

THE SOCIETY OF BLACK ACADEMIC SURGEONS



AND THE

UNIVERSITY OF CINCINNATI

DEPARTMENT OF SURGERY

PRESENT THE

SIXTEENTH ANNUAL SCIENTIFIC SESSION

APRIL 6-8, 2006

AT THE

HILTON CINCINNATI NETHERLAND PLAZA

CINCINNATI, OH



The 16th Annual Scientific Meeting of the Society of Black Academic Surgeons is being held at the Hilton Cincinnati Netherland Plaza in Cincinnati, Ohio. The University of Cincinnati Department of Surgery is the hosting institution.

The 2006 annual meeting will feature substantial basic science and clinical research presentations by a distinguished panel of pre-eminent surgeons, faculty, surgical residents and fellows.

Purpose or Overview:

The purpose of this program is to disseminate knowledge of recent advances in basic science and clinical research in surgery. This forum is designed to efficiently present and discuss new information relevant to the treatment of surgical patients.

Learning Objectives:

1. Explain current concepts of cardiothoracic and vascular surgery, trauma management, oncology, and clinical trials outcomes.
2. Discuss the usefulness of clinical pathways in the development of evidence based patient care protocols.
3. Explain specific risk factors and diagnostic interventions influencing the management of trauma patients.
4. Discuss technical approaches to the surgical treatment of cardiovascular disorders.
5. Explain the diagnosis and management of venous disorders.
6. Explain the emerging technology in the treatment of solid tumors.
7. Describe the changing trends in cardiac surgery.

Continuing Education Credit:

The University of Cincinnati College of Medicine designates this educational activity for a maximum of 12 AMA PRA Category 1 Credit(s)TM. Physicians should only claim credits commensurate with the extent of their participation in the activity.

The University of Cincinnati College of Medicine is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

The University of Cincinnati College of Medicine is committed to resolving all conflicts of interest issues which may arise as a result of prospective faculty members' significant relationships with drug or device manufacturer(s). The University of Cincinnati College of Medicine mandate is to retain only those speakers with financial interest that can be reconciled with the goals and educational integrity of the program.

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John H. Blue Professor and Section Chief of Gastrointestinal Surgery
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Trauma Services
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Associate Professor of Surgery, Department of Surgery
& Institute of Molecular Pharmacology and Biophysics
University of Cincinnati

Society Historian

Frederick D. Cason, Jr, MD

Medical University of Ohio
Toledo, OH

Executive Council

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Clive O. Callender, MD
Haile T. Debas, MD

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Danny O. Jacobs, MD

Lynt B. Johnson, MD

Anthony A. Stallion, MD

Jeffrey S. Upperman, MD

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Kenneth Davis, Jr, MD – Chair

Karyn L. Butler, MD – Co-Chair

Jeffrey B. Matthews, MD

Department of Surgery

University of Cincinnati

GUEST LECTURERS

Jeffrey B. Matthews, MD

Christian R. Holmes Professor
Chairman, Department of Surgery
University of Cincinnati

Kirby I. Bland, MD

Professor and Chairman
Department of Surgery
University of Alabama at Birmingham

Michael S. Saag, MD

Professor of Medicine
Director, UAB Center for AIDS Research
Department of Medicine
University of Alabama at Birmingham

2006 HONORARY FELLOWS

Jeffrey B. Matthews, MD

Christian R. Holmes Professor
Chairman, Department of Surgery
University of Cincinnati

Carlos A. Pellegrini, MD

Henry N. Harkins Professor and Chairman
Department of Surgery
University of Washington

HISTORY OF THE SOCIETY OF BLACK ACADEMIC SURGEONS

The Society of Black Academic Surgeons (SBAS) was founded in 1989. Its goal is to stimulate academic excellence among its members by providing a forum of scholarship in collaboration with the leading Departments of Surgery in the U.S. It encourages and supports professional development of black surgical residents and attempts to recruit the best and brightest medical students into a career in surgery.

The annual meetings of SBAS, attended by members as well as numerous residents and students, provide outstanding programs in both the science and practice of surgery. The first Annual Meeting was hosted by Dr. David Sabiston at Duke University. Annual meetings since then have been hosted by Departments of Surgery throughout the U.S., including Harvard (1991), University of California at Davis (1993), University of Texas Medical Branch at Galveston (1994), the University of North Carolina at Chapel Hill (1995), the University of Colorado at Denver (1996), SUNY Buffalo (1997), Howard University College of Medicine (1998, 2004), the University of Louisville (1999), Charles R. Drew University of Medicine and Science (2000), Harvard University (2001), Morehouse School of Medicine (2002), the University of Alabama at Birmingham (2003), and the University of Pittsburgh (2005).

SBAS is governed by an Executive Committee and has more than 200 members throughout the United States. Membership is not restricted by race; the criteria for membership requires that the prospective member be a “reputable surgeon or surgical investigator who occupies a faculty position in a university department of surgery or free-standing surgical residency program.” In addition to its Annual Meeting, a website (www.SBAS.net) has been established to improve communication with its constituency and persons interested in the organization. The journal *American Journal of Surgery* is the official publication of SBAS.

