m²
- >35% area of asynergy (akinesia/dyskinesia)

STICH SVR Hypothesis and Design Overview

STICH SVR Hypothesis: Adding SVR to CABG in ischemic HF patients will decrease death/cardiac rehospitalization

1,000 HF pts (2002-2006)
CAD, EF <35%
Anterior wall scar amenable to SVR

499 CABG only
501 CABG + SVR

Mean Age 62 years
Female 16%
14%

Kaplan-Meier Estimates of Outcomes in STICH
Median Follow-up of 4 years

Death from Any Cause
Death from Any Cause

No. at risk
319
120
71
52
32
25
99
23
499
434
417
363
201
59

CABG
CABG + SVR

P=0.90
HR=0.99
CI=0.84 – 1.17

P=0.98
HR=1.00
CI=0.79 – 1.26

CABG
CABG + SVR

Results
Baseline LV Volumes

STICH SVR Hypothesis Group

ESVI or EDVI (mL/m²)

Median
66 mL/m²
95 mL/m²
79.6 mL/m²

Mean
86.1 mL/m²
117.5 mL/m²

LV ESVI (n= 917)

LV EDVI (n=846)

Median
111.5 mL/m²

Definition of LV Shape

- Shape determination reflects extent of asynergy and not type of asynergy
- Endofars of SVR related to extent of asynergy and probably not to type of asynergy
- Type III has greatest burden of MR
- Percutaneous valve is effective at controlling of anterior wall curvature and increased short axis diameter

- Source of Data
  - CMR 275 (28%)
  - Echo 332 (33%)
  - SPECT 240 (24%)
  - Site 153 (15%)

- Median LVEF

- Patients (%)

- LVEF (%)
Why SVR did not work in STICH?

- Adverse effects of SVR
- Patient Selection
- Conduct of Operation

Unanswered Questions

- Is there a subgroup of patients who benefit from SVR?
- Is LV volume reduction associated with any clinical benefit?
  - Rigorous analysis of LV geometry
  - Threshold reduction in LV volume?
  - Smaller volumes or greater change associated with benefit?
- LV geometry and outcome?
  - Akinesia vs dyskinesia?
  - Extent of asynergy?
  - Threshold for LV size +/- geometry predicts benefit?
Congestive heart failure is one of the world’s leading causes of morbidity and mortality. As our population ages and our medical care improves, the number of patients suffering from end-stage heart failure continues to rise. In the USA alone, there are nearly 6 million suffering with heart failure. Yet of the 500,000 new patients diagnosed each year, less than 3000 are offered transplantation due to limitations of age, co-morbid conditions, and donor availability. This deficiency has led to surgical alternatives, such as mitral reconstruction, to treat heart failure LV.

One of the most common and serious problems in cardiomyopathy is the development of mitral regurgitation (MR) which is associated with a significant reduction in long-term survival. The progressive dilatation of the left ventricle initially gives rise to MR that begets more MR and further ventricular dilatation. Mitral reparative surgery for end-stage heart failure may interrupt this cycle of ventricular deterioration through the restoration of normal cardiac physiology.

Effective management of mitral regurgitation begins with an understanding of the functional anatomy of the mitral valve. The mitral valve apparatus consists of the annulus, leaflets, chordae tendineae, papillary muscles and the entire left ventricle. Though the pathogenesis of MR in cardiomyopathy is multifactorial, it must be recognized that its etiology is based upon changes in the ventricle. With normal ventricular geometry, the redundant mitral leaflets are responsible for a zone of coaptation that is more than twice the area of the mitral valve orifice. As the failing ventricle dilates, the progressive expansion of the mitral annulus results in incomplete leaflet coaptation and a central regurgitant jet of functional mitral insufficiency. Therefore a significant determinant of leaflet coaptation in functional MR is the diameter of the mitral valve annulus.

Furthermore with cardiomyopathy, a combination of factors contributes to mitral regurgitation. Here, functional MR from annular and ventricular dilatation is compounded by pathologic changes in the subvalvular structures of the ventricle, including annular dilation, leaflet tenting and apical displacement, and posterior leaflet restriction.
Symptoms of congestive heart failure are often amplified when MR is present. Clinical signs of decreased cardiac output and pulmonary congestion such as dyspnea and reduced exercise tolerance increase in proportion to the progression of MR. Some of these patients develop myocardial irritability that may manifest in the form of ventricular arrhythmias or sudden death.

Physical examination of MR in cardiomyopathy typically reveals a hyperdynamic cardiac impulse and a characteristic blowing holosystolic murmur that radiates to the axilla, back, or neck. However, with severely depressed ventricular function these clinical findings may be inconspicuous. Radiographically, patients usually have an enlarged cardiac silhouette indicative of left ventricular or atrial enlargement. Typical EKG findings include left atrial enlargement and ventricular hypertrophy.

An initial transthoracic echocardiogram (TTE) is helpful to estimate the severity of MR and assess ventricular function. Measurements of left ventricular end-systolic dimension are less dependent on preload than ejection fraction and thus provide a more accurate assessment of ventricular performance. Prior to operation, a TEE must be obtained to evaluate the mitral valve and the coexistence of coronary artery disease should be ruled out by coronary angiography.

Once MR is documented, it is essential to define the mitral pathoanatomy by TEE clearly. Since the surgical approach for functional MR may be different, a detailed understanding of leaflet and chordal excursion including the regurgitant jet characteristics is helpful to plan the correct operation effectively. Though color Doppler may provide a semiquantitative analysis of MR, this method is often sensitive to load conditions, ventricular pressure, jet eccentricity and left atrial size and thus may lead to incorrect estimations of the true degree of MR. Proximal flow convergence analysis, which calculates the regurgitant volume by measuring the flow proximal to the mitral valve orifice, may be a preferable method to quantify the extent of MR in heart failure patients more accurately.

The successful management of mitral regurgitation in heart failure involves a combined medical and surgical approach. Pre-operatively, the patient is optimized with an aggressive regimen of diuretic and vasodilator therapy to minimize ventricular afterload and normalize the patient’s circulating volume. This also may include bi-ventricular pacing (CRT). Once patients are medically optimized, they are approached surgically based
on the etiology and anatomy of their MR. The majority of heart failure patients with MR will have a symmetric central jet from mitral annular dilatation. Annular geometry is restored by means of reduction annuloplasty using a circumferential rigid ring. By undersizing the annuloplasty to overcorrect the defect, one may effectively restore the optimal zone of coaptation and function of the mitral apparatus.

Numerous studies of mitral reconstruction (MVR) in CHF have noted acceptably low operative mortality (less than 5%). The ACORN series was a prospective, multi-institutional, multi-surgeon experience in 193 patients with MR and a mean EF of 19%. In that report, MVR was performed with a 1.6% 30 day mortality. However, while mitral repair in these high risk patients has been shown to be safe and result in improved quality of life, NYHA class, Minnesota living with heart failure score, less hospitalizations, and lower medical financial burden, long term mortality benefit remains unconvincing.

A retrospective analysis compared the effect of MVR repair with medical therapy in CHF patients with severe MR. In this non-randomized, but propensity-matched series, the results showed qualitative improvement, but little mortality benefit of MVR repair in advanced heart failure and severe LV dysfunction over the 10 year period of the trial. The only predictor of mortality was reverse remodeling. Unfortunately, in many early MVR-CHF studies, there was an early recurrence rate of MR of up to 40%, which certainly negatively influenced any survival benefit in these series. Therefore, MVR surgery for these patients remains controversial, as residual or recurrent MR and limited LV reverse remodeling certainly mitigate any mortality benefits.

Recent MVR trials in CHF, using a complete rigid ring and certain subvalvar influencing techniques have shown a much lower rate of MR recurrence. Cardiologists and surgeons have been disappointed with direct ventricular remodeling attempts in CHF, such as the Batistta operation, the STITCH trial of the SAVR operation or even complex devices such as the ACORN and the Myocor/Coapsys device. A randomized prospective re-evaluation of mitral repair in CHF, with a thorough understanding of the ventricular pathogenesis of this disease may be warranted.
11:20 a.m. – 11:40 a.m.  Ventricular Restraint Therapies: Beyond Acorn and HeartNet
Michael A. Acker, MD
University of Pennsylvania

Abstract not available at the time of printing.
11:40 a.m. – 12:00 p.m. Mechanical Circulatory Support in 2010: An Update
Nicholas G. Smedira, MD
Cleveland Clinic Foundation

OBJECTIVES
1. Review devices available for "long-term" mechanical circulatory support.
2. Review patient and device matching and risk assessment models.

Changing Landscape
With the January 2010 approval of the Heartmate II left ventricular assist system for destination therapy the era of implantable pulsatile or displacement LVAD therapy has ended. For both bridge to transplantation and destination therapy the HM II has shown superiority over the pulsatile Heartmate XVE with fewer driveline and pump infections, fewer neurologic events and greater durability and reliability. At least for the short term, continuous flow is well tolerated and has been shown to effectively support large patients, reduce elevated pulmonary pressures and improve end organ function in a manner that indistinguishable from the much larger and less durable pulsatile systems.

Device Selection
As compared to 10 years ago, the options for mechanical circulatory support are now fairly limited. The Novacor LVAS, Ventrassist System, Coraide, LionHeart and Abiocor Total Artificial Heart are not in clinical use. Going forward the HM II will be the device of choice for univentricular support. Other radial flow pumps include the Jarvix 2000 and Micromed (now the Heart Assist 5) pumps but they have yet to get full FDA approval. Interest in centrifugal pumps such as the Heartware and Terumo Duraheart systems are based on the lack of bearing wear, smaller size (Heartware), lower RPM, more reliable flow estimates and potential for a more physiologic Starling response to flow demands. Trials of these devices are in progress with little data to date to suggest one centrifugal system is superior to another or will be superior to the radial flow devices. The SynCardia Total Artificial Heart and Thoratec BIVADs are important tools in the mechanical circulatory support armamentarium. They are used primarily in patients with severe biventricular failure and can be quite helpful in patients with catastrophic large myocardial infarctions with post infarction VSDs or contained ruptures.
Patient Selection

The fact remains that the sicker the patient the greater the peri-operative and 1 year mortality. Estimates of peri-operative mortality have categorized patients by severity of illness (INTERMACS Score), by estimating a score using a modification of the Seattle Heart Failure Model or use of the MELD score, by determining use or non-use of biventricular support or by looking at the sequential number of devices needed to stabilize a patient. In a recently published paper by the experienced team at John Hopkin's, an INTERMACS score of greater than 2 or and Seattle score of more than 3.5 predicted a 50–60% 12 month mortality. The University of Michigan found that an elevated MELD score predicted mortality and blood transfusion requirement. Use of more than one form of mechanical support device, including temporary devices either before or after permanent LVAD significantly increased mortality as did delayed use of biventricular support.

Bibliography


12:00 p.m. ADJOURN
Surgeons have traditionally performed procedures to treat diseases by gaining direct access to the internal structures involved, and using direct visual inspection. Much effort has gone into identifying the most appropriate incisions and approaches to enable full access inside body cavities, specific organs or musculoskeletal structures. Imaging has traditionally been used primarily for pre-operative diagnosis and at times for surgical planning. Intra-operative imaging, when used, was meant to provide further diagnostic information or to assess adequacy of repair. In most cases X-ray static images or fluoroscopy has been used in the operating room. As the application of less invasive procedures has progressed, other imaging technology has been applied in an effort to address the limitations of simple X-ray or fluoroscopy. The most commonly used technique is optical imaging through a scope and camera system. This permits visualization of the surface of structures but has not been applicable for cardiac or vascular surgery due to the presence of blood.

Alternative imaging techniques however, do offer the capability of providing the surgeon with a “view” of the surface and the internal structures that optical imaging cannot provide. These include ultrasound, x-ray in the form of fluoroscopy or CT, and magnetic resonance imaging. Of these only fluoroscopy and ultrasound currently provide real-time imaging, which the surgeon needs for conducting a reconstructive procedure. Fluoroscopy has the limitation of using ionizing radiation and also projecting 3D information onto a 2D image plane thus making navigation of instruments to a target area more difficult. Ultrasound however, is not ionizing radiation and can provide 3D information regarding structures and the relationship of instruments to these structures. The main drawback of ultrasound is the spatial resolution and artifact from metallic instruments in the field of view.
Three-dimensional ultrasonographic (3DUS) imaging has made significant progress in the past few years, with improvements in spatial and temporal resolution. The ease of data acquisition, real-time 3D volume rendering, the ability to focus on a specific anatomic structure, and a variety of additional quantification tools have enabled virtually routine application of 3DUS in cardiology practice. Real-time 3D echocardiography (RT3DE) is an ideal imaging modality for septal defect closure because it provides precise anatomic images of complex anatomic structures, such as the atrial or ventricular septum and margins of septal defects. Obstacles to application of 3DUS, such as acoustic interference from metallic instruments caused by shadowing and side-lobe artifacts in the field of view, can be minimized with instrument modifications, such as the application of surface coating and by varying the angle of instrument navigation with respect to the ultrasonographic probe. The high frame rates currently achieved with RT3DE obviate the need for electrocardiographic or respiratory gating for image acquisition.

**Specialized Instruments**

As for any endoscopic procedure, surgical instrument must be designed to navigate within the confined spaces of the operative field, but one additional factor that is important in image guided surgery is the ability to detect the location of the surgical tool. Furthermore, for image guided interventions, the instrument must be made of material that is compatible with the imaging modality. To navigate complex trajectories, instrument flexibility and steerability are very desirable features. Dexterity, or the ability to smoothly manipulate tool position and orientation with as many degrees of freedom as possible, is also an essential feature for most endoscopic procedures.

**Surgical Procedures**

We have demonstrated that epicardial RT3DE provides adequate anatomic detail for surgical task performance in an in vitro model and in vivo model of beating-heart patch ASD closure. We have also shown that a similar technique can be used for closing muscular VSDs in the beating heart. In the VSD closure procedure, trans-apical insertion of the patch and patch fixation anchors was done reliably and reproducibly. (Figure)

Current work has focused on beating heart repair of mitral valve regurgitation. To achieve a comprehensive repair, techniques to address leaflet
prolapse as well as annular dilatation must be developed. We have developed methods for plication of the prolapsing segment of the posterior leaflet in vivo and are working on annuloplasty techniques as well.

References


10:15 a.m. – 10:20 a.m. DISCUSSION
10:20 a.m. – 10:35 a.m.  Mechanical Assist for Single Ventricle Failure
Mark D. Rodefeld, MD
Indiana University

Single ventricle failure is a highly complex problem and is associated with poor outcomes. Mechanical circulatory support (MCS) options for single ventricle failure are limited and suboptimal. Existing devices fail to address physiologic issues which are unique to the Fontan circulation, principally elevated systemic venous pressure, relative pulmonary arterial hypotension, and suboptimal ventricular filling—issues which form the basis of the so-called Fontan paradox.1

Shortcomings of existing devices: The majority of MCS support for failing SV circulations (either shunted single ventricle or Fontan) consists of ECMO or VAD support. Although anecdotal successes have been reported with VAD support, use of these devices occurs principally on a salvage basis.2 Currently available MCS devices are designed principally for biventricular systemic support—many as scaled down versions of adult MCS devices. Although ECMO/VAD support applied to failing SV circulations may provide systemic support, it does not address other co-existing physiologic problems.

Issues unique to single ventricle mechanical support: Single ventricular failure is complex and multi-factorial. The etiology may be more related to the right-heart issues associated with the palliative state, rather than primary or intrinsic myocardial failure. In other words, the target for Fontan support may be the cavopulmonary circulation rather than (or in addition to) the systemic circulation. These physiologic issues may include hypoxemia, ventricular volume over/underload, and impaired diastolic coronary perfusion. To advance the field, we are addressing the inability of existing devices to address this physiologic:mechanical mismatch with new approaches.3,4

Powering the Fontan: We have taken a radically different investigative approach to single ventricle palliation and support. By adding a modest power source to the cavopulmonary junction of the univentricular Fontan circulation, the single ventricle Fontan circulation can be restored to one which resembles more stable 2-ventricle physiology. This may solve serious problems unique to single ventricle palliation. A means to provide this type support in the complex anatomic and physiologic circum-
stances of a univentricular Fontan circulation is dissimilar to any other mechanical circulatory support application. No such pump currently exists.

In this presentation, the technologic considerations for development of a mechanical device to function in this role will be discussed. Our technologic approach, and that of other groups, will be outlined.

References


10:40 a.m. – 10:55 a.m. Automated Remote Ischemic Preconditioning in Pediatric Patients
Andrew N. Redington
The Hospital for Sick Children

Syllabus material available as a separate handout.

10:55 a.m. – 11:00 a.m. DISCUSSION
11:00 a.m. – 11:15 a.m. Robotic Surgery for Congenital Heart Disease
Johannes Bonatti, MD
University of Maryland

Rationale
Robotic technology allows difficult surgical maneuvers in narrow spaces and enables completely endoscopic suturing. At present robotic techniques are applied for totally endoscopic coronary artery bypass grafting, for minimally invasive mitral valve repair, for totally endoscopic arrhythmia surgery, for endoscopic removal of cardiac tumors, and for minimally invasive surgical treatment of congenital heart disease. For the latter robotic technology provides the additional advantage that surgeon movements can be downscaled and that microsurgery is feasible. Robotic technology also holds a potential for fetal heart surgery.

History
The first congenital malformations that were treated using robotic technology were septum secundum ASD and patent foramen ovale. Lucia Torraca of Milan published the first series of six patients in 2001 [Torraca 2001]. She applied the first generation of daVinci systems (Intuitive Surgical, Sunnyvalve CA)

General remarks
At the present stage of development only non-complex congenital disease can be taken into consideration. One major challenge is remote access heart lung machine perfusion and endoscopic induction of cardioplegia. Some repairs are still in an early experimental stage in animal models.

Procedures
1. Robotic totally endoscopic repair of patent foramen ovale and septum secundum defect
Repair of these lesions is usually straightforward in the open sternotomy setting. They were therefore the first congenital lesions which were tackled using robotic technology. The initial series were carried out in adults.

The procedure is performed through minithoracotomy or in a port-only, totally endoscopic approach [Torraca 2001, Wimmer Greinecker 2003, Argenziano 2003, Bonaros 2006] The patient is cannulated in the groin.
With the right lung collapsed ports are placed on the right chest. The pericardium is opened anterior to the phrenic nerve and both venae cavae are encircled with umbilical tapes. Ascending aortic balloon endoocclusion or transthoracic aortic clamping are used for induction of cardioplegia. The right atrium is opened and the defect is closed by either direct suture or a patch. Suturing maneuvers are very well feasible using the robotic system. Most groups use 4/0 Gore-Tex. After robotic closure of the right atrium the patient is weaned from cardiopulmonary bypass and decannulated. A single chest tube is used and the portholes and/or the minithoracotomy are closed.

All reports on robotic ASD repair state early return to normal activities. According to these publications procedure safety seems to be adequate.

As remote access perfusion and endoaortic balloon occlusion become difficult in patients < 50 kg alternative methods have to be found. One option is to perform the procedure in hypothermic ventricular fibrillation without placement of an aortic crossclamp. Baird and coworkers described such a case in 2006 [Baird 2006].

2. Totally endoscopic removal of atrial septal occlusion devices

The Innsbruck team performed the world’s first completely endoscopic robotic removal of a tilted Amplatz ASD occlusion device. After removal the defect was closed with a synthetic patch [Bonatti 2008].

3. Robotic closure of patent ductus arteriosus and division of vascular ring

Le Bret published a series of 56 patients in 2002. 26 of them underwent ductus closure by conventional videoscopic technique, 26 were operated on using the Zeus robotic system. One patient in the robotic group was converted to a conventional approach. Operative times were longer in the robotic group [Le Bret 2002]. The Zeus system due to inferior dexterity as compared to the daVinci system is not produced anymore. Suematsu reported on 9 patients who underwent robotic endoscopic PDA repair and 6 patients in whom a vascular ring was divided with the daVinci system [Suematsu 2005]. One patient was converted due to adhesions. The operative result was perfect in all others.
4. **Totally endoscopic closure of ventricular septal defect**

Amin and Coworkers performed robotic completely endoscopic VSD closures in Yucatan pigs [Amin 2006]. The right ventricular wall was approached in completely endoscopic fashion, a delivery sheath was introduced, and an appropriately sized Amplatzer™ device was deployed into the VSD. Five of seven deployments were successful. Follow up in four pigs showed mild to moderate left to right ventricular shunt in one.