PAST PRESIDENTS OF THE SOCIETY OF BLACK ACADEMIC SURGEONS

1989-1991: Arthur Fleming, MD
1991-1993: Onye E. Akwari, MD
1993-1995: Eddie L. Hoover, MD
1995-1997: Claude H. Organ, Jr, MD
1997-1998: LaSalle D. Leffall, Jr, MD
1998-1999: Haile T. Debas, MD
1999-2001: L. D. Britt, MD, MPH
2001-2003: Clive O. Callender, MD
2003-2004: Edward Cornwell, III, MD
2004-2005: Robert L. McCauley, MD



SCIENTIFIC SESSIONS



SOCIETY OF BLACK ACADEMIC SURGEONS
SIXTEENTH ANNUAL
SCIENTIFIC SESSION

THURSDAY

APRIL 6, 2006

- 3:30-7:00 p.m. **Registration**, Hilton Cincinnati Netherland Plaza, Lobby
- 5:00-6:30 p.m. **Reception for Women Surgeons**, Hilton Cincinnati Netherland Plaza, Suite 740
- 6:30-8:30 p.m. **Welcome Reception**, Hilton Cincinnati Netherland Plaza, Mezzanine

FRIDAY

APRIL 7, 2006

- 6:00-7:15 a.m. **Registration and Breakfast Buffet**, Hilton Cincinnati Netherland Plaza, Continental Ballroom (Mezzanine Level)
- 7:15 a.m. Board Buses, Hilton Cincinnati Netherland Plaza, Fifth Street Entrance (revolving door)
- 7:30 a.m. Buses Depart for University of Cincinnati
- 8:00-11:15 a.m. **Local Program**, University of Cincinnati Department of Surgery, Surgical Amphitheater
- 8:00-8:15 a.m. **Welcome**

SCIENTIFIC SESSIONS

FRIDAY (CONTINUED)

APRIL 7, 2006

- 8:15-10:00 a.m. **Local Presentations**
(Kenneth Davis, Jr, MD, Moderator)
- 8:15 a.m. Timothy M. Crombleholme, MD
"Fetal Surgery: Current Status"
- 8:30 a.m. Karyn L. Butler, MD
"Support for the Sick at Heart: Cardioprotection in a Clinically Relevant Model of Heart Disease"
- 8:45 a.m. Calvin A. Selwyn, Jr, MD
"Endoluminal Surgery: Current and Future Techniques"
- 9:00 a.m. Karl S. Matlin, PhD
"Surgical Research"
- 9:15 a.m. Amy B. Reed, MD
"Vascular Surgery Training in the 21st Century"
- 9:30 a.m. Randall K. Wolf, MD
"The Future of Surgery"
- 9:45 a.m. Syed A. Ahmad, MD
"The UC Experience with Total Pancreatectomy and Autologous Islet Cell Transplantation for Chronic Pancreatitis"
- 10:00-10:15 a.m. Break
- 10:15-11:15 a.m. **Asa Yancey Lecture: "Surgical Biodiversity"**
Jeffrey B. Matthews, MD
- 11:15 a.m. Board Buses and Depart for Hilton Cincinnati Netherland Plaza
- 12:00-1:00 p.m. Luncheon, Hilton Cincinnati Netherland Plaza (Rosewood, Fourth Floor)

SCIENTIFIC SESSIONS

FRIDAY (CONTINUED)

APRIL 7, 2006

Scientific Sessions, Hilton Cincinnati Netherland Plaza,
Continental Ballroom

- 1:00-3:00 p.m. **Scientific Session I - Clinical Trials/
Education**
- 3:00-3:45 p.m. **State of the Art Lecture:** *"The Integration of
Mentorship into Translational Surgical
Research: Student, Resident, and Faculty
Implications"*
Kirby I. Bland, MD
- 3:45-4:00 p.m. Break
- 4:00-5:00 p.m. **Scientific Session II - Oncology**
- 6:30 p.m. Board Buses, Hilton Cincinnati Netherland
Plaza, Fifth Street Entrance (revolving door)
- 6:45 p.m. Buses Depart for National Underground
Railroad Freedom Center
- 7:00-9:00 p.m. **Cocktail/Dinner Reception**, National
Underground Railroad Freedom Center,
50 East Freedom Way, Cincinnati
- Guest of Honor:** Mark Mallory, Mayor
of the City of Cincinnati
- Welcome:** Spencer Crew, PhD, President,
National Underground Railroad Freedom
Center
- 9:00 p.m. Buses Depart for Hilton Cincinnati
Netherland Plaza

SCIENTIFIC SESSIONS

SATURDAY

APRIL 8, 2006

6:30-8:00 a.m. **Registration and Breakfast Buffet**,
Hilton Cincinnati Netherland Plaza,
Continental Ballroom (Mezzanine Level)

Scientific Sessions, Hilton Cincinnati Netherland Plaza

- 8:00-10:00 a.m. **Scientific Session III - Trauma/
Critical Care**
- 10:00-10:15 a.m. Break
- 10:15-11:15 a.m. Business Meeting (members only)
- 11:15 a.m.-12:00 p.m. **State of the Art Lecture:** *"Principles of
HIV Therapy"*
Michael S. Saag, MD
- 12:00-1:00 p.m. Lunch (Rosewood, Fourth Floor)
- 1:00-2:45 p.m. **Scientific Session IV - Cardiothoracic/
Vascular**
- 2:45-3:00 p.m. Break
- 3:00-4:15 p.m. **Panel Discussion - "Critical Skills for
Academic Success"** (Karyn L. Butler, MD,
Moderator)
- 4:15-5:00 p.m. **Presidential Address:** Selwyn M. Vickers,
MD
- 6:30-7:30 p.m. **Presidential Reception**, Hilton Cincinnati
Netherland Plaza, Fourth Floor Foyer
- 7:30-10:00 p.m. **Presidential Dinner** (Black Tie), Hilton
Cincinnati Netherland Plaza, Pavilion
Ballroom, Fourth Floor
Guest Speaker: Oscar Robertson,
Cincinnati businessman & basketball icon



SOCIETY OF BLACK ACADEMIC SURGEONS

SIXTEENTH ANNUAL SCIENTIFIC SESSION

SESSIONS AT A GLANCE

SESSION I

Clinical Trials/Education

FRIDAY, APRIL 7, 2006

1:00 P.M.-3:00 P.M.

**Moderators: Paul R. Cunningham, MD,
and Orlando Kirton, MD**

1. MULTIVARIATE ANALYSIS OF RISK FACTORS FOR ALLOGRAFT LOSS IN BLACK & WHITE RECIPIENTS OF RENAL ALLOGRAFTS
Joseph Keith Melancon, MD, Johns Hopkins Hospital
2. PATIENT LEVEL PREDICTORS OF TIMELY PROPHYLACTIC ANTIBIOTIC ADMINISTRATION
Stephen H. Gray, MD, University of Alabama at Birmingham
3. FACTORS AFFECTING INNOVATION IN PEDIATRIC SURGERY: HOSPITAL TYPE AND APPENDECTOMIES
Cedric V. Pritchett, BS, PA-C, Ohio State University
4. A WEB-BASED PROGRAM FOR THE ELECTRONIC SUBMISSION OF MORBIDITY AND MORTALITY
Selwyn O. Rogers, Jr, MD, MPH, Brigham & Women's Hospital
5. MYOCELLULAR CREATINE TRANSPORTER FUNCTION IS MODULATED BY CHANGES IN ITS PHOSPHORYLATION STATUS
Marcus D. Darrabie, BS, Duke University Medical Center
6. TGF-BETA SIGNALING MAY PLAY A PROTECTIVE ROLE IN PREVENTING LIVER FROM INSULIN INDUCED STEATOSIS
Krit Kitisin, MD, Georgetown University Hospital
7. BEARD SYCOSIS AND DISFIGURING KELOID SCARRING OF THE FACE, NECK, AND SCALP: A 15-YEAR EXPERIENCE
Fritz Jean-Pierre, MD, Rosalind Franklin University of Medicine and Science/The Chicago Medical School
8. IN VIVO MODEL OF ADIPOGENESIS USING A HYDROGEL BIOMATERIAL
Lamont Cathey, MD, Carolinas Medical Center

SOCIETY OF BLACK ACADEMIC SURGEONS

SIXTEENTH ANNUAL SCIENTIFIC SESSION

SESSIONS AT A GLANCE

SESSION II

Oncology

FRIDAY, APRIL 7, 2006

4:00 P.M.-5:00 P.M.

**Moderators: Cassann N. Blake, MD, MPH,
and Wade G. Douglas, MD**

9. DISRUPTION OF TGF- β SIGNALING AND ITS RESTORATION MODULATED HEPATOCELLULAR CARCINOGENESIS IN HEPATOCELLULAR CANCER CELL LINES AND ELF+/- MUTANT TISSUES
Krit Kitisin, MD, Georgetown University Hospital
10. PANCREATIC STELLATE CELLS PROMOTE MALIGNANT POTENTIAL IN PANCREATIC CANCER
Keith D. Amos, MD, University of Texas MD Anderson Cancer Center
11. ESOPHAGEAL ADENOCARCINOMA IN AFRICAN-AMERICANS: RARE AND DEADLY
Craig M. Hooker, MPH, Johns Hopkins Medical Institutions
12. EARLY-AGE-ONSET FAMILIAL PANCREATIC ADENOCARCINOMA MAY IMPACT LONG-TERM SURVIVAL
Ted A. James, MD, University of Vermont





SOCIETY OF BLACK ACADEMIC SURGEONS

SIXTEENTH ANNUAL SCIENTIFIC SESSION

SESSIONS AT A GLANCE

SESSION III

Trauma/Critical Care

SATURDAY, APRIL 8, 2006

8:00 A.M.-10:00 A.M.