5. **Totally endoscopic coarctation repair**

To our knowledge such a procedure has not been carried out clinically but reports on endoscopic suturing of the aorta are available. Smith and coworkers investigated the feasibility of aortic suturing in a sheep model using the daVinci™ robotic system [Smith 2005]. The robotic instruments were introduced through intercostal ports and the descending thoracic aorta was crossclamped using transthoracic clamps. A segment of the thoracic aorta was excised and replaced. Both polypropylene sutures and nitinol clips were used, the average anastomotic time was 37 min. This study demonstrates basic feasibility of robotic endoscopic aortic suturing. A major challenge concerning clinical applicability is the insertion of ports in the presence of large intercostal collaterals. Stadler et al have recently published a report on 100 cases of robotic endoscopic abdominal aortic repair [Stadler 2008]. This study demonstrates clinical feasibility of completely endoscopic suturing of grafts to the aorta. Given these reports attempts to perform robotic coarctation repair can be expected.

**General Results**

The majority of clinical reports on robotic congenital heart surgery state relatively long operative times. Learning curves are also described which take at least 20 to 30 cases. Early patient discharge and early return to normal activities are clear benefits of robotic procedures.

**References**


11:15 a.m. – 11:20 a.m. DISCUSSION
11:20 a.m. – 11:35 a.m.  Real-Time MRI-Guided Procedures
Keith A. Horvath, MD
National Heart, Lung and Blood Institute

MRI Guided Cardiac Surgery
Keith A. Horvath
Cardiothoracic Surgery Research Unit
National Heart, Lung & Blood Institute

Why MRI?
- Minimizes, low morbidity
- Provides anatomic images
- Provides unique information in surgical navigation
- Provides real-time information of:
  - surgical orientation
  - surgical function
  - position of the graft vessels
  - position of the myocardium
  - precise positioning of devices

Imaging and Access

MRI Guided OR Suite

Automatic Image Registration

MR Guided OR Floor Plan
11:35 a.m. – 11:40 a.m.  DISCUSSION
The correction of many congenital defects necessitates the use of vascular grafts, including surgical reconstruction of the right ventricle to pulmonary artery continuity or the aorta. Children who undergo these types of operations with either prosthetic or homograft materials frequently require multiple reinterventions related to conduit failure. All of the clinically available vascular replacements are nonliving, foreign materials with limited long-term function. They lack the potential for growth and remodeling and are associated with an increased risk of thromboembolism and infection. The goal of tissue engineering is to create new conduits from biodegradable or decellularized biomaterials seeded with autologous cells. The essential characteristics of such materials were described by Dwight E. Harken and included durability, absence of thrombogenicity, resistance to infections, lack of immunogenicity, and the potential for growth. Tissue-engineered constructs consisting of a biodegradable scaffold seeded with autologous cells provides early structural integrity and induces rapid and extensive ingrowth of autologous cells, including prompt endothelialization. As the scaffold degrades, a vascular neotissue forms and a living, biocompatible conduit is created. In addition, these constructs have the ability to grow and remodel as the child becomes an adult. Progress in the development of tissue-engineered conduits has been slow but progressive.

Our initial studies employed a gelatin mesh seeded with fetal cardiomyocytes (JTCVS 2000;119:368). The patch beat and was able to engraft in the heart. However, the allogeneic cells were eventually rejected despite the use of cyclosporine. In addition, the gelatin mesh lacked sufficient tissue strength to withstand the pressure of the left ventricle. Therefore we investigated alternative biodegradable biomaterials.

Dr. John Mayer and his group at the Boston Children’s Hospital seeded a biodegradable mesh with multiple cell types and implanted the construct as a conduit from the right ventricle to the pulmonary artery in young lambs (JTCVS 1998;115:536). The constructs maintained patency, but extensively remodeled demonstrating the importance of cellular infiltration.

Dr. Toshiharu Shin’oka, a cardiac surgeon who worked with Dr. Mayer in Boston initiated a clinical trial after his return to the Tokyo Women’s
Hospital. He employed a new composite biodegradable mesh developed by Dr. Ikada, a chemical engineer from Kyoto, consisting of a loose inner mesh which encouraged cell engraftment and dissolved within 6 months and a stronger outer layer which dissolved after 2 years. Bone marrow stem cells were seeded into the construct which was then employed during the repair of congenital heart defects in children. The first case was reported in 2001 (NEJM 2001;344:532). The graft maintained its patency after the biomaterial dissolved and appeared to grow as the girl matured. A midterm (JTCVS 2005;129:1330) and a late (2010;139:431) report suggested that the tissue engineered conduits were functioning well.

We demonstrated that this construct encouraged the ingrowth of autologous cells and engrafted in the right ventricular outflow (JTCVS 2002;124:1157). However, seeding the construct with autologous cells prior to implantation markedly facilitated the engraftment of the scaffold and prevented thinning and dilatation of the region repaired. These initial preclinical and clinical investigations will require more extensive documentation before this approach becomes a viable alternative for surgical repair of congenital defects.

Other studies have documented the growth potential of cell-seeded biodegradable conduits (Circ. 2006;114[suppl I]:I-159). However, the addition of cytokines or their genes which enhance cell engraftment offers to improve the healing of these tissue engineered grafts. Unfortunately, the development of a tissue engineered valved conduit has not yet been successful. The recent availability of induced pluripotent stem cells suggests that a beating conduit might become available in the future. These encouraging preliminary results suggest that cell-seeded constructs may avoid the complications of inert materials. However, the ideal conduit would have a functioning valve and would beat in synchrony with the recipient heart.

In summary, tissue-engineered patches and conduits have been developed and evaluated in a small number of patients. The preliminary results are encouraging. However, a tissue-engineered valved and beating conduit will be required before we can build a new heart.

11:55 a.m. – 12:00 p.m. DISCUSSION

12:00 p.m. ADJOURN
Treatment for high grade dysplasia or early (T1a) esophageal cancer aimed at esophageal preservation is utilized successfully by many centers. These approaches include endoscopic mucosal ablation, and endoscopic mucosal resection (EMR). These techniques are becoming increasingly adopted in clinical practice, but require a diligent approach to a labor intensive treatment regimen and frequent surveillance performed throughout follow-up. Indications and limited evidence supporting the use of these strategies is reviewed.

Pathologist Interpretation of High-Grade Dysplasia

It is critical that the pathologic interpretation of the esophageal lesion be confirmed. Histologic criteria for dysplasia were described in 1988 by Reid and Haggitt et al.\textsuperscript{1} Despite these criteria being accepted nearly 20 years ago, significant inter-observer variability still exists among pathologists experienced in gastrointestinal dysplasia.\textsuperscript{2} Ormsby et al. found that among experienced gastrointestinal pathologists, inter-observer agreement for distinguishing HGD from invasive cancer was only fair at $k = 0.56$. Furthermore, agreement did not substantially improve following establishment
of uniform criteria. Among the existing studies regarding HGD, almost all had two experienced pathologists confirm the histology.

**Mucosal Ablation of High-Grade Dysplasia**

Several methods of mucosal ablation have been reported for HGD. Of these, PDT provides an example of feasibility, although most centers are currently using RFA in place of PDT.

In 2005, a multicenter randomized controlled trial comparing PDT plus omeprazole versus omeprazole alone for treatment of Barrett’s esophagus with HGD was published. This study included 208 patients; 138 patients received PDT with omeprazole (20 mg twice a day), and 70 received omeprazole (20 mg twice a day) alone. Surveillance endoscopies were performed every 3 months, until four consecutive quarterly biopsies were negative for HGD, then every 6 months thereafter. In short follow-up (24.2 months PDT and 18.6 months in the omeprazole cohort) HGD was eliminated in 77% of the patients receiving PDT plus omeprazole and in 39% receiving omeprazole alone (p < 0.0001). Invasive cancer developed in 13% of the PDT patients compared to 28% treated with omeprazole alone (p = 0.006). This study confirmed the feasibility of PDT for ablation of HGD, but several concerns exist. The development of cancer in a significant number of patients treated with PDT argues against the use of this modality in patients eligible for esophagectomy. Additionally, pseudo-regression after PDT risks occult progression of invasive cancer, potentially to an incurable stage.

**Radiofrequency Ablation for HGD**

Radiofrequency ablation (RFA) using the HALO System (BARRX Medical Inc., Sunnyvale, Ca) uses a balloon-based electrical array to deliver ablative energy to the circumference of the esophagus. Depth of penetration of the energy is controlled via a feedback loop through the catheter. This technique of endoscopic mucosal ablation is relatively new, however, results in the short term appear promising. RFA can be used as a solitary modality for flat HGD or can be combined with mucosal resection for patients with nodular HGD. There has been a recent randomized multi-center trial which enrolled 127 patients with dysplastic Barrett’s to treatment with either RFA of sham ablation. The authors report that “among patients with high-grade dysplasia, complete eradication occurred in 81.0% of those in the ablation group, as compared with 19.0% of those in
the control group (P < 0.001). Overall, 77.4% of patients in the ablation group had complete eradication of intestinal metaplasia, as compared with 2.3% of those in the control group (P < 0.001). Patients in the ablation group had less disease progression (3.6% vs. 16.3%, P = 0.03) and fewer cancers (1.2% vs. 9.3%, P = 0.045).8

Endoscopic Mucosal Resection

EMR can be utilized either as a diagnostic technique or therapeutic modality to treat targetable areas of HGD or early intramucosal carcinoma (IMC), as well as to remove entire segments of metaplastic mucosa. From a diagnostic standpoint, nodular areas of Barrett’s esophagus can undergo EMR to determine if IMC exists, or if known IMC, accurately determining the depth of the lesion may alter surgical decisions. Circumferential EMR is generally utilized in shorter segments of metaplasia. Originally described in Japan, esophageal EMR was modified from experience with colon and gastric lesions. In the esophagus, it was used for excision of flat or polypoid esophageal mucosal tumors, particularly squamous cell carcinoma.9,10

Prior to EMR for carcinoma, endoscopic ultrasonography (EUS) can be performed to explore the depth of invasion and potential submucosal spread. Deeply invading tumors that are clearly penetrating into the muscularis propria are not amenable to removal by this technique. For tumors that appear to be confined to the submucosa (T1b) on EUS, a diagnostic EMR will confirm the depth of invasion, given the inaccuracy of EUS in determining submucosal involvement. A major advantage of EMR compared to mucosal ablative techniques is the availability of tissue samples for histologic assessment including margins, both lateral and deep. Currently, EMR as a therapeutic modality for cancer is appropriate only for neoplasms limited to the mucosa, where the incidence of lymph node metastasis has been shown to be minimal.11 As the incidence of nodal metastasis increases significantly with tumor penetration into the submucosa, esophagectomy with lymphadenectomy should be considered in reasonable performance patients.

EMR for treatment of Barrett’s esophagus with HGD has been described in multiple observational studies.11,12,14 EMR is targeted to identifiable areas of HGD followed by RFA ablation of any remaining metaplastic mucosa.

A single-center prospective study by Ell, et al evaluated 100 patients with early adenocarcinoma of the esophagus.12 Lesions included in the study
were polyoid or flat (not ulcerated) mucosal nodules, less than 20 mm in diameter, well or moderately-differentiated adenocarcinoma limited to the mucosa and without evidence of lymphvascular invasion (LVI). All patients received EMR and 49 of the patients underwent subsequent mucosal ablation. Complete local remission was achieved in 99 of the 100 patients in the initial study period. Significant complications were absent. During follow-up averaging 36.7 months, recurrent or metachronous carcinomas were detected in 11% of patients. Repeat EMR, feasible in all cases, resulted in long-term freedom of disease in 100% of patients. Overall 5-year survival was 98% owing to two deaths related to other causes.

In follow up, the same unit reported on an expanded group of 349 patients who underwent a variety of endoscopic ablative therapies including 279 who had EMR. HGD was seen in 61 patients. At a mean follow-up of 63.6 months, complete response was seen in 96.6%. Surgical resection was only required in 3.7% and 5-year survival was 84%.

Thus, the success of EMR depends upon the ability of adjunctive mucosal ablation to eliminate residual foci of metaplastic or neoplastic tissue, or upon prompt endoscopic recognition of recurrent disease. While preliminary results of circumferential EMR for resection of Barrett's esophagus associated with HGD or intramucosal carcinoma have been reported in a small number of patients with short-term follow-up, the published experience is far too limited at present to derive appropriate conclusions regarding efficacy, safety and applicability of the technique.

**Surveillance**

As mentioned, endoscopic therapy for HGD and IMC is a labor intensive project that requires diligent follow up. Rigorous adherence to process must be maintained to assure the quality of outcomes. Once the initial episode of treatment is performed to clear visible disease, routine surveillance with biopsies must be performed in order to correct/arrest any progression that may arise after treatment. All studies reporting success with endoscopic ablation therapies have employed surveillance protocols. Biopsy taken every 1–2 centimeters in four quadrants within the Barrett's segment is considered standard. We perform endoscopy quarterly with advanced endoscopic imaging technologies such as narrowband imaging (NBI), or confocal laser endomicroscopy in attempts to improve detection of dysplasia. Another approach is the use of vital stains, such as methylene blue, acetic acid or indigo carmine, which can
help direct and reduce the number of biopsies required to detect HGD within a segment of Barrett’s. We routinely use NBI and find it invaluable in detecting residual tiny islands of metaplasia that would otherwise go undetected.

References


10:15 a.m. – 10:30 a.m.  Esophagectomy
Kemp H. Kernstine, MD, PhD
City of Hope National Medical Center

Esophagectomy has been performed for over 100 years, nearly all for cancer. Other indications in include benign diseases may require esophagectomy as well; including Barrett’s with high-grade dysplasia, achalasia, diffuse esophageal spasm, rare esophageal motility disorders, intractable reflux, scleroderma and Chaga’s disease where all means of functional recovery have been exhausted. In spite of the current technologies, the results of esophageal resection and replacement, at best, result in some degree of abnormal swallowing which may so severe to result in chronic reflux with possible regurgitation of food and recurrent aspiration.

Esophageal cancer is the most common indication for esophagectomy. The incidence of esophageal cancer in the United States is rising at a faster rate than any other solid tumor.1-4 Nationally, the estimated totals are nearing 16,470 new cases for 20095 and, worldwide, it is the 5–6th leading cause of cancer death.6 Of the newly diagnosed patients, approximately 90% will die within 2–5 years.7-9 At clinical presentation, 30 to 40% have loco-regional disease where surgery is most effective.10,11 There is no uniformly effective screening program.12-15 Symptoms are the most common means to select patients for treatment. Unfortunately, by the time symptoms develop patients are frequently late in the course of their disease. The vast network of intramural and periesophageal lymphatics increases the likelihood for loco-regional and systemic metastases. Surgery alone is not sufficient to affect a cure in most patients.

The current methods of staging esophageal cancer include a high-resolution chest computed tomogram (CT) from the ear lobes to the umbilicus, an FDG-positrion emission tomogram (PET) and/or CT/PET and an esophagogscopy and endoscopic ultrasound (EUS). Each of these tests has complementary information and is equipment/observer dependent. Details of their indications and accuracy are reviewed elsewhere.16-19

Thorough evaluation of esophageal cancer patients should identify patients most likely to benefit from surgery alone and those to be treated with induction therapies and those unlikely to benefit from surgery. Although those with deeply invading tumors and/or involved periesophageal lymph nodes have a greater likelihood for recurrence and cancer-related death, there still appears to be a surgical survival advantage if an
R0 resection can be performed.\textsuperscript{20-22} CT and PET and as necessary history/physical exam-directed tests, such as brain magnetic resonance imaging and/or skeletal series, are used to assess for stage IV disease or disease in which patients are more likely to recur early after surgery. The EUS is the most sensitive test for depth of invasion and periesophageal nodal involvement.\textsuperscript{23-26} Ideally, careful staging should identify those potential esophagectomy patients who are likely to benefit from induction therapy, those patients with clinically-unidentified microscopic systemic disease.

Surgical technique varies significantly among surgeons and even within different techniques, the body cavities involved, radial resection margin, extent of lymphadenectomy and esophagectomy, and the method of reconstruction. The variability makes it very difficult to study effectiveness and the complications associated with surgical resection. Esophageal cancer surgeons should apply multiple techniques to provide the optimal care for the co-morbidities and tumor biology present focusing on the goals, primarily postoperative survival and a high quality-of-life. There is no single technique that meets the needs in all situations.

For cancer, esophagectomy may be seen by referring physicians and health care systems as too risky and expensive. The mortality in prospective trials is approximately 5–13\%\textsuperscript{27-29} and in other published retrospective series is approximately 3–4\%.\textsuperscript{30-32} Morbidity is 40 to 80\%, the higher rates being reported in prospective trials.\textsuperscript{27} With the addition of chemotherapy or chemoradiotherapy, the mortality increases by 1.7\% and 3.4\%, respectively.\textsuperscript{33}

Although, esophagectomy is performed in a variety of ways, the goals are the same:

1. Accurately assess biology, staging
2. Complete removal of all malignancy achieving negative margins (R0)
3. Resect all involved nodal tissue and sufficient nodal tissue to stage
4. Provide a functional reconstruction

From the results of trials like RTOG 8501,\textsuperscript{34} German Induction Trial\textsuperscript{28} and FFCD 9102,\textsuperscript{29} some are referring to esophagectomy as a "big biopsy," providing staging information rather than a significant impact on survival. Esophagectomy must have a low complication rate and nearly normal
long-term functional status. To accomplish this, there are 4 approaches to resection and reconstruction of which the esophageal surgeon should be familiar. The procedure should be chosen according to the patient's physiology and comorbidities, and the tumor biology.

The transhiatal esophagectomy involves the resection of the esophagus and some nodal tissue through 2 incisions, a laparotomy and cervical incision. Patients who are best served by this technique have early cancer, Barrett's with high-grade dysplasia, achalasia, motility dysfunction or a severely damaged or malfunctioned esophagus. Fewer nodes are resected with this method. In spite of this, transhiatal esophagectomy for cancer does not appear to have a poorer survival compared with other techniques, although some reports are demonstrating long-term advantage of the transthoracic methods.

The next method is the near-total esophagectomy with an in-chest anastomosis, when performed on the right chest, referred to as the Ivor Lewis. Nodal resection is performed through a laparotomy and thoracotomy. To achieve a R0, the radial margins may include other mediastinal structures. The anastomosis is usually at or above the level of the azygous vein. Leak rates are low, approximately 5%, but when they occur can be catastrophic resulting in increased morbidity, length-of-stay, cost-of-care and surgical mortality. The whole stomach or a gastric tube is most commonly used as the conduit, but the colon and small bowel may be used. The Ivor Lewis allows for more than twice the number of nodes than for the transhiatal, 9 versus 18.

The more extensive esophageal and nodal resections can be performed by the “three-hole” or “modified” McKeown technique. A right thoracotomy is usually performed first where the radial resection and varying degrees of lymphadenectomy are performed. It affords extensive dissection and can allow for removal of all 3 major esophageal nodal stations: cervical, thoracic and upper abdominal. Some perform an en bloc resection of all periesophageal tissue including a 1-inch cuff of the diaphragm, both pleura, the adjacent pericardium, the thoracic duct and the azygous vein along with the deep and superficial cervical nodes to include a thorough dissection around the recurrent laryngeal nerves. The anastomosis is performed in the neck, most commonly on the left side to avoid damage to the right recurrent laryngeal nerve. The esophageal anastomotic leak rate is reported to be as high as 10–15% with an increasing procedure-related morbidity. There may be some long-term survival
advantage in some cases, especially with the *en bloc* resections,\textsuperscript{44,45} but the patients must be able to tolerate this more extensive procedure.