**Moderators: Robert A. Cherry, MD,
and Kimberly Joseph, MD**

13. SCREENING FOR BLUNT THORACIC AORTIC INJURIES: ARE CHEST X-RAYS ADEQUATE?
A. Peter Ekeh, MD, Wright State University
14. A COMPREHENSIVE INJURY RISK ASSESSMENT SCREENING TOOL FOR SENIOR POPULATIONS: PRELIMINARY FINDINGS FROM A MINORITY COMMUNITY
Lodze Quitel, MD, Harlem Hospital
15. ANASTOMOTIC STAPLE LINES: DOES REINFORCEMENT MATTER?
Rory J. Howard, MD, SUNY Upstate Medical University
16. APO E IS ASSOCIATED WITH INCREASED MORTALITY IN SEPTIC RATS
Michael T. Schell, MD, University of California, San Francisco
17. MURINE BURN WOUND INFLAMMATION THAT INCREASES BURN DEPTH MAY BE CAUSED BY LOCAL REPERFUSION INJURY
Freeman Suber, MD, Brigham and Women's Hospital / Harvard Medical School
18. HOSPITAL-BASED VIOLENCE INTERVENTION PROGRAMS WORK
Carnell Cooper, MD, University of Maryland
19. EARLY ENTERAL NUTRITION IMPROVES GUT EDEMA-ASSOCIATED ILEUS
Stacey D. Moore-Olufemi, MD, University of Texas-Houston
20. CYCLOSPORIN A INHIBITS HEMORRHAGIC SHOCK-INDUCED HYPERPERMEABILITY & MITOCHONDRIAL TRANSITION PORES
Georgia Holder-Haynes, MD, Texas A&M University

SOCIETY OF BLACK ACADEMIC SURGEONS

SIXTEENTH ANNUAL SCIENTIFIC SESSION

SESSIONS AT A GLANCE

SESSION IV

Cardiothoracic/Vascular

SATURDAY, APRIL 8, 2006

1:00 P.M.-2:45 P.M.

**Moderators: Eddie L. Hoover, MD,
and Girma Tefera, MD**

21. AN EVALUATION OF OUTCOMES AFTER CAROTID ENDARTERECTOMY WITH EXTRA- AND INTRA-CRANIAL TANDEM LESIONS
Rory J. Howard, MD, SUNY Upstate Medical University
22. ISCHEMIC PRECONDITIONING IN MYOCARDIAL HYPERTROPHY INDUCES A DISTINCT PATTERN OF JAK-STAT ACTIVATION
Karyn L. Butler, MD, University of Cincinnati
23. THE ROLE OF TOLL-LIKE RECEPTORS IN THORACIC AORTIC ISCHEMIA REPERFUSION
John E. Jones, MD, Massachusetts General Hospital
24. RACIAL DISPARITY IN PATIENT PRESENTATION FOR CAROTID ENDARTERECTOMY
David C. Chang, PhD, MPH, MBA, Johns Hopkins University
25. 360-DEGREE FEEDBACK SURVEY TO ASSESS FACULTY COMPETENCY IN A CARDIOTHORACIC SURGERY PRACTICE
Robert S. D. Higgins, MD, MSHA, Rush University
26. BRACHIAL AND INFRA-BRACHIAL ARTERIAL RECONSTRUCTION FOR CHRONIC ISCHEMIA
Kakra Hughes, MD, Arizona Heart Institute and Hospital
27. EXTRACORPOREAL PHOTOPHERESIS CAUSES DELETION OF CARDIAC ALLOGRAFT SPECIFIC T CELLS IN VIVO
Christie Gooden, MD, University of Alabama at Birmingham



ASA YANCEY LECTURE

Jeffrey B. Matthews, MD

Christian R. Holmes Professor
Chairman, Department of Surgery
University of Cincinnati
"Surgical Biodiversity"

STATE OF THE ART LECTURES

Kirby I. Bland, MD

Professor and Chairman
Department of Surgery
University of Alabama at Birmingham
*"The Integration of Mentorship into Translational Surgical
Research: Student, Resident, and Faculty Implications"*

Michael S. Saag, MD

Professor of Medicine
Director, UAB Center for AIDS Research
University of Alabama at Birmingham
"Principles of HIV Therapy"

DR. CLAUDE H. ORGAN, JR. RESIDENT AWARD WINNERS

- 2003 **Richard E. Redlinger, Jr, BS**
Children's Hospital of Pittsburgh
Pittsburgh, PA
- Donn H. Spight, MD**
University of Cincinnati
Cincinnati, OH
- 2004 **Zara R. Cooper, MD, MSc**
Brigham and Women's Hospital
Boston, MA
- 2005 **Sonya Walker, MD**
University of Pittsburgh
Pittsburgh, PA

PANEL DISCUSSION

"Critical Skills for Academic Success"

Karyn L. Butler, MD, Moderator

Associate Professor of Surgery
Department of Surgery
Institute of Molecular Pharmacology and Biophysics
University of Cincinnati
Cincinnati, OH