The final technique is the left thoracotomy approach.\textsuperscript{46,47} This is ideal for gastroesophageal junction tumors especially those with significant bulk and/or potential invasion of the peri-GE junction structures. Access to the abdominal contents can be performed by a trans-diaphragmatic incision or a thoracoabdominal extension of the thoracotomy. It provides excellent access for resection of tumors from the lower two thirds of the esophagus, gastroesophageal junction and gastric cardia. The esophago-gastric anastomosis can be performed either in the chest or the left neck.

Reconstruction to achieve GI continuity can be challenging. The stomach is the preferred conduit, the blood supply is based on the right gastro-epiploic artery. The gastric tissue is thick, durable and more resilient than the small or large bowel and it has an extensive submucosal blood supply. The postoperative functional status is good. Whole stomach and wide gastric tubes appear to have less length and often require a Kocher maneuver to allow the reach necessary for an anastomosis. Narrow gastric tubes, less than 3–4 cm, provide extra length to the conduit, but the wider tube, 4–5 cm or more in width, appears to have a lower anastomotic leak rate.\textsuperscript{48} Gastric tubes have the disadvantage of creating a long staple line that may leak. Preservation of the right gastric and/or small vascular tributaries in the gastrosplenic ligament may provide extra blood supply to the anastomosis. Preoperative chemotherapy and radiation also appear to increase the leak rate as well; especially in those cases when the remnant native esophagus is within the radiation field. Wrapping the anastomosis with pedicled omentum may reduce the leak rate.\textsuperscript{49,50} “Supercharging” or anastomosing an internal mammary or adjacent neck vessel to a threatened gastric or colonic conduit may provide extra blood supply.\textsuperscript{51,52} Also reported is ischemic preconditioning, the early ligation of the left gastric artery several days to weeks prior to performing an esophagectomy.\textsuperscript{53,54} Proponents claim significant benefit.\textsuperscript{55}

In the literature, the extent of resection may be determined by the tumor biology. To account for submucosal and “skip” lesions a 10 cm proximal and 5–6 cm distal with a microscopically negative radial margin are considered sufficient.\textsuperscript{56,57} Nodes may be involved a significant distance away from the primary tumor and their resection may provide survival advantage. Squamous cell, especially in Asian populations, can have extensive nodal spread where lymphadenectomy appears relevant to long term
survival. In adenocarcinoma nodal metastases may be more predictable, patients with lower esophageal and nearly GE junction tumors, may metastasize to the mid, rarely the upper thoracic and cervical nodes; but the gastroesophageal junction and gastric cardia patients less frequently do. Tailoring the resection to this information remains controversial. Many feel that resecting and counting 12–25 nodes in the specimen is sufficient to adequately stage and possibly provide a survival advantage.58-60

There are multiple ways to manage the pylorus in patients that have a gastric conduit for their reconstruction. With the removal of both vagus nerves the effect to gastric motility and the pylorus may result in gastric outlet obstruction. Pyloroplasty or pyloromyotomy are performed to promote emptying and reduce the likelihood of postoperative gastric stasis. For whole stomach conduits, the pyloroplasty appears to improve gastric emptying and reduce gastric outlet obstruction.61,62 There was no difference in the clinically relevant gastric outlet obstruction when comparing pyloromyotomy to no pyloric procedure, the rate being 10–15%.63 Botulinum toxin injection of the pylorus has been used in both a prospective clinical and multicenter trial with a 5% or less rate of gastric outlet problems. There appears to be a lower incidence of bile reflux in the injected group.64

For patients with very superficial malignant or premalignant lesions where deep invasion or nodal involvement is unlikely or with nonmalignant disease, a nerve sparing esophagectomy may provide better short and long-term function.65 By preserving the pyloro-antral innervation it may help to minimize dumping syndrome, improve gastric emptying and reduce bile reflux potentially improving quality-of-life. Too there is the potential of maintaining immunological and cardiovascular functions provided by vagal innervation. To date, data supports this approach in this select patient population.66

The method of anastomosis has been addressed in the literature. A water and airtight anastomosis can be achieved by either a hand-sewn or stapled technique. Anastomotic leak rates appear to be less with the stapled technique.67 A higher stenosis rate occurs with a two layer closure whether performed in the chest or the neck.68 Risk factors for stenosis include history of cardiac disease, anastomotic leak, poor blood supply, cervical compared with chest anastomosis, bleeding during surgery, circular staple size smaller than 33 mm.69 Both a linear side-to-side,
functional end-to-end and circular anastomoses have been performed with similar results. The introduction of a trans-oral circular stapling anvil has been utilized with varying success and may enable the minimally invasive chest anastomosis.\textsuperscript{70}

Minimally invasive technology was introduced in the 1990s and the proponents claim similar short and long-term opportunities while reducing the encumbrances of the open techniques potentially providing esophagectomy to "high risk" patients. The largest series reports a 30-day mortality of less than 2% with a median length of stay of 7 days and anastomotic leak rate of approximately 12%.\textsuperscript{71} The same group has led a phase II trial in 106 patients from 16 institutions that demonstrated similar results.\textsuperscript{72}

Although the computer-assisted technology has been introduced, there is yet to be an advantage demonstrated.\textsuperscript{73} Operating times are long and there appears to be similar results to the other minimally invasive techniques. Investigation is ongoing.

Surgeon training and experience and hospital volume appear to be important to perform esophagectomy, but the manner and case volume remain to be determined.\textsuperscript{74-77} The use of induction therapies including targeted agents as directed by the patient's own pathology and determining the response to therapy as well as different radiation therapy techniques, such as intensity modified radiation therapy with the ultimate goal being to achieve a complete response, are subjects of investigation.\textsuperscript{78-82} The use of the PET scan and circulating biomarkers and tumor cells have the potential of determining the response to therapy and detecting early recurrence.\textsuperscript{83-85} Between 10–45% of induction patients will have a complete pathological response and for those that do they will have a 50–70% 5-year survival.\textsuperscript{80,86} Biologically selected long term adjuvant therapies may provide long-term opportunities to further reduce the likelihood for recurrence.\textsuperscript{87,88} A new staging system is soon to be released through a worldwide collaboration.\textsuperscript{89}

Currently, esophagectomy has an important role in the treatment of benign and malignant esophageal pathologies and relies on the skill of the surgeon, surgical team and the institution to select, surgically-treat and manage esophageal disease patients. In the wrong situation, the procedure can have a high mortality and may be highly morbid and debilitating, incapable of providing long-term survival. Healthcare systems may
choose alternative therapies to treat patients with end-stage esophageal pathologies unless we can provide a safe, functional and cost effective treatment resulting in a superior quality-of-life to non-surgically treated patients.

References


10:30 a.m. – 10:40 a.m. DISCUSSION
Management of Clinical Stage I NSCLC: SBRT/RFA

Malcolm M. DeCamp, Jr., MD
Chief, Division Thoracic Surgery
Northwestern Memorial Hospital
Professor of Surgery
Feinberg School of Medicine at Northwestern University
Chicago, IL

Standard Therapies for Stage I NSCLC
• Lobar Resection – “gold standard”
• Open / VATS approaches
• Sublobar resections
• Segmental/wedge resections
• Currently viewed as a compromise operation for high-risk patients
• External Beam Radiation

Therapeutic Options
• Sleeve/Bronchoplastic procedures
• Segmentectomy
• Wedge resection +/- Brachytherapy
• Radio-frequency Ablation (RFA)
• Stereotactic Radiosurgery (SRS)

Comparison of Rx Modalities for Stage I NSCLC

<table>
<thead>
<tr>
<th>Modality</th>
<th>5-year survival</th>
<th>Local Recurrence (most by 2yrs)</th>
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</thead>
<tbody>
<tr>
<td>Lobar resection</td>
<td>60-70%</td>
<td>6-4%</td>
</tr>
<tr>
<td>Sub-lobar resection</td>
<td>50-60%</td>
<td>17.2%</td>
</tr>
<tr>
<td>External Beam Radiation</td>
<td>5-20%</td>
<td>15%</td>
</tr>
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</table>
Can we reduce the higher local recurrence that occurs with sublobar resection using radiation?

RFA

Temperature and time effects on tissue

- 60°C – irreversible cellular damage (4-6 minutes)
- 90°C – instantaneous cell death
- 46°C – irreversible cellular damage at 60 minutes
- Thermo injury starts at 42-45°C – hypothermia

RFA system

- 3 components
  - An RFA generator
  - An active electrode
  - Dispersive electrodes (bovie pads)
- RF energy (alternating current) moves from the active-dispersive-active electrodes, resulting in frictional heating of tissue

RFA Technical Considerations

Intra-op Images

- RITA XL
- Boston Scientific
- Valleylab
RFA for the treatment of NSCLC
- 18 patients with 21 NSCLC tumors
  - 10 Men & 8 women
  - Median age 75 (range 58 to 86) years
  - Cancer stages
    - I (n=9)
    - II (n=2)
    - III (n=3)
    - IV (n=4) 3 recurrent cancer / 1 synchronous liver mets

Fernando et al; JTCVS 2005 129(3):639-44

Overall Survival After RFA

RFA : Patients Characteristics
- N=19 Stage I NSCLC patients
  - All medically inoperable
  - Stage IA 13 patients
  - Stage IB 6 patients
  - Tumor size: Mean 2.6 cm (Range 1.6 - 3.8)
  - Median Age – 78 yrs
  - No perioperative mortality

Pennathur et al; JTCVS 134; 2007

Stage I NSCLC RFA: Survival
- Median follow-up 23 (range 6 – 51) months
- 13/19 (68.4%) patients alive
- Estimated probability of 1 year survival – 95%
- Local progression 42%
- Median time to progression 27 months
- Progression Free 50%

Pennathur et al; JTCVS 134; 2007

Conventional Radiotherapy
- Standard approach 45 – 66 Gy total dose in 1.8 to 2 Gy fractions
- 5 year survival rates 10 – 30%
- Treatment duration 5-6 weeks

Stereotactic Radiosurgery (SRS)

Stereotactic Body Radiotherapy (SBRT)

Dose Escalation
- Benefit to dose escalation
  - dose – response related to
  - local control and survival
- Dose escalation limited by toxicity
  - radiation pneumonitis
3-D Conformal RT
- More sophisticated planning techniques that limit treatment volumes and radiation to normal lung tissue to allow safer dose escalation
- Still have significant local failure

Lung Stereotactic Radiotherapy
- Systems to improve targeting and decrease lung target motion
- Allow dramatic reduction of treatment volumes facilitating hypofractionation
  - increased daily doses
  - increased biologic equivalent dose
  - reduced overall treatment time

Stereotactic high-dose RT for stage I NSCLC (Japanese multi-institutional study)
- 300 pts
  - T1N0: 193 pts; T2N0: 107 pts
  - median age 75 yrs
  - 100 medically inoperable; 110 refused surgery
  - median tumor size 28 mm (7-58 mm)
  - 18-75 Gy at isocenter in 1-22 fx
  - BED (\(\gamma_3\)/\(\gamma_5\)) median 108 Gy (57-180 Gy)
  - Median F/U for survivors 38 mos (2-128 mos)
  - Lung complications NCI grade 3: 3.0%
  - CT response:
    - CR: 26%
    - PR: 59%
  - 5 yr local control:
    - BED > 100 Gy: 86%
    - BED > 100 Gy: 87% (p=0.01)
  - 5 yr OS:
    - operable pts: 65% (74% if BED > 100 Gy; 37% if BED < 100 Gy)
    - inoperable pts: 37%
  - Recommend 48 Gy / 4 fx (BED = 105.6 Gy)

Once-weekly SBRT for NSCLC
- Oakwood Hospital, Dearborn, MI (n=60)
  - Local control:
    - IA: 100% (45/45)
    - IB: 93% (14/15)
  - 5-yr CSS:
    - IA: 74%
    - IB: 64%
  - 5-yr OS:
    - IA: 48%
    - IB: 42%
  - Toxicity:
    - grade 2: 3%
    - grade 3-4: 0%

Onishi H. ASCO 2004, abstr #7007; ASCO 2006, abstr #7045
Salazar O. IJROBP, (Epub) May 1, 2008

Pt: FA  Pre CK
Pt: FA  Pre CK
396

90TH ANNUAL MEETING MAY 1–MAY 5, 2010
TORONTO, ON, CANADA

SRS Tumor response
- Georgetown University, Washington, DC
  - 26 patients with 45–60 Gy in 2 fractions
  - 6% rate limited subacute radiation pneumonitis
  - No local progression in short term F/U
- St Joseph's Hospital, St. Paul, MN
  - 61 patients mean dose 39 Gy (range 28-40) with mean 3 fractions
  - 4 patients with self limited radiation pneumonitis
  - 96% local control with 7 mo F/U
  - CR 28%, PR 50%, stable 18%, progression 6%

Conclusions
- Stage I NSCLC appears better controlled with hypofractionated radiation to a higher biologic dose and appears well tolerated
- Multicenter prospective trial of 20 Gy x 3 fractions to assess tumor response, long-term complications
- Assess survival

Conclusions
- Stereotactic lung radiosurgery can afford superior local tumor control to conventional radiation therapy
- Effectiveness is limited in large tumors and central tumors
- We await results of long term survival data
10:55 a.m. – 11:10 a.m.  Thoracoscopic Lobectomy and Segmentectomy

Thomas A. D’Amico, MD
Duke University

Strategy for Thoracoscopic Lobectomy

After bronchoscopy and mediastinoscopy (when indicated), single-lung anesthesia is established using a dual lumen endotracheal tube or bronchial blocker. The patient is positioned in full lateral decubitus position with slight flexion of the table at the level of the hip, which provides splaying of the ribs to improve thoracoscopic access and exposure. Port placement is a matter of surgeon preference. Most surgeons use 3 or 4 incisions, although lobectomy can usually be accomplished using only 2 incisions. The first incision, a 10 mm port access used predominantly for the thoracoscope, is placed in the 7th or 8th intercostal space in the midaxillary line. The second incision, an anterior access incision (4.5–6.0 cm) for dissection and specimen retrieval, is placed in the 5th or 6th intercostal space.

Instrumentation for thoracoscopic lobectomy is critical to successful completion of the procedure. The thoracoscope should be a 30-degree angled scope, to optimize the ability to achieve panoramic visualization during dissection and to minimize competition with the operative instruments. A spectrum of surgical instruments may be employed for dissection, including conventional instruments and dedicated thoracoscopic or laparoscopic instruments. It is especially beneficial to use curved instruments for retraction during dissection, as it will minimize the tendency for instruments to compete or collide with each other. Thoracoscopic (linear) mechanical staplers are employed for control of the vessels (2.0 or 2.5 mm staples), bronchus (3.5 or 4.8 mm staples) and fissure.

Individual vessel dissection is not performed through the fissure; rather, dissection is performed beginning with the anterior hilum, and continuing posteriorly. For any anatomic thoracoscopic lobectomy, hilar dissection is begun with mobilization of the pulmonary vein. For upper lobectomy, the lung is reflected posteriorly and inferiorly to facilitate dissection. For lower lobectomy, the lung is retracted superiorly. Moving the thoracoscope to the anterior incision may improve visualization of the superior hilum and may facilitate placement of the linear stapler for upper lobectomy, if introduced through the midaxillary port.
Results

The safety and efficacy of thoracoscopic lobectomy for patients with early-stage lung cancer has been established. Although there are no prospective, randomized series that compare thoracoscopic lobectomy to conventional approaches, a sufficient number of series have been published, both single-institution and multi-institution experiences, to conclude that thoracoscopic lobectomy is a reasonable strategy for patients with clinical stage I lung cancer.

The Cancer and Leukemia Group B (CALGB) reported on the results of a multi-institutional series of 97 patients who underwent thoracoscopic lobectomy. In this series, the mortality was 2%, the operative time was 130 minutes, and the median length of stay was 3 days. Daniels and colleagues reported the results of thoracoscopic lobectomy in 170 consecutive patients. The 30-day mortality was 2%, with no intraoperative deaths. The conversion rate was 1.8%, and none were emergent. The median chest tube duration was 3 days and median length of stay was 3 days. Follow-up review of 500 patients in that series demonstrates a mortality of 1% and chest tube duration of 2 days. In that series, atrial fibrillation occurred in only 10% of patients post-operatively.

Demmy and colleagues reported on their results in a series of patients, who underwent either thoracoscopic lobectomy or conventional thoracotomy. In this series, the percentage of patients reporting severe pain was 6% in those patients after thoracoscopic lobectomy and 65% after thoracotomy. Moreover, the percentage of patients reporting minimal or no pain was 63% in those patients after thoracoscopic lobectomy and 6% after thoracotomy. Other studies analyzing acute pain have concluded that VATS either causes less pain or lower analgesia requirement in the early postoperative period.

In addition, several studies have recently demonstrated that the incidence of postoperative complications is lower after thoracoscopic lobectomy, as compared to thoracotomy. In one study, 283 patients who underwent open lobectomy were compared with 300 patients who underwent thoracoscopic lobectomy. Using a propensity matched analysis, thoracoscopic lobectomy was associated with fewer overall complications, including atrial fibrillation. In a second study, using a case-matched strategy, 122 patients undergoing thoracoscopic surgery and 122 patients undergoing thoracotomy were compared. Overall, complications were
AMERICAN ASSOCIATION FOR THORACIC SURGERY

lower in the thoracoscopic group (17.2% vs 27.9%, P = .046). In another study, focusing on elderly patients (age ≥70 years), a retrospective, matched case-control study was performed evaluating the perioperative outcomes after lobectomy by thoracoscopy and thoracotomy. After matching based on age, gender, presence of co-morbid conditions, and preoperative clinical stage, there were 82 patients in each group. Thoracoscopic lobectomy resulted in a significantly lower rate of complications compared with thoracotomy (28% vs 45%, p = 0.04). No patients undergoing thoracoscopic lobectomy had higher than grade 2 complications, whereas 7% of complications in the open lobectomy group were grade 3 or higher. There were no perioperative deaths in the thoracoscopic lobectomy patients compared with an in-hospital mortality rate of 3.6% for thoracotomy patients.

Using a prospective database, the outcomes of patients who underwent lobectomy at Duke from 1999–2009 were analyzed with respect to postoperative complications. Propensity-matched groups were analyzed, based on preoperative variables and stage. Of the 1079 patients in the study, 697 underwent thoracoscopic lobectomy and 382 underwent lobectomy by thoracotomy. In the overall analysis, thoracoscopic lobectomy was associated with a lower incidence of atrial fibrillation (p = 0.01), atelectasis (p = 0.0001), prolonged air leak (p = 0.0004), transfusion (p = 0.0001), pneumonia (p = 0.0001), sepsis (p = 0.008), renal failure (p = 0.003), and death (p = 0.003). In the propensity-matched analysis based on preoperative variables, comparing 284 patients in each group, 196 patients (69%) who underwent thoracoscopic lobectomy had no complications, versus 144 patients (51%) who underwent thoracotomy (p = 0.0001). In addition, thoracoscopic lobectomy was associated with a lower incidence of atrial fibrillation (13% vs 21%; p = 0.01), less atelectasis (5% vs 12%; p = 0.006), fewer prolonged air leaks (13% vs 19%; p = 0.05), fewer transfusions (4% vs 13%; p = 0.002), less pneumonia (5% vs 10%; p = 0.05), less renal failure (1.4% vs 5%; p = 0.02), shorter chest tube duration (median 3 vs 4 days; p < 0.0001) and shorter length of hospital stay (median 4 vs 5 days; p < 0.0001).

Similar results were obtained when the STS database was analyzed. All patients undergoing lobectomy as the primary procedure via thoracoscopy or thoracotomy were identified in the STS database from 2002–2007. After exclusions, 6323 patients were identified: 5042 thoracotomy, 1281 thoracoscopy. A propensity analysis was performed, incorporating
preoperative variables, and the incidence of postoperative complications was compared. Matching based on propensity scores produced 1281 patients in each group for analysis of postoperative outcomes. After VATS lobectomy, 945 patients (73.8%) had no complications, compared to 847 patients (65.3%) who had lobectomy via thoracotomy (p < 0.0001). Compared to open lobectomy, VATS lobectomy was associated with a lower incidence of arrhythmias \( n = 93 (7.3\%) \) vs. \( 147 (11.5\%) \); \( p = 0.0004 \), reintubation \( n = 18 (1.4\%) \) vs. \( 40 (3.1\%) \); \( p = 0.0046 \), and blood transfusion \( n = 31 (2.4\%) \) vs. \( n = 60 (4.7\%) \); \( p = 0.0028 \), as well as a shorter length of stay \( 4.0 \) vs. \( 6.0 \) days; \( p < 0.0001 \) and chest tube duration \( 3.0 \) vs. \( 4.0 \) days; \( p < 0.0001 \). There was no difference in operative mortality between the two groups.

Finally, an analysis of high risk patients was performed, defined as age >70 years. During the study period, 338 patients older than 70 years (mean age 75.7 ± 0.2) underwent lobectomy (219 thoracoscopy, 119 thoracotomy). Operative mortality was 3.8% (13 patients) and morbidity was 47% (159 patients). Patients with at least one complication had increased length-of-stay \( 8.3 ± 0.6 \) vs. \( 3.8 ± 0.1 \) days, \( p < 0.0001 \) and mortality \( 6.9\% (11 \) of 159) vs. \( 1.1\% (2 \) of 179), \( p = 0.008 \). Significant predictors of morbidity by multivariable analysis included age (odds ratio 1.09, \( p = 0.01 \)) and thoracotomy as surgical approach (odds ratio 2.21, \( p = 0.004 \)). Thoracotomy remained a significant predictor of morbidity when the propensity to undergo thoracoscopy was considered (odds ratio 4.9, \( p = 0.002 \)).

Thoracoscopic lobectomy has recently been demonstrated to be effective in selected patients after induction therapy. Finally, the use of thoracoscopic lobectomy may improve compliance with adjuvant chemotherapy, allowing a greater fraction of patients to undergo the combination of surgery and adjuvant therapy.

**Summary**

Minimally invasive approaches to lung cancer treatment have been demonstrated to be safe and effective for patients with early-stage lung cancer. Thoracoscopic lobectomy is designed to achieve the same oncologic result as conventional lobectomy: complete hilar dissection and individual vessel control. The recognized advantages of thoracoscopic anatomic resection include less short-term postoperative pain, shorter hospital stay, faster return to full activity, and preserved pulmonary function. In
addition, thoracoscopic lobectomy may preserve immunologic response and may improve compliance with adjuvant therapy. Although there are no prospective randomized studies comparing the thoracoscopic approach to conventional thoracotomy, there is no data from published series to suggest any difference in oncologic efficacy.

References


Thoracoscopic Lobectomy

AATS General Thoracic Symposium

Thomas A. D’Amico MD
Professor of Surgery, Duke University Medical Center
Program Director and Section Chief, Thoracic Surgery
Co-Director, Thoracic Oncology Research Laboratory
Director, Clinical Thoracic, Duke Comprehensive Cancer Center

Thoracoscopic Lobectomy

Oncologic Outcome

- There are no studies that report or suggest a difference in ability to achieve complete resection in patients with Stage I or II NSCLC
- There is evidence that there is equivalence in selected patients with Stage IIIA after induction therapy

Thoracoscopic Lobectomy: Advantages

1. Less postoperative pain
2. Shorter chest tube duration and length of stay
3. Faster return to full activity
4. Preservation of pulmonary function
5. Lower inflammatory cytokine response; preserved immunologic status

Thoracoscopic Lobectomy is Associated with Fewer Postoperative Complications

Western Thoracic Surgical Association
June 2008
- Compared outcomes after lobectomy (n=1079)
- Thoracoscopic (n=997) vs Thoracotomy (n=782)
- Propensity analysis (n=284 each) matching preoperative variables and stage
- Analysis of postoperative complications

Duke Series: Survival

Thoracoscopic Lobectomy

Duke Approach

Lobectomy in STS Database
Thoracoscopic Lobectomy is Associated with Lower Morbidity than Open Lobectomy: A Propensity-Matched Analysis from the STS Database

Subroto Paul MD, Jeffrey L. Port MD, Shubin Sheng PhD, Paul C. Lee MD, David H. Harpole MD, Mark W. Onaitis MD, Brendon M. Stiles MD, Nasser K. Altorki MD, Thomas A. D’Amico MD

Propensity Matching: Greedy 5 to 1 Algorithm

Demographics: Sex Status Comorbidity PFTS Other

Age Gender HTN CAD CHF WBC CRP
Diabetes ASA PVD DM DM
Smoking Steroid Use Preop Chemo Preop XRT
CVD CRI Elective Status

Thoracotomy N=1281 Thoracoscopic N=1281

Outcomes: Propensity Matched

<table>
<thead>
<tr>
<th>Feature</th>
<th>THOR (n=284)</th>
<th>VATS (n=284)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Complication, n (%)</td>
<td>144 (51%)</td>
<td>196 (69%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Atrial Fibrillation, n (%)</td>
<td>61 (21%)</td>
<td>37 (13%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Atelectasis, n (%)</td>
<td>34 (12%)</td>
<td>15 (5%)</td>
<td>0.006</td>
</tr>
<tr>
<td>Prolonged air leak, n (%)</td>
<td>55 (19%)</td>
<td>37 (13%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Pneumonia, n (%)</td>
<td>27 (10%)</td>
<td>14 (5%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Transfusion, n (%)</td>
<td>36 (13%)</td>
<td>11 (4%)</td>
<td>0.02</td>
</tr>
<tr>
<td>Renal Failure, n (%)</td>
<td>15 (5%)</td>
<td>4 (1.4%)</td>
<td>0.02</td>
</tr>
<tr>
<td>Death, n (%)</td>
<td>15 (5%)</td>
<td>8 (3%)</td>
<td>0.20</td>
</tr>
</tbody>
</table>

Chest tube duration (days): THOR 4 VATS 3 P <0.0001

Length of stay (days): THOR 5 VATS 4 P <0.0001

Duke and STS Databases: Conclusions

Thoracoscopic lobectomy → Improved outcomes:
- More patients with no complications
- Fewer overall complications
- Lower rates of atrial arrhythmias and cardiac events
- Lower rates of pneumonia and pulmonary events
- Lower blood transfusion rate
- Shorter chest tube duration and LOS

Thoracoscopic Lobectomy in High Risk Patients

- Under 70 Poor PFT
  - Low Risk n=386 (42%)
  - High Risk n=267 (22%)

- Over 70 Poor PFT
  - Low Risk n=135 (14%)
  - High Risk n=203 (22%)
Thoracoscopic Lobectomy in High Risk Patients

Under 70 Poor PFT
n=207 (22%)

Over 70 Poor PFT
n=135 (14%)

Over 70 Good PFT
n=203 (22%)

Thoracoscopic Lobectomy is Associated with Fewer Postoperative Complications in Patients >70 years


General Thoracic Surgical Club 2009

- Compared outcomes after lobectomy (>70 years)
- Risk Model and Propensity analysis, matching preoperative variables and stage
- Analysis of overall postoperative complications

Logistic Regression Model of Risk Factors for Morbidity

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Patients (n=338)</th>
<th>Propensity Anal (n=125)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical Approach</td>
<td>Thoracotomy</td>
<td>VATS</td>
</tr>
<tr>
<td></td>
<td>2.71</td>
<td>5.99</td>
</tr>
<tr>
<td></td>
<td>(1.57-4.70)</td>
<td>(2.06-17.4)</td>
</tr>
<tr>
<td></td>
<td>0.004</td>
<td>0.001</td>
</tr>
<tr>
<td>Age</td>
<td>1.09</td>
<td>1.25</td>
</tr>
<tr>
<td></td>
<td>(1.02-1.17)</td>
<td>(1.09-1.44)</td>
</tr>
<tr>
<td></td>
<td>0.01</td>
<td>0.002</td>
</tr>
<tr>
<td>FEV1 (% predicted)</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td></td>
<td>(0.97-1.00)</td>
<td>(0.96-1.01)</td>
</tr>
<tr>
<td></td>
<td>0.07</td>
<td>0.34</td>
</tr>
<tr>
<td>Smoking History</td>
<td>1.71</td>
<td>5.72</td>
</tr>
<tr>
<td></td>
<td>(0.79-3.98)</td>
<td>(0.97-33.9)</td>
</tr>
<tr>
<td></td>
<td>0.17</td>
<td>0.055</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.74</td>
<td>2.45</td>
</tr>
<tr>
<td></td>
<td>(1.26-3.39)</td>
<td>(0.69-8.75)</td>
</tr>
</tbody>
</table>

Complications after Thoracoscopic Lobectomy Propensity Analysis (>70 yrs)

Thoracotomy VATS p

- No Cx 36% 41% 0.0001
- A-fib 28% 18% 0.04
- Respiratory Cx 12% 5% 0.03
- Transfusion 16% 7% 0.01
- Delirium 13% 5% 0.003

Pulmonary Function Tests Predict Complications after Open but not Thoracoscopic Lobectomy

Southern Thoracic Surgical Association November 2009

- Compared outcomes after lobectomy in patients with FEV1 or DLCO <60% predicted
- Propensity analysis, matching preoperative variables and stage
- Analysis of overall postoperative complications

A Cost Utility Analysis of Lobectomy: Thoracoscopic Versus Thoracotomy

European Society of Thoracic Surgeons, June 2009

- Thoracoscopic lobectomy was $2,000 less expensive than lobectomy by thoracotomy
- Costs were less for TL at every phase of care
- This represents a potential cost savings of $100 million per year in the United States alone
Thoracoscopic Lobectomy: Adjuvant Chemotherapy

- Chemotherapy improves survival after resection for patients with stage II and III NSCLC
- The ability to deliver adjuvant chemotherapy may be limited by post-operative issues
- Does thoracoscopic lobectomy improve the delivery of adjuvant therapy, compared to thoracotomy?

Thoracoscopic Lobectomy Facilitates The Delivery Of Adjuvant Chemotherapy After Resection For NSCLC


- 100 consecutive patients undergoing lobectomy and adjuvant chemotherapy (1999-2004)
  - Thoracoscopic: 57
  - Thoracotomy: 43

**Efficacy of Chemotherapy Delivery**

<table>
<thead>
<tr>
<th></th>
<th>Thoracoscopic N=57</th>
<th>Thoracotomy N=43</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any delayed chemo doses</td>
<td>18%</td>
<td>58%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Any reduced chemo doses</td>
<td>26%</td>
<td>49%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>75% of planned regimen</td>
<td>61%</td>
<td>40%</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Improving Outcomes: Thoracoscopic Lobectomy

The role of thoracoscopic lobectomy continues to evolve, with increasing relative effectiveness

- Superior QOL outcomes
- Superior technical outcomes: fewer complications
- Higher relative advantages in high-risk patients
- Superior oncologic outcomes: adjuvant therapy
- Lower cost

11:10 a.m. – 11:20 a.m. DISCUSSION
SESSION III:
MEDIASTINAL STAGING

Moderator: G. Alec Patterson, MD, Washington University School of Medicine

11:20 a.m. – 11:35 a.m. Role of EBUS

Kazuhiro Yasufuku, MD, PhD
Toronto General Hospital

Role of EBUS in Mediastinal Staging

- Non-invasive staging (imaging)
  - CT, PET, PET/CT, MRI
- Invasive staging (Tissue)
  - Surgical biopsy (EBUS, VATS)
  - Needle biopsy (EBUS-TBNA, EUS-FNA, TBA)

Convex Probe EBUS (CP-EBUS)
EBUS-TBNA vs Mediastinoscopy

- EBUS-TBNA is a less invasive, less painful, and less expensive procedure with a shorter recovery time.

- EBUS-TBNA is recommended as the first line procedure for hilar lymph node staging.

Advantages of EBUS over Mediastinoscopy

- EBUS is less invasive and painful.
- EBUS is less expensive.
- EBUS has a lower risk of complications.

Conclusion

EBUS-TBNA is a more effective and less invasive procedure with a shorter recovery time.

EBUS-TBNA should be considered the first line procedure for hilar lymph node staging.

In cases with surgically incurable lung cancer, there remains a role for mediastinoscopy to exclude metastasis in non-invasive cases.
Accurate surgical thoracic lymph node staging is recommended for lung cancer patients to direct therapy, demonstrate treatment response, and predict survival. One barrier to the use of mediastinoscopy or other traditional staging methods is PET imaging that has a negative predictive value that is acceptable to many clinicians. An adequate PET reduces patient incentive to accept the relatively low risk of complications or inconvenience of surgical staging. However, many cases remain understaged and some patients are overstaged by PET if surgical biopsy is not used as a confirmatory test.

Lymph nodes can be sampled for staging or removed in their entirety for potential therapeutic benefits. Less invasive methods are emerging for both LN sampling and lymphadenectomy. Ultrasound-guided FNA of lymph nodes by either the transbronchial or transesophageal routes offer better accuracy than “blind” FNA and, if positive, can avoid more costly or invasive staging operations. Furthermore, these procedures are useful if mediastinoscopy is impractical, dangerous (e.g. Tracheostomy), has been performed already, or is planned for later (e.g. after downstaging by neoadjuvant therapy). The route of these natural passages limits access to certain stations (See Table 1); however, the sizes of the targets and variations in airway/esophageal anatomy can make other areas accessible. In addition, combining these methods may increase the overall negative predictive value. Failure to recover malignant cells or other plausible diagnostic material from suspicious nodes leads to the need for larger biopsies afforded by procedures like mediastinoscopy. FNA guided by CT or transcutaneous ultrasound can also sample mediastinal or cervical nodes to provide staging information but generally this is in the context of bulky lymphatic disease with evidence of advanced disease elsewhere.

Mediastinoscopy instruments are enhanced by the addition of a video optics and expandible blades that provide better exposure to remove larger amounts of lymphatic tissue. With this enhanced scope, Video-assisted Mediastinal Lymphadenectomy (VAMLA) is performed with a similar incision as mediastinoscopy and Transcervical Extended Mediastinal Lymphadenectomy (TEMLA) is performed through a larger “collar” incision similar to that used for thyroidectomy. In addition, TEMLA (established and popularized by Zielinski) uses a sturdy sternal retractor to elevate
### Table 1. Less Invasive Mediastinal Staging Options.

<table>
<thead>
<tr>
<th>Methodology</th>
<th>Accessible Stations</th>
<th>High Yield Stations</th>
<th>Sensitivity/ NPV %</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Traditional</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervical Mediastinoscopy</td>
<td>1,2,3,4,7,10</td>
<td>2,4,7</td>
<td>86/94.5</td>
<td>– Rapid access to multiple stations</td>
<td>Incidence of RLN injury or vascular injury (low)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>– Large experience</td>
<td></td>
</tr>
<tr>
<td>Mediastinotomy (Chamberlain)</td>
<td>5,6</td>
<td>5,6</td>
<td>55/96</td>
<td>AP window access</td>
<td>Chest wall pain extending hospital stay</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Limited exposure, lymph tissue not always recovered</td>
</tr>
<tr>
<td>VATS/Robotic</td>
<td>2,3,4,5,6,7,8,9,10,11</td>
<td>4,5,6,7,8,9,10</td>
<td>100/100</td>
<td>Lymph node access approaching thoracotomy</td>
<td>Chest wall pain</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Chest drainage needed</td>
</tr>
<tr>
<td><strong>Newer</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VAMLA</td>
<td>2,4,7,8</td>
<td>2,4,7</td>
<td>100/98</td>
<td>Better lymph node clearance</td>
<td>Incidence of RLN injury or vascular injury (low)</td>
</tr>
<tr>
<td>TEMLA</td>
<td>1,2,3a,3p,4,5,6,7,8</td>
<td>2,3a,4,5,7,8</td>
<td>94/97</td>
<td>Highest lymph node clearance and level 5,6 access</td>
<td>Incidence of RLN injury or great vessel injury (low)</td>
</tr>
<tr>
<td>EUS</td>
<td>4L,5,7,8,9</td>
<td>7,8,9</td>
<td>69/88</td>
<td>No incision</td>
<td>Limited biopsy size</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Technology expensive</td>
</tr>
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</table>
the manubrium to increase exposure so that much of the case can be performed by direct viewing. The popularity of these "extended" approaches is growing and recurrence of disease in mediastinal lymph nodes has been unusual event for patients who have had successful, negative initial TEMLA. Furthermore, surgical restaging with TEMLA or VAMLA may offer an advantage in negative predictive value over endoscopic techniques. Some key points are listed in Table 1 as well. Another potential benefit to the use of more aggressive transcervical lymph node dissections is the reduction of dissection needed during subsequent thoracoscopic lobectomy operations (if performed before adhesions form).

Thoracoscopy is an excellent alternative or supplement to other surgical staging methods. It may have a higher rate of successful recovery of lymphatic tissue from the level 5/6 stations than mediastinotomy and it is being used as an adjunct to endoscopic dissections like VAMLA.

Finally, quite a few preclinical experiments have been performed using Natural Orifice Transendoscopic Surgery (NOTES) with acceptable acute and chronic results in animals. While still experimental, this methodology may provide future options to access critical nodes in patients for whom the above methods are unsuccessful or potentially more hazardous.
In summary, all the current methodologies for mediastinal lymph node staging should be considered complementary and used in combination where needed to achieve assessment of all nodes at risk. While proficiency at any one technique can reduce the need for the others, none can be eliminated entirely as clinical options.