L.D. Britt, MD, MPH

Brickhouse Professor of Surgery
Chairman, Department of General Surgery
Eastern Virginia Medical School
Norfolk, VA

Edward E. Cornwell, III, MD

Director, Adult Trauma Service
The Johns Hopkins Hospital
Baltimore, MD

Jeffrey B. Matthews, MD

Christian R. Holmes Professor
Chairman, Department of Surgery
University of Cincinnati
Cincinnati, OH

Toni Robinson-Smith, MD

Assistant Professor
Department of Pathology & Laboratory Medicine
University of Cincinnati
Cincinnati, OH

SESSION MODERATORS

Cassann N. Blake, MD, MPH

Assistant Professor of Surgery
Karmanos Cancer Institute
Wayne State University
Detroit, MI

Robert A. Cherry, MD

Chief, Section of Trauma and Critical Care
Penn State Milton S. Hershey Medical Center
Hershey, PA

Paul R. Cunningham, MD

Professor and Chair of Surgery
State University of New York, Upstate Medical University
Syracuse, NY

Wade G. Douglas, MD

Assistant Professor of Surgery
Marshall University Joan C. Edwards School of Medicine
Edwards Comprehensive Cancer Center
Huntington, WV

Eddie L. Hoover, MD

Department of Surgery
State University of New York at Buffalo
Buffalo Veterans Affairs Medical Center
Buffalo, NY

Kimberly Joseph, MD

Director, Trauma Intensive Care Unit
Cook County Hospital
Chicago, IL

Orlando C. Kirton, MD

Chief of Surgery - Hartford Hospital
Professor of Surgery, University of Connecticut
Hartford, CT

Girma Tefera, MD

Assistant Professor of Surgery
Section of Vascular Surgery
University of Wisconsin - Madison
Madison, WI

LOCAL PROGRAM LECTURERS

Syed A. Ahmad, MD

Assistant Professor of Surgery
Division of Surgical Oncology
University of Cincinnati

Karyn L. Butler, MD

Associate Professor of Surgery
Department of Surgery
Institute of Molecular Pharmacology and Biophysics
University of Cincinnati

Timothy M. Crombleholme, MD

Professor of Surgery, Obstetrics and Gynecology,
Molecular and Developmental Biology
Director, Fetal Care Center of Cincinnati
Director, Center for Molecular Fetal Therapy
Cincinnati Children's Hospital Medical Center
University of Cincinnati

Karl S. Matlin, PhD

Professor of Surgery
Director, Division of Research
Director of the Epithelial Pathobiology Research Group
Department of Surgery
University of Cincinnati

Amy B. Reed, MD

Assistant Professor of Surgery
Director, Vascular Surgery Fellowship Program
Division of Vascular Surgery
University of Cincinnati

Calvin A Selwyn, Jr, MD

Assistant Professor of Surgery
Division of Gastrointestinal and Endocrine Surgery
University of Cincinnati

Randall K. Wolf, MD

Professor of Surgery and Biomedical Engineering
Director, Center for Surgical Innovation
Ethicon-Endosurgery Chair for Innovation in Surgery
University of Cincinnati



ABSTRACTS

1

MULTIVARIATE ANALYSIS OF RISK FACTORS FOR ALLOGRAFT LOSS IN BLACK AND WHITE RECIPIENTS OF RENAL ALLOGRAFTS

CE Simpkins, R Ugarte, D Chang, E Kraus, RA Montgomery, AA Zachary, C Handley, D Segev, D Warren, J Locke, JK Melancon. Johns Hopkins University School of Medicine, Departments of Surgery and Medicine, Baltimore, MD.

Introduction: Ethnicity-based inequities in access to medical care, delivery of medical interventions, and outcomes following interactions with the health care system have been described across a broad range of medical fields. The goal of this study was to assess for organ allocation and patient level differences that may contribute to the explanation of disparity in allograft outcomes following renal transplantation for black vs. white recipients.

Methods: Retrospective cohort study of prospectively collected registry information from a single university hospital experience between 1/1/1994 and 12/31/2004. Adult white (W) and black (B) recipients of a renal allograft from a living (LD) or deceased donor (DD) were identified. Outcome measures included renal allograft survival by Kaplan-Meier product limit estimate and multivariable analysis of independent risk factors for renal allograft loss by Cox proportional hazards methodology.

Results: 454 B recipients and 735 W recipients were identified for analysis. Live donor renal transplantation was more commonly performed in W vs. B recipients (63.8% vs. 19.3% of all LD transplant procedures for W vs. B, respectively). Median duration on the UNOS deceased donor waiting list was longer for B recipients of both LD and DD organs (LD: 235 days vs. 160 days, $p=0.03$; DD: 824 days vs. 526 days, $p<0.001$). Duration of cold ischemic preservation time (CIT) was longer for B vs. W recipients of DD organs (30 hours vs. 24 hours, $p<0.001$). In an unadjusted Cox proportional hazards model stratified by race, HLA-DR mismatch (HR: 2.10, 95% CI: 1.03-4.30), number of previous transplant procedures (HR: 2.12, 95% CI: 1.06 - 4.25), and history of hypertension in the recipient (HR: 1.64, 95% CI: 1.01 - 2.67) were found to be statistically significant predictors of death-censored allograft losses in B recipients. Adjusted household income $> \$40,000$ demonstrated a trend in protection against allograft loss in this group of patients (HR: 0.47, 95% CI: 0.20 - 1.09). For W recipients,

donor age > 55 years (HR: 2.40, 95% CI: 1.37 - 4.17), and HLA-DR mismatch (HR: 1.90, 95% CI: 1.02 - 3.54) were found to be statistically significant predictors of death-censored allograft loss. Risk of allograft loss for cold ischemic time > 30 hours (HR: 1.75, 95% CI: 0.91 - 3.36) was found to approach statistical significance. In a parsimonious multivariate Cox proportional hazards model, the risk of death-censored allograft loss for cold ischemic time > 30 hours and donor age > 55 years was found to be lower in black recipients (interaction p -values: 0.03 and 0.006, respectively). In addition, the effect of increasing household income over $\$40,000$ on preservation of graft function was more prominent in black recipients than in white recipients (interaction p -value: 0.04).

Conclusion: Differences exist in renal allograft outcomes between white and black recipients in the United States. Complex, multifactorial contributors, including physiologic and socioeconomic factors, appear to play a role in the differences in allograft survival that have been observed between these two groups of recipients.

NOTES

2

PATIENT LEVEL PREDICTORS OF TIMELY PROPHYLACTIC ANTIBIOTIC ADMINISTRATION

SH Gray,^{1,2} TK Houston,^{3,4} D Ordin,⁵ K Itani,⁶ M Bishop,⁶ CC Vick,^{1,4} MT Hawn.^{1,4} ¹University of Alabama at Birmingham Department of Surgery, ²Health Services & Outcomes Research Training Program, University of Alabama at Birmingham Department of Medicine, ³University of Alabama at Birmingham Department of Medicine, ⁴Deep South Center on Effectiveness, Birmingham Veterans Affairs Medical Center, ⁵Department of Veterans Affairs Office of Quality & Performance, and ⁶Department of Veterans Affairs Offices of Patient Care Services.

Background: Prophylactic antibiotic administration within 1 hour prior to incision has been proven to decrease surgical site infections (SSI). The Veterans Affairs (VA) health system has undertaken nationwide surgical infection prevention (SIP) quality improvement projects to improve performance for prophylactic antibiotic administration. This study assesses patient level characteristics that are associated with desired SIP performance.

Methods: A cross-sectional analysis was performed using data from the external peer review process (EPRP) SIP indicator for surgical patients from January through September 2005. Frequencies were tabulated on all variables of interest and Chi-Square tests were used to examine differences in proportions. Stepwise backward elimination logistic regression was used to examine possible predictors of timely administration of prophylactic antibiotic administration.

Results: The distribution of the 10,137 procedures were: Coronary artery bypass graft (CABG) (23.5%), non-CABG cardiac (4.3%), hip arthroplasty (14.7%), knee arthroplasty (23.5%), colon surgery (20.7%), hysterectomy (3.0%), and vascular surgery (10.3%). Overall, timely antibiotic administration occurred in 75.6% of the cases. Results of the Univariate analysis found procedure start time prior to 10AM to have on-time antibiotic administration in 80.3% of cases vs. 67.1% after 10AM ($p < 0.0001$). There were significant differences in SIP performance by procedure ($p < 0.0001$), with lowest performance for CABG (70.43%) and the highest performance for hysterectomy (88.64%). The proportion of timely administration increased by quarter during 2005 from 68.80% to 80.56% ($p < 0.0001$). Results from the multivariable analyses found procedure

start time, fiscal quarter, and undergoing a hysterectomy or orthopedic procedure to be significant predictors of timely antibiotic administration (see Table). Cardiac procedures were less likely to have timely antibiotic administration.

	OR Timely Administration	95% Confidence Interval
Hysterectomy	3.072	2.073-4.552
Operation 6:00-10:00AM	2.303	2.079-2.552
Qty 3 or Qty 4	1.629	1.474-1.801
Orthopedic Procedure	1.580	1.398-1.785
Cardiac Surgery	0.730	0.646-0.825

Conclusion: Measurement of SIP performance led to an increased proportion of patients receiving appropriately timed prophylactic antibiotics. The improved SIP performance with the first case is likely due to more accurate prediction of incision time. Spread of successful processes for SIP performance to the cardiac service line should be undertaken.

NOTES

FACTORS AFFECTING INNOVATION IN PEDIATRIC SURGERY: HOSPITAL TYPE AND APPENDECTOMIES

CV Pritchett, DJ Chisolm, BC Nwomeh. Ohio State University College of Medicine & Public Health, and the Office of Clinical Sciences - Columbus Children's Research Institute, Columbus, OH.