References


11:50 a.m. – 12:00 p.m. DISCUSSION

12:00 p.m. ADJOURN
2009-2010
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<td>Philadelphia, PA</td>
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<td></td>
<td>Vaughn A. Starnes, MD</td>
<td>Los Angeles, CA</td>
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Donna McRitchie, MD Toronto, ON, Canada

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Federico Venuta  
Rome, Italy

Jakob Vinten-Johansen  
Atlanta, GA

Richard D. Weisel  
Toronto, ON, Canada

Seminars in Thoracic and Cardiovascular Surgery
Editor  
(2009)
Timothy J. Gardner  
Wilmington, DE

Co-Editor  
(2010)
David H. Adams  
New York, NY
Michael A. Maddaus  
Minneapolis, MN

Thoracic Surgery News
Editor  
Yolonda L. Colson  
Boston, MA

Associate Editor  
John G. Byrne  
Nashville, TN
Michael J. Liptay  
Chicago, IL
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Baltimore, MD
William G. Williams  
Toronto, ON, Canada

Operative Techniques in Thoracic and Cardiovascular Surgery
Editor  
Fred A. Crawford  
Charleston, SC

Pediatric Cardiac Surgery Annual
Editor  
(2009)
Richard A. Jonas  
Washington, DC

Editor  
(2010)
Thomas L. Spray  
Philadelphia, PA
# American Association for Thoracic Surgery Representatives 2009–2010

<table>
<thead>
<tr>
<th>Organization</th>
<th>Representative</th>
<th>Location</th>
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<tr>
<td>American Association of Blood Banks</td>
<td>Gus J. Vlahakes</td>
<td>Boston, MA</td>
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<td>American Board of Thoracic Surgery</td>
<td>David A. Fullerton</td>
<td>Denver, CO</td>
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<td>Bruce W. Lytle</td>
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<td>Curtis G. Tribble</td>
<td>Jackson, MS</td>
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<td>American College of Surgeons Advisory Council for Cardiothoracic Surgery</td>
<td>John G. Byrne</td>
<td>Nashville, TN</td>
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<td>Joe B. Putnam</td>
<td>Nashville, TN</td>
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<td>American College of Surgeons Board of Governors</td>
<td>W. Randolph Chitwood, Jr.</td>
<td>Greenville, NC</td>
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<td>Bryan F. Meyers</td>
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<td>American Medical Association House of Delegates</td>
<td>Michael J. Liptay</td>
<td>Chicago, IL</td>
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<td>Kirk R. Kanter</td>
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<td>Richard J. Shemin</td>
<td>Los Angeles, CA</td>
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<td>Association of Physician Assistants in Cardiovascular Surgery</td>
<td>Neal D. Kon</td>
<td>Winston-Salem, NC</td>
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<td>Cardiothoracic Surgery Industry Alliance</td>
<td>Irving L. Kron</td>
<td>Charlottesville, VA</td>
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<td>G. Alec Patterson</td>
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<td>David J. Sugarbaker</td>
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<td>Commission on Accreditation of Allied Health Education</td>
<td>Erle H. Austin, III</td>
<td>Louisville, KY</td>
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<td>CTSNET Board of Directors</td>
<td>David H. Adams</td>
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<td>Joint Council on Thoracic Surgery Education Board of Directors</td>
<td>Irving L. Kron</td>
<td>Charlottesville, VA</td>
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<td>Boston, MA</td>
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<td>Rochester, MN</td>
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<tr>
<td>National Association for Biomedical Research</td>
<td>Keith A. Horvath</td>
<td>Bethesda, MD</td>
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Perfusion Affairs (AMSECT, ABCPT, ACPE, CAHEA)
Harold L. Lazar Boston, MA
Joseph B. Zwischenberger Lexington, KY

Thoracic Surgery Foundation for Research and Education Board of Directors
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David J. Sugarbaker Boston, MA
# Past Meetings and Presidents of the American Association for Thoracic Surgery

<table>
<thead>
<tr>
<th>Year</th>
<th>Meeting Location</th>
<th>President</th>
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<tbody>
<tr>
<td>2008-2009</td>
<td>Boston, MA</td>
<td>Thomas L. Spray</td>
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<tr>
<td>2007-2008</td>
<td>San Diego, CA</td>
<td>D. Craig Miller</td>
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<td>2006-2007</td>
<td>Washington, DC</td>
<td>Bruce W. Lytle</td>
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<td>2004-2005</td>
<td>San Francisco, CA</td>
<td>Tirone E. David</td>
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<tr>
<td>2003-2004</td>
<td>Toronto, ONT</td>
<td>Joel D. Cooper</td>
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<td>2002-2003</td>
<td>Boston, MA</td>
<td>Fred A. Crawford, Jr.</td>
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<td>Washington, DC</td>
<td>Timothy J. Gardner</td>
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<td>James L. Cox</td>
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<td>Lawrence H. Cohn</td>
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<td>Floyd D. Loop</td>
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<td>David B. Skinner</td>
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<td>Robert B. Wallace</td>
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<td>Aldo R. Castaneda</td>
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<td>Chicago, IL</td>
<td>John L. Ochsner</td>
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<td>1984-1985</td>
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<td>Thomas B. Ferguson</td>
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<tr>
<td>1959-1960</td>
<td>Miami Beach, FL</td>
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<td>Emile Holman</td>
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<td>Eric J. Topol, MD</td>
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<td>Implications for Gene Therapy in Treating Coronary Artery Disease and Lung Cancer</td>
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<td>Timothy A. Springer, PhD</td>
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<td>Andrew S. Wechsler, MD</td>
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<td>Kurt Benirschke, MD</td>
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<td>Fritz H. Bach, MD</td>
<td>Transplant Immunology: A Broadening of the Concept for the Future</td>
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<td>Louis Siminovitch, MD</td>
<td>Advances in Cancer Research – Bench to Bedside</td>
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<td>Russell Ross, MD</td>
<td>The Pathogenesis of Atherosclerosis</td>
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<td>Raj K. Goyal, MD</td>
<td>Physiology and Pathophysiology of Esophageal Peristalsis</td>
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<td>1987</td>
<td>Gustav J.V. Nossal, MD</td>
<td>Immuno-Regulation: The Key to Transplantation and Autoimmunity</td>
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### HONORED GUEST LECTURERS

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<td>Too Big to Fail? Healthcare Reform in the U.S. and Canada</td>
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<td>Professor Michio Kaku</td>
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<td>50 Years of Cardiothoracic Surgery Through the Looking Glass and What the Future Holds</td>
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<td>A World in Need: Lessons Learned in Medical Diplomacy. The Project HOPE Perspective</td>
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<td>Marc R. de Leval, MD</td>
<td>Beyond Flatland</td>
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<td>Tolerance to Allogeneic and Xenogeneic Transplants</td>
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<td>Edmund D. Pellegrino, MD</td>
<td>Medical Ethics in the 21st Century: DNR or CPR?</td>
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<td>Rodolfo Herrera-Llerandi, MD</td>
<td>A Thoracic Tale of Two Cities</td>
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<td>The Structure and Function of Tissue Valves; Some Lessons Learned from the Fate of Implanted Heart Valves</td>
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<td>The Generality of Surgery</td>
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<td>Experience with Human Heart Transplantation</td>
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<td>Narrowing of the Aortic Isthmus and Enlargement of the Mind</td>
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<td>Norman R. Barrett, MD</td>
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<td>Earle W. Wilkins, Jr., MD</td>
<td>Experience With 500 Cases of Hiatus Hernia</td>
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<td>Thoracic Surgery in the Commonwealth of Medicine</td>
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<td>Alfonso Topete, MD</td>
<td>New Findings in the Coronary-Encephalic Perfusion in Depressive Surgical Cases</td>
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The Graham Education and Research Foundation supports the Evarts A. Graham Memorial Traveling Fellowship and the Research Scholarship programs. Since the inception of the Graham Traveling Fellowship program in 1951, 56 young surgeons from 29 countries have completed their training at thoracic surgery centers throughout North America.

Planned Gifts to the Foundation
Dr. and Mrs. Roger R. Ecker, Alameda, CA Charitable Remainder Trust

For more information about planned giving, please contact the Foundation at 900 Cummings Center, Suite 221-U, Beverly, Massachusetts, 01915, or by phone at (978) 927-8330.
The Evarts A. Graham Memorial Traveling Fellowship was established in 1951 by the American Association for Thoracic Surgery. Funded and administered through the Graham Education and Research Foundation, it provides grants to young surgeons from abroad who have completed their formal training in general, thoracic and cardiovascular surgery. The award allows the recipient to study a year to intensify his training in a program of special interest and to travel to several sites to broaden his overall training and increase his contacts with thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 56 young surgeons from 29 countries have completed their training at thoracic surgical centers.

1. 1951-52 L.L. Whytehead, MD, FRCS, Canada
2. 1953-54 W.B. Ferguson, MB, FRCS, England
3. 1954-55 Lance L. Bromley, M., Chir, FRCS, England
4. 1955-56 Raymond L. Hurt, FRCS, England
5. 1956-57 Mathias Paneth, FRCS, England
6. 1957-58 Peter L. Brunnen, FRCS, Scotland
7. 1958-59 N.G. Meyne, MD, Holland
8. 1960-61 Godrej S. Karai, MD, India
9. 1961-62 Fritz Helmer, MD, Austria
10. 1962-63 Theodor M. Scheinin, MD, Finland
11. 1963-64 Masahiro Saigusa, MD, Japan
12. 1963-64 Adar J. Hallen, MD, Sweden
13. 1964-65 Stuart C. Lennox, MD, England
14. 1964-65 Elias Carapistolis, MD, FACS, Greece
15. 1965-66 Gerhard Friehs, MD, Austria
16. 1965-66 Ary Blesovsky, MD, England
17. 1966-67 C. Peter Clarke, FRACS, Australia
18. 1966-67 G.B. Parulkar, MD, India
19. 1967-68 Claus Jessen, MD, Denmark
20. 1969-70 Peter Bruecke, MD, Austria
21. 1970-71 Michel S. Slim, MD, Lebanon
22. 1971-72 Severi Pellervo Mattila, MD, Finland
23. 1972-73 Yasuyuki Fujiwara, MD, Japan
24. 1973-74 Marc Roger de Leval, MD, England
25. 1974-75 J.J. De Wet Lubbe, MD, South Africa
26. 1975-76 Mieczyslaw Trenkner, MD, Poland
27. 1976-77 Bum Koo Cho, MD, Korea
28. 1977-78 Alan William Gale, MD, FRACP, FRACS, Australia
29. 1978-79 Eduardo Otero Coto, MD, Spain
31. 1981-82 Claudio A. Salles, MD, Brazil
32. 1982-83 Yasushi Shimazaki, MD, Japan
33. 1983-84 Georg S. Kobinia, MD, Austria
34. 1984-85 Aram Smolinsky, MD, Israel
35. 1985-86 Florentino J. Vargas, MD, Argentina
36. 1986-87 Ari L.J. Harjula, MD, Finland
37. 1987-88 Byung-Chul Chang, MD, Korea
38. 1988-89 Wang Cheng, MD, P.R. China
39. 1989-90 Christopher Knott-Craig, MD, South Africa
40. 1991-92 Ko Bando, MD, PhD, Japan
41. 1992-93 Timothy E. Oaks, MD, United States
42. 1993-94 Alain Serraf, MD, Morocco
43. 1995-96 Cornelius McKown Dyke, MD, United States
44. 1996-97 Monica Robotin-Johnson, MD, France
45. 1997-98 Jun Wan, MD, P.R. of China
46. 1998-99 Christian Kreutzer, MD, Argentina
47. 1999-00 Anders Franco-Cereceda, MD, Sweden
48. 2000-01 Albertus M. Scheule, MD, Germany
49. 2001-02 Anna Maria Ciccone, MD, Italy
50. 2002-03 Cliff K.C. Choong, MD, New Zealand
51. 2003-04 Edvin Prifti, MD, Albania
52. 2004-05 Smruti Ranjan Mohanty, MD, India
53. 2005-06 Zsolt Toth, MD, Hungary
54. 2006-07 Ari Mennander, MD, Finland
55. 2007-08 Ioannis Troupoulis, MD, Greece
56. 2008-09 Sachin Talwar, MD, India
57. 2009-10 Juan J. Fliba, MD, PhD, FETCS, Spain
58. 2010-11 Mohsen Ibrahim, MD, Italy
Research Scholarship Recipients
The American Association for Thoracic Surgery Research Scholarship was established by the Association in 1985. Funded by the Graham Education and Research Foundation, the Research Scholarship provides opportunity for research, training and experience for North American surgeons committed to pursuing an academic career in cardiothoracic surgery. The program is undertaken within the first three years after completion of an approved cardiothoracic residency and is two years in duration.

Third Alfred Blalock Research Scholarship
2010-2012 Matthias Peltz, MD
University of Texas Southwestern Medical Center

Third Edward D. Churchill Research Scholarship
2009-2011 Prasad S. Adusumilli, MD
Memorial Sloan-Kettering Cancer Center
William M. Yarbrough, MD
Medical University of South Carolina

Norman E. Shumway Research Scholarship
2008-2010 Alexander S. Krupnick, MD
Washington University
Michael P. Fischbein, MD
Stanford University

Second Dwight Harken Research Scholarship
2007-2009 Shu S. Lin, MD
Duke University Medical Center
American Association for Thoracic Surgery

John W. Kirklin Research Scholarship
2006-2008  Daniel Kreisel, MD
           Washington University
           Christine Lau, MD
           University of Michigan

Second Andrew G. Morrow Research Scholarship
2005-2007  Marc de Perrot, MD, MSc
           University of Toronto/Toronto General Hospital
           Frederick Y. Chen, MD
           Brigham & Women’s Hospital

Second John Alexander Research Scholarship
2004-2006  King F. Kwong, MD
           University of Maryland

Second Robert E. Gross Research Scholarship
2003-2005  Ross M. Bremner, MD, PhD
           St. Joseph’s Hospital
           Vivek Rao, MD, PhD
           Toronto General Hospital

Second Alton Ochsner Research Scholarship
2002-2004  Yolonda L. Colson, MD
           Brigham & Women’s Hospital
           Michael S. Mulligan, MD
           University of Washington Medical Center

Second John H. Gibbon, Jr. Research Scholarship
2001-2003  Richard J. Battafarano, MD, PhD
           University of Maryland
           Carmelo A. Milano, MD
           Duke University Medical Center
Second Alfred Bialock Research Scholarship
2000-2002 Abbas Ardehali, MD
UCLA School of Medicine
Thomas K. Waddell, MD, MSc, PhD
University of Toronto and Toronto General Hospital

The Second Edward D. Churchill Research Scholarship
1999-2001 Joseph B. Shrager, MD
University of Pennsylvania

Dwight Harken Research Scholarship
1998-2000 Bruce Rosengard, MD
Massachusetts General Hospital

Andrew G. Morrow Research Scholarship
1997-1999 Stephen C. Yang, MD
Johns Hopkins University School of Medicine

John Alexander Research Scholarship
1996-1998 Richard Norris Pierson, III, MD
University of Maryland

Robert E. Gross Research Scholarship
1994-1996 Mehmet C. Oz, MD
Columbia Presbyterian Medical Center
Thoralf M. Sundt, III, MD
Mayo Clinic

Alton Ochsner Research Scholarship
Brigham and Women's Hospital
John H. Gibbon, Jr., Research Scholarship
1990-1992 Donald D. Glower, MD
Duke University Medical Center

Alfred Blalock Research Scholarship
1988-1990 Gus J. Vlahakes, MD
Massachusetts General Hospital and Harvard Medical School

Edward D. Churchill Research Scholarship
1986-1988 Mark K. Ferguson, MD
University of Chicago, Department of Surgery

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

Recipients
2010 Richard D. Weisel, Toronto, ON, Canada
2008 Andrew S. Wechsler, Philadelphia, PA
2007 Gerald D. Buckberg, Los Angeles, CA
2005 Alain F. Carpentier, Paris, France
2000 Denton A. Cooley, Houston, TX
1999 Michael E. DeBakey, Houston, TX
1998 Norman E. Shumway, Stanford, CA
1995 John W. Kirklin, Birmingham, AL
Lifetime Achievement Award

The American Association for Thoracic Surgery established the Lifetime Achievement Award in 2003. The Award serves to recognize individuals for their significant contributions to cardiothoracic surgery in the areas of patient care, teaching, research, or community service. Honorees receive a plaque for Lifetime Achievement from the Association presented by the president at the Annual Meeting.

Recipients

2009    Thomas B. Ferguson, St. Louis, MO
2007    Frank C. Spencer, New York, NY
2004    F. Griffith Pearson, Toronto, ON, Canada
C. WALTON LILLEHEI RESIDENT FORUM

Through a generous unrestricted educational grant from St. Jude Medical, Inc., this Forum recognizes the extraordinary contributions to our specialty by a great innovator in congenital and vascular disease. Selected by the Cardiothoracic Residents Committee, the recipient receives a $5,000 award.

Winners

2009  J. Raymond Fitzpatrick, Philadelphia, PA, USA
Tissue Engineered Pro-Angiogenic Fibroblast Matrix Improves Myocardial Perfusion and Function and Limits Ventricular Remodeling Following Infarction

2008  Turner C. Lisle, Charlottesville, VA
Inflammatory Lung Injury After Cardiopulmonary Bypass Is Attenuated by Adenosine A2A Receptor Activation

2007  Leo M. Gazoni, Charlottesville, NC
Timing Is Everything. Pretreatment of Donor Lungs with the Adenosine 2A Receptor Agonist ATL-313 Results in Superior Protection From Lung Ischemia Reperfusion Injury Versus Administration During Reperfusion

2006  Jae Y. Kim, San Francisco, CA
WNT Inhibitory Factor Inhibits Lung Cancer Cell Growth
Amir M. Sheikh, Durham, NC
Proteomics of Brain Injury in a Neonatal Model of Deep Hypothermic Circulatory Arrest

2005  Paul W. Fedak, Toronto, ON Canada
Cell Transplantation Preserves Matrix Homeostasis: A Novel Paracrine Mechanism
2004  Filiberto Rodriguez, Palo Alto, CA  
Alterations of Transmural Strains In the Ischemic Border Zone During Acute Mid-Circumflex Occlusion

Mark F. Berry, Philadelphia, PA  
Targeted Overexpression of Leukemia Inhibitory Factor Preserves Myocardium In Postinfarction Heart Failure

2003  Sunil Singhal, Philadelphia, PA  
Preoperative Viral Gene Transfer of Interferon-Beta Prevents Recurrence and Improves Survival In Advanced Thoracic Malignancies

2002  Subhasis Chatterjee, Philadelphia, PA  
Viral Gene Transfer of the Anti-Apoptotic Factor ARC Protects Against Post Ischemic Heart Failure

2001  Tomasz A. Timek, Palo Alto, CA  
Septal-Lateral Annular Cinching Abolishes Acute Ischemic Mitral Regurgitation In Sheep

2000  Allan S. Stewart, Philadelphia, PA  
Gene Transfer of Bcl-2 Does Not Affect Myocardial Stunning But Ameliorates the Deleterious Effects of Chronic Remodeling

1999  Andrew I. Campbell, Toronto, ON, Canada  
Angiogenic Therapy with Vascular Endothelial Growth Factor Reverses Pulmonary Hypertension

1998  Stephen D. Cassivi, Toronto, ON, Canada  
Transgene Expression Following Adenoviral-Mediated Retransfection of Rat Lungs Is Increased and Prolonged by Transplantation-Level Immunosuppression
ARTICLE 1. NAME

The name of this Corporation is The American Association for Thoracic Surgery (hereinafter the “Association”).

ARTICLE II. PURPOSE

The purposes of the Association shall be:

To associate persons interested in, and carry on activities related to, the science and practice of thoracic surgery, the cure of thoracic disease and the related sciences.

To encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology and therapy, and to correlate and disseminate such knowledge.

To hold scientific meetings featuring free discussion of problems and developments relating to thoracic surgery, and to sponsor a journal for the publication of scientific papers presented at such meetings and other suitable articles.

To succeed to, and continue to carry on the activities formerly conducted by The American Association for Thoracic Surgery, an unincorporated association.
ARTICLE III. MEMBERSHIP

Section 1. There shall be three classes of members: Honorary, Senior, and Active. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws. Honorary members shall have the privilege of voting but shall not be eligible to hold office.

Section 2. Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with concurrence of the Association.

Section 3. The number of Senior Members shall be unlimited. Active Members automatically advance to Senior Membership at the age of seventy years or upon request after the age of sixty-five. In addition, a younger Active Member may be eligible for Senior Membership by petition to and approval by the Council.

Section 4. Active Membership shall be limited to seven hundred. A candidate to be eligible must be a physician and 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 and Article IX hereof. 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.** Members in good standing may resign at any time. Resignation shall not relieve a member of the obligation to pay outstanding dues or assessments. The Council may delay or refuse to accept the resignation of any member who has received written notification that the Ethics Committee is conducting an investigation concerning the member’s conduct until a final decision on the matter has been issued in accordance with the Procedural Guidelines for Ethics Complaints Against AATS Members established by the Council. A member whose resignation is not accepted by the Council is not required to pay dues or assessments after submission of the resignation.

**Section 9.** The Council shall recommend that any Active Member whose dues are in arrears for two years shall have his/her membership terminated, except that if the Ethics Committee is conducting an investigation concerning the member’s conduct, such member’s membership shall not be terminated until a final decision on the matter has been issued in accordance with the Procedural Guidelines for Ethics Complaints Against AATS Members established by the Council.

**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, and six Councilors. The Editor of the Association and the chairs of the Education Committee and the Scientific Affairs and Government Relations Committee shall be members 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:

It may not levy any general assessments against the membership but it may, in individual cases, waive annual dues or assessments.

It may not change the Articles of Incorporation or By-Laws.

It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article III, Sections 8 and 9 and Article IX.

The Council may adopt such rules and regulations for the conduct of the Association's affairs as the Council deems necessary or advisable, including specifically the Procedural Guidelines for Ethics Complaints Against AATS Members.

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 five 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. When appropriate, one of the Councilors shall be elected from among the non-North American members of the Association to serve for a three-year term of office. No Councilor may be reelected to succeed himself/herself. Any Councilor so elected shall take office upon the conclusion of the annual meeting at which he/she 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/she shall preside at all meetings 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/she 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/she shall serve as Treasurer of the Association and as a member of the Program Committee. When deemed appropriate an Active or Senior Member may be elected to serve as an understudy to the Treasurer in anticipation of the latter's retirement from office.

Section 8. The Editor of the Association is not an officer of the Association. The Editor 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 be reappointed to no more than two additional one-year terms. The Editor shall
serve as the Editor of the Official Journal and shall be ex officio the Chair 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 eight 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.