Objective: Increased application of minimally invasive surgical techniques may have implications for healthcare outcomes in children. There is little data on whether patient or hospital characteristics affect the utilization of innovative surgical techniques in children, especially with respect to laparoscopic appendectomy, whose benefit over existing treatment remains unproven. This study evaluates the patterns of laparoscopic appendectomy (LA) using a national database, focusing on variations in care between children's and general hospitals.

Methods: We collected data from the year 2000 Healthcare Costs and Utilization Project Kid's Inpatient Database admissions for patients between 5 and 20 years of age with either ICD-9-CM procedure code 47.09 (laparoscopic appendectomy) or 47.01 (other appendectomy). Demographic variables included patient age, gender, race, primary payor, and zip code median. Hospital characteristics included teaching status, rural/urban status, pediatric appendectomy volume, and pediatric categorization. Chi-squared statistics were used for univariate laparoscopy rate comparison. A logistic regression model assessed multivariate relationships.

Results: The study sample included 50,825 pediatric appendectomy patients (26% LA) representing 97,205 cases in the nation. Children's hospitals were significantly more likely to provide LA (36%) than children's units or general hospitals (28% and 25%, respectively). LA rates were also significantly positively associated with higher patient age, female gender, non-perforated appendicitis, private insurance, and White patient race. The children's hospital effect (adjusted OR 2.11, 95% CI 1.88-2.38) and all other relationships remained significant in the multivariate model.

Conclusion: While the majority of pediatric appendectomies occur in general hospitals, utilization of LA is significantly higher in children's hospitals. Children's hospitals appear more likely to adopt innovative surgical procedures, such as laparoscopic appendectomy, even when clear benefit over standard treatment has not yet emerged.

4

A WEB-BASED PROGRAM FOR THE ELECTRONIC SUBMISSION OF MORBIDITY AND MORTALITY

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Purpose: The Morbidity and Mortality Conference (M&MC) is a weekly educational conference that allows surgeons to review their complications. Traditionally, chief residents report complications on a weekly basis and often in a haphazard manner. The purpose of this work is to test the feasibility of designing a web-based program for event reporting that allows residents to document complications at the time that they occur using a National Surgery Quality Improvement Program (NSQIP) template.

Methods: On surgical services, adverse events (AE) can often lead to drastic outcomes, including death. M&MC provides the opportunity for surgeons to review their complications to prevent similar complications in the future. Unfortunately, many of these adverse events go undocumented and underreported. We hypothesized that a program that allowed for continuous AE reporting could lead to improved accuracy of complication reporting as well as improvements in patient safety. We created a web-based program for event reporting. The program consists of a list of possible complications arranged by organ system with NSQIP data elements. We hid the data elements under each organ-system heading. When that specific organ system is selected, a list of possible complications, including all NSQIP required data, explodes and is then filled out based on the complication. All unselected data points are assumed to be negative and are not included in the final report. This allows for increased efficiency, while incorporating all of the necessary data.

Results: Using this web-based NSQIP platform, we developed a queryable database to determine rates of AE for a large number of different complications. The program generates a printed report of the complication that can then be added to the patient's written medical record. The printed report includes the patient's identifying information, covering housestaff and attending, time of incident, and a full report of the complication, and the patient's current condition.

Conclusion: With web-based technology, we developed a robust morbidity and mortality NSQIP-based reporting instrument to improve our accuracy and frequency in the reporting of complications. We anticipate that this tool will allow us to further study our outcomes in order to make changes to improve patient safety and quality improvement.

MYOCELLULAR CREATINE TRANSPORTER FUNCTION IS MODULATED BY CHANGES IN ITS PHOSPHORYLATION STATUS

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Background: Muscle weakness and wasting occur in critically ill, malnourished patients. Phosphocreatine, and therefore creatine, play integral roles in myocytes to buffer energy demands. The only source of creatine (Cr) for myocytes is uptake by the creatine transporter (CrT), which may become deregulated in sepsis, heart failure, and other pathological states where energy metabolism is affected. Cr supplementation increases muscle mass and performance in humans. However, the mechanisms by which the CrT is regulated are poorly understood. We hypothesized that CrT function could be altered by specific changes in its phosphorylation status.

Methods: We used site-directed mutagenesis to introduce conservative point mutations at specific threonine/tyrosine residues on the cDNA encoding the human CrT. Using the kinetic parameters of Vmax and Km, we characterized the function of mutant CrT proteins and wildtype CrT after transfection into Griptite cells (a derivative of Human Embryonic Kidney-293 cell line, stably expressing macrophage scavenger receptor type 1). Mutant CrT protein expression was determined by western blot analysis using an epitope tag or CrT specific antibody and compared with wildtype CrT expression.

Results: The mutant CrT proteins were functional and showed significant alterations in Km and Vmax when compared with wild type CrT.