Section 3. The Program Committee shall consist of at least 16 members: the President, the President-Elect, the Vice President, the Secretary, the Treasurer, the Editor, the Chair of the Education Committee, and at least 9 members-at-large, three each representing the areas of adult cardiac, pediatric cardiac and general thoracic surgery. The President or his/her designee shall serve as the Chair of this Committee. 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 Chair shall serve as Historian and the Council may, if it so desires, appoint one of its own members to serve as Chair 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 Chair. 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 Chairs 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 Council. 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 Graham Fellowship candidates to the Graham Education and Research Foundation, and to carry out other business pertaining to the Fellowship and Fellows, past, present and future.
Section 8. The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, ex officio, the chairman of this board and shall be privileged to appoint and indefinitely reappoint such members of the Association, regardless of class of membership, and such non-members of the Association as in his/her opinion may be best calculated to meet the editorial requirements of the Association.

Section 9. The Ethics Committee shall consist of seven members appointed by the Council, one of whom shall be named as Chair by the Council. The members shall serve a five year term, and may be reappointed. The Ethics Committee shall investigate complaints alleging ethical breaches of Association members received in writing or it may investigate a member's conduct on its own initiative. A majority of the Ethics Committee shall constitute a quorum, and the act of a majority of the members present at a meeting at which a quorum is present shall be the act of the Ethics Committee.

The Ethics Committee may discipline any member for (i) failure to meet the requirements for membership in the Association, (ii) conduct in violation of these By-Laws, the Code of Ethics adopted by the membership of the Association, or the Association's rules and policies, (iii) being convicted of or pleading guilty to a felony or any crime arising out of the practice of medicine or involving moral turpitude, (iv) being disciplined by, or otherwise having the member's right to practice medicine limited, suspended, terminated or otherwise affected by, any medical licensing authority, or (v) engaging in conduct inconsistent with the purposes of the Association.

Section 10. The Education Committee shall consist of nine (9) members with three (3) members being appointed each year by the Council for a three (3) year term. At least three (3) members shall represent the areas of adult cardiac, pediatric cardiac and general thoracic surgery. In addition, a chair shall be appointed by the Council for a three (3) year term who shall be a member of the Council, ex officio without vote and a member of the Program Committee. The committee shall be responsible for identifying areas within the specialty for which additional training and education are necessary and the selection of topics and chairs for postgraduate activity to address these areas.

Section 11. The Committee on Publications shall consist of the Secretary as Chair, the President-Elect, the Vice President, the Treasurer, and the Executive Director. The Committee shall oversee the business relationships
between the Association and the publisher of its journal maintain liaison among the publisher, the Editor, and the Council, and shall have advisory oversight for all official scientific publications of the Association and make recommendations to the Editor and the Council.

Section 12. The Cardiothoracic Residents Committee shall consist of eight members appointed by the Council. Two members shall be appointed each year for a four-year term with the senior two members of the Committee serving as Co-Chairs. At least two members shall represent adult cardiac surgery, general thoracic surgery, congenital heart surgery, and the Editorial Advisory Board of THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY. The duties of the committee shall include the development of educational activities specifically directed at cardiothoracic residents, the review of scientific material submitted for any resident award program and the selection of any such awardees and the responsibility for recommending to the Council the generation of new programs of interest to cardiothoracic residents.

Section 13. The Scientific Affairs and Government Relations Committee shall consist of a Chair, appointed by the Council who shall serve a term of three (3) years and shall be a member of the Council, ex officio, without vote, the Secretary who shall serve ex-officio and such members as the Council may deem appropriate to fulfill the responsibilities of the committee who shall serve for one (1) year. The committee shall be responsible for identifying and interacting with the various Federal agencies and institutions which affect research activities and funding in cardiothoracic surgery. It shall serve as a resource to the membership in the development of programmatic activities appropriate to research efforts in the specialty.

Section 14. The Research Scholarship Committee shall consist of nine (9) members appointed by the Council. Two members shall be appointed each year for a four-year term with the senior two members of the Committee serving as Co-Chairs. Two members shall represent adult cardiac surgery, general thoracic surgery, congenital heart surgery and transplant surgery. The duties of the Committee shall include the review of scientific material submitted for all research award programs and to recommend research award candidates to the Graham Education and Research Foundation, and the responsibility for recommending to the Council the generation of new research programs of interest to cardiothoracic surgeons.
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 the Council. If, at the end of any fiscal year, there is a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Active members to contribute the necessary amount so that no deficit is carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment which shall become an obligatory charge against the classes of members affected thereby.

Section 3. To meet the current expenses of the Association, there shall be available all revenue derived by the Association.

ARTICLE VIII. MEETINGS

Section 1. The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council and the provisions of the By-Laws.

Section 2. Notice of any meeting of the Association shall be given to each member of the Association not less than five nor more than forty days prior to any annual meeting and not less than thirty nor more than forty days prior to any special meeting by written or printed notice delivered personally or electronically by mail, by or at the direction of the Council, the President or the Secretary. Such notice shall state the place, day and hour of the meeting and in the case of a special meeting shall also state the purpose or purposes for which the meeting is called.

Section 3. A special meeting of the Association may be called by the Council or on the written request of fifteen members delivered to the Council, the President or the Secretary. The specific purposes of the meeting must be stated in the request.

Section 4. Attendance at annual meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members, but it shall be expected from all Active Members.
Section 5. Each annual meeting shall have at least two executive sessions.

Section 6. When the Association convenes for its annual meeting, it shall immediately go into the first executive session, but the business at this session shall be limited to:

- Appointment of necessary committees.
- Miscellaneous business of an urgent nature.

Section 7. The second executive session of the Association shall be held during the afternoon of the second day of the meeting. The business at this session shall include, but is not limited to:

- Reading or waiver of reading of the minutes of the preceding meetings of the Association and the Council.
- Report of the Treasurer of the last fiscal year.
- Audit Report.
- Report of the Necrology Committee.
- Report of the Program Committee.
- Action on amendments to the Articles of Incorporation and By-Laws, if any.
- Action on recommendations emanating from the Council.
- Unfinished Business.
- New Business.
- Report of the Membership Committee.
- Election of new members.
- Report of Nominating Committee.
- Election of officers.

Section 8. Except where otherwise required by law or these By-Laws, all questions at a meeting of the members shall be decided by a majority vote of the members present in person and voting. Voting by proxy is not permitted.

Section 9. Fifty voting members present in person shall constitute a quorum at a meeting of members.
Section 10. While the scientific session of the annual meeting is held primarily for the benefit of the members of the Association, it may be open to non-members who are able to submit satisfactory credentials, who register in a specified manner, and who pay such registration fee as may be determined and published by the Council from year to year.

Section 11. There shall be an annual meeting of the Council held during the annual meeting of the Association. Additional meetings of the Council may be called on not less than seven days' prior written or telephonic notice by the President, the Secretary or any three members of the Council.

Section 12. Six 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. DISCIPLINE

Any member of the Association may be admonished, censured, placed on probation, suspended or expelled for (i) failure to meet the requirements for membership in the Association, (ii) conduct in violation of these By-Laws, the Code of Ethics adopted by the membership of the Association, or the Association’s rules and policies, (iii) being convicted of or pleading guilty to a felony or any crime arising out of the practice of medicine or involving moral turpitude, (iv) being disciplined by, or otherwise having the member’s right to practice medicine limited, suspended, terminated or otherwise affected by, any medical licensing authority, or (v) engaging in conduct inconsistent with the purposes of the Association. Disciplinary actions shall be conducted in
accordance with the Procedural Guidelines for Ethics Complaints Against AATS Members established by the Council, which procedures shall include written notification to the member of the infraction and an opportunity for a hearing.

**ARTICLE X. INDEMNIFICATION**

Section 1. The Association shall indemnify any and all of its Councilors (hereinafter in this Article referred to as "directors"), officers, employees, committee members and agents, or former directors, officers, employees, committee members and agents, or other 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, officers, employees, committee members or agents of the Association, or directors or officers of such other corporation or association, provided, however, that the foregoing shall not apply to matters as to which any such director, officer, employee, committee member or agent, or former director, officer, employee, committee member or agent 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, officers, employees, committee members or agents, or former directors, officers, employees, committee members or agents, 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, officers, employees, committee members or agents of the Association or directors or officers 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 XI. PAPERS

Section 1. All papers read before the Association shall become the property of the Association. Authors shall provide original or electronic copies of their manuscripts to the Editor, prior to 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 XII. INITIATION FEES, DUES AND ASSESSMENTS

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

Section 2. Annual dues for Active Members shall be established by the Council 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 established by the Council.

Section 5. Active Members must subscribe to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY to retain their membership status.

Section 6. Subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY is optional for Senior Members.

Section 7. Bills for membership dues and for subscriptions to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY will be mailed to members by the Treasurer at the beginning of the fiscal year.

ARTICLE XIII. 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 XIV. 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, 2009
JOINT AATS/STS POLICY STATEMENT ON EDUCATIONAL COURSES

The American Association for Thoracic Surgery (AATS) and the Society of Thoracic Surgeons (STS) strongly discourage others from holding educational events in the general locations of STS or AATS sponsored meetings, including the STS and AATS Annual Meetings and the jointly sponsored Tech-Con Programs and Postgraduate Courses, during the 48 hours preceding those meetings. Such unauthorized events are deemed detrimental to STS and AATS educational efforts, and create excessive demands on the time and resources of cardiothoracic surgeons. Both STS and AATS urge their members and corporate partners, as well as other education providers, to respect and adhere to this policy. In turn, each organization will seek to accommodate others in their efforts to reach the cardiothoracic surgery audience in conjunction with their respective and jointly conducted meetings.

GUIDELINES FOR USING THE CARDIOTHORACIC OPERATION AS A TEACHING INSTRUMENT

1. When planning to record or broadcast an operation, surgeons must pay special attention to the needs and rights of the potential patient-subject.
   a. A patient’s informed consent for participating as a subject in a live or taped broadcast must be obtained directly by the operating surgeon.
   b. The surgeon must disclose the fact of increased risks of harm to the patient and the uncertainty of the degree of such risks, as well as the composition and size of the audience and estimates of the potential educational benefits to participating surgeons.
   c. The attending surgeon must take all necessary steps to protect the patient-subject’s privacy and to ensure confidentiality of all medical information.
2. Generally, recorded broadcasts, either edited or unedited, are preferable to live surgery broadcasts because recordings intended for later broadcast pose fewer risks of harm to patients.
3. Teaching surgical techniques by live surgery observation in the surgeon's home operating room is a time honored, acceptable practice.

4. Surgeons should not participate in live surgery broadcasts to the public using any medium, including television and the Internet.

5. National and international cardiothoracic societies should consider prohibiting live surgery broadcasts to large audiences at their annual meetings.

6. Live surgery broadcasts to professional audiences of any size become progressively less acceptable with more rigid scheduling constraints, increasing complexity of the operation, decreasing educational value of the procedure, greater intensity of the surgeon's interaction with the audience, and less familiarity of the surgeon with the operating room environment. On these grounds, live surgery broadcasts are subject to the following conditions:

a. Cardiothoracic surgeons should not participate in live surgery broadcasts when rigid broadcast schedules constrain the operation's starting time or duration or when a specific predetermined operation must be fit into a specific time frame. Operations selected for live surgery broadcasts are most acceptable when the operation focuses solely on a particular patient who has a condition that warrants live broadcast.

b. Operations of greater educational value to the surgeons in the audience, relative to their clinical needs, should be chosen over operations of lesser educational value. Operations are inappropriate for live broadcast if intended to show that an operation can be done rather to demonstrate to others how to do it. Cardiothoracic surgeons should not participate in broadcasts of operations that have a major purpose of aggrandizement of the surgeon or of the surgeon's operating facility.
d. The operating surgeon should be thoroughly familiar with and experienced in the procedure being broadcast and with the specific medical devices and tools being demonstrated. Innovative operations and rare procedures that the surgeon has never or only occasionally performed previously should not be broadcast because they lack educational value and increase the need for the surgeon's undivided attention.

e. Whenever possible, surgery should be broadcast from the surgeon's home operating room. When this is not possible, the operative facility should be configured as closely as possible to the surgeon's home operating room environment. Only highly experienced operating room staff who are fluent in the surgeon's preferred language should participate, preferably the surgeon's own staff. The surgeon must ensure that the video crew does not interfere with the progress of the operation, whether filming is intended for live or recorded broadcast.

f. Because discussion with a remote audience during an operation may distract the surgeon, discussions should be one-way, from surgeon to audience. If a two-way discussion is demonstrably essential to the educational value, questions and comments from the audience should be controlled, for example, relayed through a moderator who alone can communicate with the surgeon.

g. Cardiothoracic surgeons should not participate in any capacity in live surgery programs that violate these guidelines.

h. The operating surgeon has a responsibility to ensure completion of the following requirements before each broadcast:

i. The operating facility, if not in the surgeon's home institution, should be suitable for the conduct of the operation to be broadcast.
ii. A preoperative conference should be held with the principal parties, including the operating surgeon and key medical and technical (filming) staff, to review the ethical guidelines and safety standards under which the operation will be performed.

iii. A reliable mechanism should be in place for the audience to receive follow-up reports on the outcome of the operation within 24 hours and the status of the patient 30 days after the broadcast.

*Adopted by AATS Council on May 11, 2008*
1. When caring for patients, members must hold the patient’s welfare paramount.

2. Members must prescribe drugs, devices, and treatment based solely on medical considerations and patients’ preferences, regardless of any direct or indirect inducements by industry.

3. Members should inform a patient of any conflicts of interest arising from their relationships with or investments in companies that manufacture or supply medications, devices, or therapies to be used for the patient. Any conflict of interest must be resolved in the best interest of the patient.

4. When faced with a conflict of interest that cannot be easily resolved, a member should consult with disinterested colleagues or an institutional ethics committee to determine if an actual or potential conflict of interest is present and, if so, how to address it.

5. Members should accept no gifts from industry, regardless of value. Drug samples intended only for the use of patients are not considered gifts.

6. Members must accept no direct or indirect financial inducements from industry for utilizing one particular device or drug over another or for switching from one manufacturer’s product to another. Unacceptable inducements include payment over and above the actual cost of completing post-marketing surveys of drug or device use.

7. Members who enter into a consulting agreement should be able to document the following.
   - The consulting service was needed.
   - The consulting service was actually provided.
The payment for consultation was not higher than fair market value.

No compensation or other incentive was based upon the volume or value of business associated with the agreement.

8. Members should disclose their own or their institution’s financial relationship with the manufacturer of a drug or device whenever clinical research or experience with a particular procedure or device is presented at a meeting or is published.

9. Members who serve as the principal investigator of any research project should report fully any influence from funding sources on designing the project, controlling access to the data, preparing a presentation or paper, or controlling timing of presentation or publication.

10. Members should accept no remuneration from industry to attend any social functions that have no educational content.

11. Members should not accept any financial support from industry for attendance at any educational event. Residency program directors should ensure that industry grants, which may be extended to residents, are made through the sponsoring institution, not directly to the resident.

12. Members who are speakers at an industry-supported educational event should accept only reasonable honoraria and reimbursement for travel, meals, and lodging; all such payments should be made by the conference sponsor, not directly by industry.

13. Members should attend a company-sponsored event only when the major purpose of the event is education and training in the proper use of the company’s products; the only financial considerations should be reimbursement for travel, meals, and lodging. Members should not accept reimbursement for attending such an educational event if the event’s location constitutes an inducement that is independent of the event’s educational value.

Adopted by AATS Council in January 2009
AMERICAN ASSOCIATION FOR THORACIC SURGERY

2009 – 2010
GEOGRAPHICAL ROSTER
(Current as of February, 2010)

NORTH AMERICA

UNITED STATES

ALABAMA

Birmingham
- Athanasuleas, Constantine L
- Cerfolio, Robert J
- Holman, William L
- Kahn, Donald R
- Kirklin, James K
- McGiffin, David C

Indian Springs
- Pacifico, Albert D

Mobile
- LoCicero, Joseph, III

Montgomery
- Simmons, Earl M

ALASKA

Anchorage
- Misbach, Gregory A

ARIZONA

Carefree
- Michaelis, Lawrence

Chandler
- Shennib, Hani

Fountain Hills
- Mullany, Charles J

Phoenix
- Pearl, Jeffrey M
- Vaughn, Cecil C

Scottsdale
- Fisk, R. Leighton
- Pluth, James R
- Shields, Thomas W
- Trastek, Victor F

Tempe
- Cornell, William P

Tucson
- Copeland, Jack G
- Sanderson, Richard G
- Sethi, Gulshan K

ARKANSAS

Little Rock
- Campbell, Gilbert S
- Jaquiss, Robert D
- Read, Raymond C
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AMERICAN ASSOCIATION FOR THORACIC SURGERY

Pebble Beach
Miller, George E, Jr

Portola Valley
Fogarty, Thomas J

Rancho Palos Verdes
Mandal, Ashis K

Rancho Santa Fe
Geha, Alexander S

Sacramento
Berkoff, Herbert A
Follette, David M
Harlan, Bradley J
Young, J. Nilas

San Diego
Dembitsky, Walter P
Jamieson, Stuart W
Lamberti, John J
Miller, Fletcher A
Moreno-Cabral, Ricardo J
Thistlethwaite, Patricia A
Trummer, Max J

San Francisco
Ellis, Robert J
Hill, J. Donald
Jablons, David M
Merrick, Scot H
Ratcliffe, Mark B
Yee, Edward S

San Jose
Oakes, David D

San Rafael
Roe, Benson B

Santa Ana
Gazzaniga, Alan B

Santa Barbara
Jahnke, Edward J
Love, Jack W

Santa Cruz
Fishman, Noel H

Santa Monica
Fonkalsrud, Eric W
Morton, Donald L
Robertson, John M

Sausalito
Zaroff, Lawrence I

Stanford
Mainwaring, Richard D
Fann, James I
Hanley, Frank L
Mark, James B.D.
Miller, D. Craig
Mitchell, R. Scott
Oyer, Philip E
Reddy, V. Mohan
Reitz, Bruce A
Robbins, Robert C
Shrago, Joseph B
Whyte, Richard I

Tiburon
Heydorn, William H

Torrance
Carey, Joseph S
Moore, Thomas C
State, David

Ventura
Brandt, Berkeley, Ill
Dart, Charles H, Jr

Walnut Creek
May, Ivan A

COLORADO

Aurora
Campbell, David N
Clarke, David R
Cleveland, Joseph C, Jr
Fullerton, David A
Grover, Frederick L
Pomerantz, Marvin

Denver
Eiseman, Ben
Hopeman, Alan R
Paton, Bruce C
Rainer, W. Gerald

Greenwood Village
Pappas, George
Parker
Olinger, Gordon N

Snowmass Village
Mills, Lawrence J

Steamboat Springs
Greenberg, Jack J

CONNECTICUT

Bridgeport
Rose, Daniel M

Essex
Jaretzki, Alfred, III

Greenwich
Waters, Paul F

New Haven
Detterbeck, Frank C
Elefteriades, John A
Kopf, Gary S
Shinoka, Toshiharu
Tellides, George

North Haven
Hammond, Graeme L

Old Greenwich
Brodman, Richard F

Waterbury
Sanchez, Juan A

Woodbridge
Stern, Harold

DELAWARE

Dover
Mannion, John D

Greenville
Norwood, William I

Newark
Banbury, Michael K
Gardner, Timothy J

DISTRICT OF COLUMBIA

Washington
Jonas, Richard A
Katz, Nevin M
Keshishian, John M
Marshall, M. Blair
Simmons, Robert L

FLORIDA

Atlantic Beach
Stranahan, Allan

Aventura
Bregman, David

Bal Harbour
Grondin, Pierre R

Belleair
Lasley, Charles H

Boca Raton
Kiser, Joseph C
Litwin, S. Bert

Coconut Grove
Center, Sol

Delray Beach
Rosensweig, Jacob

Fernandina Beach
Malm, James R

Gainesville
Alexander, James A
Wheat, Myron W, Jr

Jacksonville
Edwards, Fred H
Koster, J. Kenneth, Jr
Landolfo, Kevin P

Jupiter
Gerbasi, Francis S

Longboat Key
Lajos, Thomas Z

Miami Beach
Spear, Harold C

564
AMERICAN ASSOCIATION FOR THORACIC SURGERY

**Miami**
Harrison, Lynn H, Jr
Jude, James R
Kaiser, Gerard A
Kurlansky, Paul A
Nguyen, Dao M
Pham, Si Mai
Reis, Robert L
Ricci, Marco
Salerno, Tomas A
Subramanian, S
Thurer, Richard J

**Naples**
Gonzalez, Luis L
Linberg, Eugene J
MacGregor, David C

**Orlando**
Accola, Kevin D
DeCampili, William M

**Palm City**
Timmis, Hilary H

**Ponte Vedra Beach**
Barnhorst, Donald A
Gilbert, Joseph, Jr

**Saint Petersburg**
Daicoff, George
Jacobs, Jeffrey P

**Tallahassee**
Kraeft, Nelson H
Lambert, Cary J

**Tamarac**
Friedlander, Ralph

**Tampa**
Angell, William W
Robinson, Lary A

**Georgia**

**Atlanta**
Chen, Edward P
Craver, Joseph M

**Gott, John P**
**Guyton, Robert A**
**Hatcher, Charles R, Jr**
**Kanter, Kirk R**
**Kirshbom, Paul M**
**Lee, Arthur B, Jr**
**Mansour, Kamal A**
**Miller, Daniel L**
**Miller, Joseph I, Jr**
**Puskas, John D**
**Symbas, Panagiotis**
**Vassiliades, Thomas A, Jr**
**Vega, J. David**
**Vinten-Johansen, Jakob**
**Williams, Willis H**

**Chickamauga**
Hall, David P

**Dunwoody**
Rivkin, Laurence M

**Evans**
Zumbro, G. Lionel, Jr

**Kennesaw**
Northrup, William F, III

**Macon**
Dalton, Martin L, Jr
Van De Water, Joseph M

**Savannah**
Yeh, Thomas J

**Hawaii**

**Honolulu**
Ching, Nathaniel P
Gebauer, Paul W
McNamara, J. Judson

**Kailua**
Young, William P

**Waikiki**
Cochran, Richard P
IDAHO

Boise
Herr, Rodney H

ILLINOIS

Burr Ridge
Blakeman, Bradford P

Chicago
Amato, Joseph J
Backer, Carl L
Barker, Walter L
Breyer, Robert H
Campbell, Charles D
DeCamp, Malcolm M
Faber, L. Penfield
Ferguson, Mark K
Goldin, Marshall D
Hanlon, C. Rollins
Higgins, Robert S.D.
Jeevanandam, Valluvan
Kittle, C. Frederick
Liptay, Michael
Massad, Malek G
McCarthy, Patrick M
Najafi, Hassan
Raffensperger, John
Raman, Jaishankar
Replogle, Robert L
Snow, Norman J
Tatooles, Constantine J
Vaneco, Robert M
Warren, William H
Zajtchuk, Rostik

Elk Grove Village
Sullivan, Henry J

Evanston
Head, Louis R

Glenview
Montoya, Alvaro

Lake Forest
Weinberg, Milton, Jr

Maywood
Love, Robert B
Pifarre, Roque

Oak Lawn
Ilbawi, Michel N

Oak Park
Hartz, Renee S

River Forest
Mason, G. Robert

Springfield
Hazelrigg, Stephen R

Western Springs
Thomas, Paul A, Jr

Willowbrook
Leininger, Bernard J

Winnetka
Fry, Willard A

INDIANA

Fort Wayne
Ladowski, Joseph S

Indianapolis
Brown, John W
Kesler, Kenneth A
King, Harold
King, Robert D
Mahomed, Yousuf
Mandelbaum, Isidore
Rodefeld, Mark D
Shumacker, Harris B, Jr
Siderys, Harry
Turrentine, Mark W

Valparaiso
O’Neill, Martin J, Jr

IOWA

Cedar Rapids
Levett, James M
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Council Bluffs
Sellers, Robert D

Des Moines
Zeff, Robert H

Iowa City
Behrendt, Douglas M
Iannettoni, Mark D
Richenbacher, Wayne E
Rossi, Nicholas P
Stanford, William

Urbandale
Phillips, Steven J

KANSAS

Kansas City
Reed, William A

Lawrence
Miller, Don R

Mission Hills
Ashcraft, Keith W

Overland Park
Killen, Duncan A

Prairie Village
Holder, Thomas M
Piehler, Jeffrey M

Shawnee Mission
Padula, Richard T

KENTUCKY

LaGrange
Cox, James L

Lexington
Crutcher, Richard R
Ferraris, Victor A
Todd, Edward P
Zwischenberger, Joseph B

Louisville
Austin, Erle H, III
Dowling, Robert D

Gray, Laman A, Jr
Mahaffey, Daniel E
Slaughter, Mark

LOUISIANA

Alexandria
Webb, Watts R

Baton Rouge
Berry, B. Eugene

Nathitoches
Bloodwell, Robert D

New Orleans
Blalock, John B
DeCamp, Paul T
DeLeon, Serafin Y
Hewitt, Robert L
Lindsey, Edward S
Moulder, Peter V
Ochsner, John L

Shreveport
Mancini, Mary C

MAINE

Cape Elizabeth
Bredenberg, Carl E

Portland
Morton, Jeremy R

Rome
Tarnay, Thomas J

Sedgwick
Siewers, Ralph D

MARYLAND

Baltimore
Attar, Safuh
Baker, R. Robinson
Battafarano, Richard J
Baumgartner, William A
Brock, Malcolm V
Brown, James M
Cameron, Duke E
Conte, John V
Gammie, James S
Greene, Peter S
Griffith, Bartley P
Haller, J. Alex, Jr
McLaughlin, Joseph S
Pierson, Richard N, III
Vricella, Luca A
Watkins, Levi, Jr
Yang, Stephen C
Yuh, David D

Bethesda
Horvath, Keith A
Schump, David S

Glen Arm
Turney, Stephen Z

Lutherville Timonium
Hankins, John R

Lutherville
Salomon, Neal W

Reisterstown
Heitmiller, Richard F

Towson
Krasna, Mark J

Worton
Walkup, Harry E

MASSACHUSETTS

Amherst
Levine, Frederick H

Beverly
Maloney, William T

Boston
Agnihotri, Arvind K
Akins, Cary W
Allan, James S
Aranki, Sary F

Austen, W. Gerald
Bolman, R. Morton, III
Bueno, Raphael
Burke, John F
Chen, Frederick Y
Cohn, Lawrence H
Collins, John J, Jr
Colson, Yolonda L
Couper, Gregory S
Daggett, Willard M
Daly, Benedict D.T.
Del Nido, Pedro J
Fernando, Hiran C
Gaiassett, Henning A
Jaklitsch, Michael T
Lazar, Harold L
Levitsky, Sidney
MacGillivray, Thomas E
Madsen, Joren C
Mathisen, Douglas J
Mayer, John E
Mentzer, Steven J
Pigula, Frank A
Rheinlander, Harold F
Rosengard, Bruce R
Sugarbaker, David J
Swanson, Scott J
Thurer, Robert L
Torchiana, David F
Urschel, John D
Vlahakes, Gus J
Wain, John C, Jr
Warner, Kenneth G
Wright, Cameron D

Boylston
Okike, Okike N

Brookline
Berger, Robert L
Ellis, F. Henry, Jr
Neirotti, Rodolfo

Cambridge
Malcolm, John A
Weintraub, Ronald M

Centerville
Lefemine, Armand A
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Chestnut Hill
Bougas, James A

Concord
Norman, John C

Falmouth
McElvein, Richard B

Framingham
Bernhard, William F

Melrose
Desforges, Gerard

North Andover
Cook, William A

Plymouth
Moran, John M

Salem
Vander Salm, Thomas J

Springfield
Engelman, Richard M
Rousou, John A

Sudbury
Shahian, David M

Wayland
Moncure, Ashby C

West Hampton
Wukasch, Don C

West Roxbury
Barsamian, Ernest M
Neptune, Wilford B

Westwood
Schuster, Samuel R

Wilkins, Earle W

Worcester
Conlan, A. Alan
Graeber, Geoffrey M

MICHIGAN

Ann Arbor
Bartlett, Robert H
Bolling, Steven F
Bove, Edward L
Chang, Andrew C
Deeb, G. Michael
Gago, Otto
Greenfield, Lazar J
Neerken, A. John
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Orringer, Mark B
Pagani, Francis D
Prager, Richard L
Sloan, Herbert E

Detroit
Arbulu, Agustin
Baciewicz, Frank A, Jr
Delius, Ralph E
Mentzer, Robert M, Jr
Stephenson, Larry W
Walters, Henry L, III
Wilson, Robert F

Grand Rapids
Harrison, Robert W
Rasmussen, Richard A
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Grasse Pointe Farms
Javid, Hushang

Grosse Point
Silverman, Norman A

West Bloomfield
Arciniegas, Eduardo

MINNESOTA

Coon Rapids
Joyce, Lyle D
### 90th Annual Meeting May 1–May 5, 2010
#### Toronto, ON, Canada

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<td>Naunheim, Keith S</td>
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<td>Pasque, Michael K</td>
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<td>Patterson, Alec</td>
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<td>Roper, Charles L</td>
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<td>Sassier, William F</td>
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<td>Webster Groves</td>
<td>Kaiser, George C</td>
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</tbody>
</table>
AMERICAN ASSOCIATION FOR THORACIC SURGERY

MONTANA

Columbia Falls
Myerowitz, P. David
Missoula
Duran, Carlos Gomez

NEBRASKA

Bennington
Fleming, William H

NEVADA

Zephyr Cove
Kerth, William J

NEW HAMPSHIRE

Hanover
Plume, Stephen K
Lebanon
Nugent, William C
Sanders, John H, Jr
Stratham
Gaensler, Edward A

NEW JERSEY

Alpine
Holswade, George R
Basking Ridge
Lewis, Ralph J
Belleville
Gerard, Franklyn P
Browns Mills
McGrath, Lynn B
Camden
Camishion, Rudolph C
Englewood
Ergin, M. Arisan
Fort Lee
Conklin, Edward F
Jersey City
Demos, Nicholas J
Moorrestown
DelRossi, Anthony J
Fernandez, Javier
Morristown
Parr, Grant V.S.
Neptune
Roberts, Arthur J
New Brunswick
Mackenzie, James W
Scholz, Peter M
Newark
Donahoo, James
McBride, Lawrence R
Parsonnet, Victor
Paramus
Korst, Robert J
Pittstown
Garzoni, Antonio A
Pompton Plains
Adler, Richard H
South Orange
Gielchinsky, Isaac
Swan, Kenneth G
Tenafly
Gerst, Paul H
Wallish, Eugene

NEW MEXICO

Albuquerque
Dietl, Charles A
Wernly, Jorge A
Alto
Sutherland, R. Duncan
Buena Vista
Thal, Alan P

Santa Fe
Davila, Julio C

NEW YORK

Albany
Moore, Darroch

Bronx
Attai, Lari A
Ford, Joseph M
Goldstein, Daniel
Hirose, Teruo

Bronxville
Frater, Robert W.M.

Brooklyn
Acinapura, Anthony J
Cunningham, Joseph N
Lahey, Stephen J
Levowitz, Bernard S
Sawyer, Philip N

Buffalo
Demmy, Todd L
Hoover, Eddie L

Canandaigua
Craver, William L

Chappaqua
Fell, Stanley C

Dewitt
Parker, Frederick B, Jr

East Amherst
Bhayana, Joginder N

East Quogue
McCormack, Patricia M

Fishers Island
Baue, Arthur E

Garden City
Hines, George L

Germantown
Reed, George E

Mamaroneck
Steichen, Felicien M

New York
Adams, David H
Altorki, Nasser K
Anagnostopoulos, C.E.
Argenziano, Michael
Bacha, Emile A
Bains, Manjit S
Boyd, Arthur D
Chen, Jonathan M
Culliford, Alfred T, III
DeAnda, Abe, Jr
Downey, Robert J
Filouf, Farzan
Flores, Raja M
Galloway, Aubrey C, Jr
Girardi, Leonard N
Green, George E
Griep, Randall B
Grossi, Eugene A
Hochberg, Mark S
Isom, O. Wayne
King, Thomas C
Kirschner, Paul A
Krieger, Karl H
Lacour-Gayet, Francois G
Lee, Paul C
Litwak, Robert S
Michler, Robert E
Moggio, Richard A
Mosca, Ralph S
Naka, Yoshifumi
Oz, Mehmet C
Pass, Harvey I
Port, Jeffrey L
Quaegbeur, Jan Modest
Redo, S. Frank
Rosa, Eric A
Rusch, Valerie W
Smith, Craig R
Sonett, Joshua R
Spencer, Frank C
Spotnitz, Henry M
Subramanian, Valavanur A
Tice, David A
Veith, Frank J
Wichert, Walter, Jr
Wolff, William I
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Northport
Soroff, Harry S

Pelham
Adams, Peter X

Plattsburgh
Potter, Robert T

Rochester
DeWeese, James A
Hicks, George L, Jr
Schwartz, Seymour I
Stewart, Scott

Roslyn
Thomson, Norman B, Jr
Wisoff, George

Stony Brook
Bilfinger, Thomas V
Rosengart, Todd K

Syracuse
Kohman, Leslie J
Meyer, John A

Valhalla
Lansman, Steven L
Spielvogel, David

Voorheesville
Foster, Eric D

Williamsville
Andersen, Murray N

NORTH CAROLINA

Asheville
Hill, Ronald C
Kroncke, George M
Takaro, Timothy

Biltmore Forest
Watson, Donald C

Chapel Hill
Bowman, Frederick, Jr
Egan, Thomas M
Feins, Richard H
Mill, Michael R
Oldham, H. Newland, Jr

Sink, James D
Starek, Peter J
Wilcox, Benson R

Charlotte
Robicsek, Francis
Selle, Jay G

Durham
Anderson, Robert W
D’Amico, Thomas A
Davis, R. Duane, Jr
Glower, Donald D
Harpole, David H, Jr
Jaggers, James
Jones, Robert H
Lowe, James E
Milano, Carmelo A
Smith, Peter K
Wolfe, Walter G

Greensboro
Van Trigt, Peter, III

Greenville
Chitwood, W. Randolph, Jr
Elbeery, Joseph R
Ferguson, T. Bruce, Jr
Kypson, Alan P

High Point
Mills, Stephen A

Highlands
Mullen, Donald C

Southport
Murray, Gordon F

Winston-Salem
Hammon, John W, Jr
Hudspeth, Allen S
Kon, Neal D
Meredith, Jesse H

OHIO

Chagrin Falls
Ankeney, Jay L

Cincinnati
Albers, John E
Gallard, George M
Eghtesady, Pirooz
Flege, John B, Jr
Helmsworth, James A
Hiratzka, Loren F
Ivey, Tom D
Manning, Peter B
Smith, J. Michael
Wilson, James Miller
Wolf, Randall K
Wright, Creighton B

Cleveland
Blackstone, Eugene H
Cobanoglu, Adnan
Cosgrove, Delos M
Duncan, Brian W
Gillinov, A. Marc
Greenberg, Roy K
Loop, Floyd D
Lytle, Bruce W
Mason, David P
Mavroudis, Constantine
McCurry, Kenneth R
Mihaljevic, Tomislav
Murthy, Sudish C
Navia, Jose L
Pettersson, Gosta B
Rice, Thomas W
Sabik, Joseph F, III
Smedira, Nicholas G
Svensson, Lars G
Ungerleider, Ross M
Van Heeckeren, Daniel W

OKLAHOMA

Oklahoma City
Elkins, Ronald C
Felton, Warren L, II
Fisher, R. Darryl
Zuhdi, M. Nazih

Tulsa
LeBeck, Martin B

OREGON

Ashland
Campbell, Daniel C, Jr

Days Creek
Miller, Arthur C

Florence
Turley, Kevin

Portland
Furnary, Anthony P
Handy, John R, Jr
Krause, Albert H
Lemmer, John H, Jr
Okies, J. Edward
Poppie, J. Karl
Starr, Albert

PENNSYLVANIA

Abington
Addonizio, V. Paul

Berwyn
Edie, Richard N
McKeown, John J, Jr

Camp Hill
Pennock, John L

Carlisle
DeMuth, William E, Jr

Dallas
Cimochowski, George E
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Glenolden
Gorman, Robert C

Hershey
Campbell, David B
Midgley, Frank M
Myers, John L
Pae, Walter E
Pierce, William S
Waldhausen, John A

Huntingdon Valley
Lemole, Gerald M

Johnstown
Kolff, Jacob

Lancaster
Bonchek, Lawrence I

Newtown Square
Jacobs, Marshall L

Philadelphia
Acker, Michael A
Bavaria, Joseph E
Bowles, L. Thompson
Bridges, Charles R
Cooper, Joel D
Diehl, James T
Edmunds, L. Henry, Jr
Friedberg, Joseph S
Gaynor, J. William
Gorman, Joseph H, III
Guerraty, Albert J
Hargrove, W. Clark, III
MacVaugh, Horace
Pochettino, Alberto
Samuels, Louis E
Spray, Thomas L
Wechsler, Andrew S
Whitman, Glenn J.R.
Woo, Y. Joseph

Pittsburgh
Christie, Neil
Hardesty, Robert L
Hattler, Brack G, Jr
Keenan, Robert J
Kormos, Robert L
Landreneau, Rodney J
Luketch, James D
Magovern, George J, Jr
Magovern, George J
Morell, Victor
Rams, James J
Toyoda, Yoshiya
Zenati, Marco A

Reading
Scott, Henry E

Sewickley
Clark, Richard E

Verona
Pontius, Robert G

Wynnewood
DiSesa, Verdi J
Goldman, Scott M

RHODE ISLAND

Providence
Moulton, Anthony L
Sellke, Frank W
Singh, Arun K

SOUTH CAROLINA

Charleston
Bradham, R. Randolph
Bradley, Scott M
Crawford, Fred A, Jr
Ikonomidis, John S
Kratz, John M
Reed, Carolyn E
Rubin, Joseph W
Sade, Robert M
Spinale, Francis G
Swenson, Orvar

Columbia
Almond, Carl H

Hilton Head Island
Humphrey, Edward W
SOUTH DAKOTA

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  Oury, James H

TENNESSEE

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  Pennington, D. Glenn

Jonesborough
  Bryant, Lester R

Knoxville
  Blake, Hu Al
  Brott, Walter H

Memphis
  Pate, James W
  Shochat, Stephen J
  Weiman, Darryl S

Nashville
  Alford, William, Jr
  Bender, Harvey W, Jr
  Byrne, John G
  Drinkwater, Davis C, Jr
  Gobbel, Walter G, Jr
  Nesbitt, Jonathan C
  Putnam, Joe B
  Randolp, Judson G
  Rankin, J. Scott
  Sawyers, John L
  Stoney, William S
  Thomas, Clarence S, Jr

TEXAS

Austin
  Tyson, Kenneth R.T.

Dallas
  Adam, Maurice
  DiMaio, J. Michael
  Estrera, Aaron S
  Forbess, Joseph
  Holland, Robert H
  Jessen, Michael E

Mack, Michael J
Magee, Mitchell J
Mandeloff, Eric N
Meyer, Dan M
Platt, Melvin R
Ring, W. Steves
Ursochel, Harold C, Jr

Dilley
  Hood, Richard H, Jr

Galveston
  Conti, Vincent R

Houston
  Cohn, William E
  Cooley, Denton A
  Coselli, Joseph S
  Dyke, Cornelius M
  Estrera, Anthony L
  Fraser, Charles D
  Frazier, O. Howard
  Gregoric, Igor D
  Hallman, Grady L
  Henly, Walter S
  Kaiser, Larry R
  Lawrie, Gerald M
  LeMaire, Scott A
 Letsou, George V
  Mattox, Kenneth L
  Mehran, Reza J
  Ott, David A
  Overstreet, John W
  Reardon, Michael J
  Reul, George J, Jr
  Rice, David C
  Roth, Jack A
  Safi, Hazim J
  Swisher, Stephen G
  Vaporciyan, Ara A
  Walker, William E
  Walsh, Garrett L

Lubbock
  Baldwin, John C
  Bricker, Donald L
  Feola, Mario
  Hood, R. Maurice

Montgomery
  Jones, James W
AMERICAN ASSOCIATION FOR THORACIC SURGERY

Plano
Edgeron, James R

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Temple
Smythe, William R
Zehr, Kenton J

UTAH

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Doty, Donald B

Park City
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Hawkins, John A
Jones, Kent W
Karwande, Shreekanth V
McGough, Edwin C
Nelson, Russell M

VERMONT

Burlington
Leavitt, Bruce J
Schmoker, Joseph D

Hartland
Marrin, Charles A.S.

Richford
Grondin, Claude M

VIRGINIA

Altavista
Pierucci, Louis, Jr

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Trachiotis, Gregory D

Charlottesville
Crosby, Ivan K
Dammann, John F
Daniel, Thomas M
Gott, Vincent L
Jones, David R
Kern, John A
Kron, Irving L
Nolan, Stanton P
Peeler, Benjamin B
Spotnitz, William D
Wellons, Harry A, Jr

Delaplane
Speir, Alan M

Falls Church
Ad, Niv
Burton, Nelson A
Lefrak, Edward A

Fredericksburg
Armitage, John M

Irvington
Muller, William H, Jr

McLean
Conrad, Peter W
Pecora, David V
Wallace, Robert B

Norfolk
Baker, Lenox D
Rich, Jeffrey B

Reston
Boyd, Thomas F

Richmond
Bosher, Lewis H, Jr

Springfield
Mills, Mitchell

WASHINGTON

Issaquah
Gentsch, Thomas O

Mercer Island
Li, Wei-i
Manhas, Dev R
Seattle
Aidea, Gabriel S
Allen, Margaret D
Cohen, Gordon A
Lupinetti, F. Mark
Mansfield, Peter B
Merendino, K. Alvin
Miller, Donald W, Jr
Mulligan, Michael S
Nelson, Ronald J
Sauvage, Lester R
Thomas, George I
Verrier, Edward D
Wood, Douglas E

Spokane
Berg, Ralph, Jr

Marshfield
Myers, William O

Milwaukee
Almassi, G. Hossein
Haasler, George B
Tector, Alfred J
Tweedell, James S

Snowmass Village
McEnany, M. Terry

WYOMING

Evanston
Kaunitz, Victor H

Shell
Scott, Meredith L

WEST VIRGINIA

Morgantown
Gustafson, Robert A

WISCONSIN

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Johnson, W. Dudley

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Chopra, Paramjeet S
Weigel, Tracey L
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CANADA

ALBERTA

Calgary
Bharadwaj, Baikunth
Miller, George E

Edmonton
Gelfand, Elliot T
Koshal, Arvind
Rebeyka, Ivan M
Ross, David B
Sterns, Laurence P

BRITISH COLUMBIA

Vancouver
Ashmore, Phillip G
Jamieson, W.R. Eric
Tyers, G. Frank O

Victoria
Stenstrom, John D

MANITOBA

Winnipeg
Barwinsky, Jaroslaw
Menkis, Alan H
Unruh, Helmut W

NOVA SCOTIA

Halifax
Hirsch, Gregory M
Johnston, Michael R

Kingsburg
Murphy, David A

ONTARIO

Almonte
Todd, Thomas R.J.

Collingwood
Heimbecker, Raymond

London
Guiraudon, Gerard M
McKenzie, F. Neil
Novick, Richard J

Mansfield
Pearson, F. Griffith

Oakville
Allen, Peter

Ottawa
Hendry, Paul J
Keon, Wilbert J
Mesana, Thierry G
Ruel, Marc
Sundaresan, R. Sudhir

Sebright
Trimble, Alan S

Toronto
Baird, Ronald J
Caldarone, Christopher A
Christakis, George T
Coles, John G
Darling, Gail E
David, Tirone E
De Perrot, Marc
Feindel, Christopher M
Fremes, Stephen Edward
Goldberg, Melvyn
Goldman, Bernard S
Keshavjee, Shaf
Kneally, Martin F
Rao, Vivek
Scully, Hugh E
90TH ANNUAL MEETING MAY 1–MAY 5, 2010
TORONTO, ON, CANADA

Trusler, George A
Van Arsdell, Glen
Waddell, Thomas K
Weisel, Richard D
Williams, William G
Yau, Terrence M

Waubaushene
Mickleborough, Lynda L

Quebec
Blundell, Peter E
Carrier, Michel
Chartrand, Claude C.C.
Chiu, Chu-Jeng (Ray)
Dobell, Anthony R.C.
Duranceau, Andre C.H.

MacLean, Lloyd D
Morin, Jean E
Mulder, David S
Pelletier, L. Conrad
Perrault, Louis P
Shum-Tim, Dominique
Tchervenkov, Christo I

Quebec
DesLauriers, Jean
Rosemere
Cossette, Robert

Ontario

Saskatchewan

Queensland
Main Beach
O’Brien, Mark F
South Brisbane
Karl, Tom R

Queensland

Queensland

South Australia
Nurioopta
Aberg, Torkel H

Victoria

Victoria

Austria

Innsbruck
Laufer, Guenther
Salzburg
Unger, Felix H
Thumersbach
Bruecke, Peter E
Vienna
Klepetko, Walter
Wolner, Ernst

Argentina

Buenos Aires
Favaloro, Roberto R
Kreutzer, Christian
Kreutzer, Guillermo O
Schlichter, Andres J

Australia

Victoria

Argentina

Argentina

Argentina
AMERICAN ASSOCIATION FOR THORACIC SURGERY

BARBADOS

St. James
Sintek, Colleen F

BELGIUM

Aalst
Casselman, Filip P
Vanermen, Hugo K.I.

Antwerp
Van Schil, Paul E

Genk
Dion, Robert A

Leuven
De Leyn, Paul
Flameng, Willem J
Lerut, Antoon E.M.R.
Sergeant, Paul T
Van Raemdonck, Dirk E.M.

Linden
Daenen, Willem J

BRAZIL

Rio de Janeiro
Meier, Milton A

Sao Paulo
Da Silva, Jose Pedro
Jatene, Adilb D
Oliveira, Sergio A

Sao Jose do Rio Preto
Braile, Domingo M

CHINA

Beijing
Hu, Shengshou
Wu, Qingyu

Shanghai
Liu, Jinfen

FINLAND

Grankulla
Mattila, Severi P

Helsinki
Harjula, Ari L. J

FRANCE

Bordeaux-Pessac
Roques, Xavier F

Bordeaux
Fontan, Francis M

Creteil
Loisance, Daniel

Le Plessis Robinson
Dartevelle, Philippe G
Planche, Claude
Serraf, Alain

Lyon
Jegaden, Olivier L
Obadia, Jean F

Marseille
Metras, Dominique R
Thomas, Pascal A

Montpellier
Thevenet, Andre A

Paris
Binet, Jean-Paul
Blondeau, Philip
Cabrol, Christian E.A.
Carpentier, Alain F
Chachques, Juan C
Chauvaud, Sylvain M
Grunenwald, Dominique H
Khonsari, Siavosh
Menasche, Philippe
Piwnica, Armand H

Pessac
Baudet, Eugene M
Couraud, Louis

Suresnes
Chapelier, Alain R
90TH ANNUAL MEETING MAY 1–MAY 5, 2010
TORONTO, ON, CANADA

GERMANY

Aachen
Messmer, Bruno J

Bad Oeynhausen
Koerfer, Reiner

Berlin
Alexi-Meskishvili, Vladimir
Hetzer, Roland

Essen
Jakob, Heinz G

Freiburg
Beyersdorf, Friedhelm

Hamburg
Reichenspurner, Hermann

Hannover
Haverich, Axel

Homburg/Saar
Schafers, Hans-Joachim

Leipziga
Borger, Michael A
Mohr, Friedrich W

Loiching
Sebening, Fritz

Lubeck
Sievers, Hans-Henric

Munich
Borst, Hans G
Daebritz, Sabine H
Lange, Rudiger S
Malec, Edward J

Neuss
Biroks, Wolfgang H

GREECE

Athens
Palatianos, George M
Sarris, George E

GUATEMALA

Guatemala City
Castaneda, Aldo R
Espada, J. Rafael
Herrera-Llerandi, Rodolfo

HONG KONG

Shatin, NT
He, Guo-Wei
Wan, Song
Yim, Anthony P

INDIA

Delhi
Sampathkumar, Arkalgud

Mogappair, Chennai
Cherian, K. Mammen

ISRAEL

Florence
Macchiariini, Paolo

Jerusalem
Shapira, Oz M

ITALY

Ancona
Brunelli, Alessandro

Bergamo
Parentzan, Lucio

Milan
Alfieri, Ottavio R
Peracchia, Alberto
Roviaio, Giancarlo
<table>
<thead>
<tr>
<th>Location</th>
<th>Members</th>
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</thead>
<tbody>
<tr>
<td><strong>Naples</strong></td>
<td>Cotrufo, Maurizio</td>
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<td>Bortolotti, Uberto</td>
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<td>Gerosa, Gino</td>
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<td>Stellin, Giovanni</td>
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<td><strong>Pisa</strong></td>
<td>Melfi, Franca M.A.</td>
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<td><strong>Rome</strong></td>
<td>De Paulis, Ruggero</td>
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<td>Di Donato, Roberto</td>
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<td>Rendina, Erno Angelo</td>
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<td>Venuta, Federico</td>
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<td><strong>San Donato Milanese</strong></td>
<td>Menicanti, Lorenzo A</td>
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<td><strong>Udine</strong></td>
<td>Livi, Ugolino</td>
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<td><strong>JAPAN</strong></td>
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<td><strong>Fukuoka</strong></td>
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<td><strong>Hayama</strong></td>
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<td>Iwa, Takashi</td>
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<td><strong>Kitakyushushi</strong></td>
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<td>Abe, Tomio</td>
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<td>Shimpo, Hideto</td>
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<td><strong>Seoul</strong></td>
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TSFRE was established in 1992 by the four leading American thoracic surgical associations, AATS, STS, WTSA and STSA, to respond to the decrease in research funding from the federal government and institutions for education and research in thoracic surgery—a challenge that continues today.

The Foundation has become a pivotal force for the growth and vitality of our specialty and its role is increasing, particularly in the areas of research, academic career development and postgraduate education. Perhaps most importantly, the Foundation has chosen to play a leading role in changing the current training paradigm for thoracic surgeons by becoming a founding organization of the Joint Council on Thoracic Surgery Education (JCTSE). Along with the American Association for Thoracic Surgery (AATS), the American Board of Thoracic Surgery (ABTS) and the Society of Thoracic Surgeons (STS), TSFRE has committed its resources to support and empower the JCTSE to overhaul the current thoracic surgery training program and coordinate all thoracic surgery education in the United States.

The Thoracic Surgery Foundation for Research and Education is your foundation and the lifeblood of your specialty. It depends on you to fund the research to support our fellow surgeons and our ability to embrace new technology and learn its application. The efforts of our supporters—through donations or networking—will impact the future of cardiothoracic surgery and the welfare of our patients.

For more information about your annual gift visit the TSFRE Booth at #500, visit the Foundation’s website at www.TSFRE.org or contact the Thoracic Surgery Foundation for Research and Education at 900 Cummings Center, Suite 221-U, Beverly, Massachusetts, 01915.
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2010 TSFRE RESEARCH AWARD Recipients

TSFRE RESEARCH FELLOWSHIPS provide support of up to $35,000 a year for up to 2 years for surgical residents who have not yet completed cardiothoracic surgical training.

Nicholas D. Andersen, MD, Duke University Medical Center
“Calcium Signaling Regulates Cardiomyocyte Growth in Hypoplastic Left Heart Syndrome”

David N. Anderson, MD, Memorial Sloan-Kettering Cancer Center
“SCCRO (DCUN1D1) Is Essential for Cellular Transformation”

Alejandro Bribriesco, BS, MD, Washington University
“Role of Non-Alloimmune Stimuli in Airway Epithelial Cell Differentiation after Lung Transplantation”

William Hiesinger, MD, University of Pennsylvania
“Myocardial Angiogenic Tissue Engineering via Ex-Vivo Modified Stem Cell Matrix”

TSFRE RESEARCH GRANTS provide operational support of original research efforts by cardiothoracic surgeons who have completed their formal training, and who are seeking initial support and recognition for their research program. Awards of up to $40,000 a year for up to 2 years are made each year to support the work of an early-career cardiothoracic surgeon (within 5 years of first faculty appointment).

Mark Onaitis, MD, Duke University
“The Mechanism of Sox2 in Lung Cancer Development”

Thomas B. Reece, MD, University of Colorado
“The Role of Specific Adenosine Receptor Activation in Ischemic Preconditioning of the Spinal Cord”

Brendon M. Stiles, MD, Weill Medical College, Cornell University
“Disseminated Tumor Cells in the Bone Marrow of Patients with Surgically Resectable Non-Small Cell Lung Cancer: Comparative Genomic Analysis to Matched Primary Tumors”
NINA STARR BRAUNWALD CAREER DEVELOPMENT AWARD provides a biennial award of $115,000 for two years to support the research career development of a woman cardiac surgeon who holds a full-time faculty appointment and who is within 10 years of completion of thoracic surgery residency.

Jennifer C. Hirsch, MD, University of Michigan
“Development of a Congenital Heart Assessment of Sensory and Motor Status (CHASMS) Instrument for Infants Following Cardiac Surgery”
2010 Education Award Recipients

Simulation in Thoracic Surgery Education Grants
Provides grants to support the demonstration study for the application of simulation in thoracic surgery education.

Leora B. Balsam, MD, New York University
“Simulator for Conventional and Limited Access Mitral Valve Surgery”

Raphael Bueno, MD, Brigham and Women's Hospital

Joanna Chikwe, MD, Mount Sinai Medical Center
“High Fidelity Simulation in Preparing Medical Students for Integrated Cardiothoracic Residency Training”

Yolonda L. Colson, MD, PhD, Brigham and Women's Hospital
“Exportable Crisis Management Assessment Curriculum (ECMAC)”

Richard Feins, MD, University of North Carolina
“Multicenter Cardiac Simulator Beta Testing”

M. Blair Marshall, MD, Georgetown University Hospital
“Development of Task Specific Cardiothoracic Simulation Models for Independent Study and Skill Acquisition”

Shari L. Meyerson, MD, University of Arizona
“Validation of a Thoracoscopic Lobectomy Simulator”
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## Future Meetings

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<th>Year</th>
<th>Dates</th>
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<tr>
<td>2011</td>
<td>May 7–11</td>
<td>Pennsylvania Convention Center, Philadelphia, PA</td>
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<td>2012</td>
<td>April 28–May 2</td>
<td>Moscone West Convention Center, San Francisco, CA</td>
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<td>2013</td>
<td>May 4–8</td>
<td>Minneapolis Convention Center, Minneapolis, MN</td>
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<td>2014</td>
<td>April 26–30</td>
<td>Metro Toronto Convention Centre, Toronto, ON Canada</td>
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<td>2015</td>
<td>April 25–29</td>
<td>Washington State Convention and Trade Center, Seattle, WA</td>
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### Schedule at a Glance

All scientific sessions and exhibits will take place at the Metro Toronto Convention Centre

#### Friday, April 30, 2010

- **1:00 p.m. – 5:00 p.m.** Registration Open

#### Saturday, May 1, 2010  
Skills Courses and Symposia

- **6:30 a.m. – 5:00 p.m.** Registration Open
- **8:00 a.m. – 12:00 p.m.** Adult Cardiac Skills
- **8:00 a.m. – 12:00 p.m.** General Thoracic Skills
- **8:00 a.m. – 12:00 p.m.** Congenital Skills
- **1:00 p.m. – 5:00 p.m.** Developing the Academic Surgeon Symposium
- **1:30 p.m. – 4:20 p.m.** Professionalism and the Cardiothoracic Specialty
- **2:00 p.m. – 4:30 p.m.** Robotic Cardiothoracic Surgery Symposium

#### Sunday, May 2, 2010  
AATS/STS Postgraduate Symposium

- **6:30 a.m. – 6:00 p.m.** Registration Open
- **8:00 a.m. – 5:00 p.m.** Adult Cardiac Surgery Symposium
- **8:00 a.m. – 5:00 p.m.** General Thoracic Surgery Symposium
- **7:55 a.m. – 5:00 p.m.** Congenital Heart Disease Symposium
- **8:00 a.m. – 5:00 p.m.** Cardiothoracic Critical Care Symposium
- **3:00 p.m. – 5:00 p.m.** 13th Annual C. Walton Lillehei Resident Forum
- **5:00 p.m. – 7:00 p.m.** Welcome Reception – Exhibit Hall A & B
- **5:00 p.m. – 7:00 p.m.** Operating Rooms: Hybrid Technologies©
- **7:00 p.m.** Various Satellite Post-Activity Symposia*

#### Monday, May 3, 2010  
Annual Meeting

- **6:30 a.m. – 5:00 p.m.** Registration Open
- **9:00 a.m. – 4:30 p.m.** Exhibits Open – Exhibit Hall A & B
- **9:00 a.m. – 4:30 p.m.** Operating Rooms: Hybrid Technologies©
- **7:30 a.m. – 7:45 a.m.** Business Session (AATS Members Only)
- **7:45 a.m. – 12:15 p.m.** Plenary Scientific Session
  - Basic Science Lecture — Susan E. Mackinnon, MD, Washington University School of Medicine
  - Presidential Address — G. Alec Patterson, MD, Washington University School of Medicine
- **12:15 p.m. – 2:00 p.m.** Lunch – Exhibit Hall A & B
- **12:15 p.m. – 2:00 p.m.** Cardiothoracic Residents’ Luncheon
- **2:00 p.m. – 5:00 p.m.** Simultaneous Scientific Sessions
- **2:00 p.m. – 5:00 p.m.** Educational Program: Building a Hybrid OR of the Future©
- **7:00 p.m.** Various Satellite Post-Activity Symposia*

#### Tuesday, May 4, 2010  
Annual Meeting

- **6:30 a.m. – 9:00 a.m.** Registration Open
- **9:00 a.m. – 4:00 p.m.** Exhibits Open – Exhibit Hall A & B
- **9:00 a.m. – 4:00 p.m.** Operating Rooms: Hybrid Technologies©
- **7:00 a.m. – 8:45 a.m.** Cardiac Surgery Forum
- **7:00 a.m. – 8:45 a.m.** General Thoracic Surgery Forum
- **8:00 a.m. – 12:00 p.m.** Educational Program: Building a Hybrid OR of the Future©
- **8:45 a.m. – 12:30 p.m.** Plenary Scientific Session
  - Honored Speaker Lecture — David Naylor, MD, University of Toronto
- **12:30 p.m. – 2:00 p.m.** Lunch – Exhibit Hall A & B
- **2:00 p.m. – 5:00 p.m.** Simultaneous Scientific Sessions
- **5:00 p.m. – 5:45 p.m.** Executive Session (AATS Members Only)
- **7:00 p.m. – 10:00 p.m.** Attendee Reception – Hockey Hall of Fame (ticketed event)

#### Wednesday, May 5, 2010  
Annual Meeting

- **6:30 a.m. – 12:00 p.m.** Registration Open
- **7:00 a.m. – 8:45 a.m.** Emerging Technologies and Techniques Forum
- **9:00 a.m. – 10:00 a.m.** Controversies in Cardiothoracic Surgery Plenary Session
- **10:00 a.m. – 12:00 p.m.** Simultaneous Scientific Sessions
- **1:00 p.m. – 5:00 p.m.** Endobronchial Ultrasound (EBUS) Training Course (Toronto Medical Discovery Tower)

*Industry-sponsored satellite programs are not part of the AATS Annual Meeting*