	CrT-myc	Y11F	Y338F
Vmax (Mean+SE)	5.49	8.48 ± 0.87	7.52 ± 0.47
Km (Mean+SE)	57.02	161.0 ± 3.41	101.9 ± 2.47

Conclusion: Our data suggest that tyrosine phosphorylation modulates CrT function either directly or in conjunction with other signaling events. Future investigation into CrT function and regulation may not only provide insights into its role in muscle wasting and heart failure, but may also lead to new, targeted therapies that could reduce morbidity in the critically ill.

6

TGF-BETA SIGNALING MAY PLAY A PROTECTIVE ROLE IN PREVENTING LIVER FROM INSULIN INDUCED STEATOSIS BY REGULATING INSULIN DRIVEN AKT INACTIVATION OF FORKHEAD TRANSCRIPTIONAL FACTOR PROTEINS

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Background: Non-alcoholic fatty liver disease (NAFLD) represents a progression of liver fibrosis, liver injury, and possibly development of hepatocellular carcinoma. Disruption of the transforming growth factor-beta (TGF-beta) signaling cascade, which plays a critical role in hepatic homeostasis and normal liver development, might also contribute to initiation of hepatic steatosis. Precise modulation of TGF-beta through Smad activation by the type I receptors is dependent upon adaptor proteins such as ELF, a beta-spectrin. Normal biliary epithelial development is absent in the *elf*^{-/-} and in the *Smad2*^{+/-/3+/-} mutant embryos. Both *elf*^{-/-} and *Smad2*^{+/-/3+/-} mice die at mid-gestation from defective liver development. Mice with *elf*^{+/-} mutant are viable, but as many as 35% (7/20) develop hepatocellular carcinoma (HCC) with significant centrilobular steatosis. Aside from the TGF-beta signaling pathway, forkhead transcriptional factor proteins, particularly FOXA2 and FOXO1, are essential in gluconeogenesis and fatty acid oxidation during starvation. Mice with either FOXA2 or FOXO1 knockout develop insulin-resistance and diabetes. We aim to investigate a possible cross-talk between TGF-beta signaling and forkhead protein regulation under PI3 kinase signaling.

Methods: 1. Embryonic liver tissues isolated from normal (wt), *elf*^{-/-} and *Smad2*^{+/-/3+/-} mutants were isolated and cultured in a large range of growth factor combinations and conditions to determine their growth properties. 2. Levels of p-AKT expression were analyzed in normal, *elf*^{+/-} and *elf*^{+/-/Smad4+/-} mutant cell lines. 3. Levels of p-AKT expression were re-analyzed after restoration of ELF protein. 4. Expression of forkhead protein was examined in human HCC specimen by utilizing immunohistochemistry.

Results: 1. Explants from *Smad2*^{+/-/3+/-} mutant embryo treated with insulin demonstrated a dramatic increase in steatosis. 2. Western blot analysis of cell lines derived from *elf*^{+/-} and *elf*^{+/-/Smad4+/-} mutants revealed significant increases in p-AKT level compared to the wild type cell line. 3. p-AKT levels in these mutant cell lines decrease with restoration of ELF protein. 4. Immunohistochemical labeling of hepatocellular cancer taken from *elf*^{+/-} mice revealed increased cytoplasmic labeling of FOXO protein.

Conclusions: In *Smad2*^{+/-/3+/-} and *elf*^{+/-} mutant embryo explants, the addition of insulin resulted in severe steatosis. The mechanisms of these findings could be secondary to the alteration in forkhead protein transcriptional factors level whereby forkhead proteins are sequestered by insulin-driven Akt, and are unable to partner with Smads and ELF to modulate expression of forkhead target genes. More importantly, in adult tissues these derangements may promote fibrosis, steatosis and hepatocarcinogenesis.

NOTES

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BEARD SYCOSIS AND DISFIGURING KELOID SCARRING OF THE FACE, NECK AND SCALP: A 15 YEAR EXPERIENCE

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Background: Beard Sycosis is a term for patients who develop disfiguring and recurrent keloid scar formation following folliculitis or pseudofolliculitis. The nomenclature for this disease process is yet to be fully elucidated. The causes of this problem include: Pseudofolliculitis barbae, Acne keloidis, and Acne keloidalis nuchae. Pseudofolliculitis barbae (razor bumps) is a common condition of the beard area occurring in African and African-American men and other people with curly hair who must shave frequently. We report our surgical experiences in treating patients with this often chronic and recurrent condition with a staged operative approach. Additionally we offer our experiences with adjuvant radiation therapy and long-term follow-up with excellent results.

Patients and Methods: Thirteen patients had disfiguring keloid lesions of the beard and in some cases the posterior nuchal region all treated at Cook County Hospital and The University of Illinois Medical Center between January 1987 and December 2002. All patients were male. The age of the patients ranged from 23-65 years of age. All of the patients were of African or African-American heritage.

Results: Twelve of the thirteen patients had satisfactory results overall. There were no major infections, no total loss of skin grafts, and no major recurrences within a two-year time period. One patient had a minor local infection which resolved without antibiotics. Eight patients had minor split-thickness skin graft loss. Three patients had minor wound separation. Three patients had small minor recurrences at the periphery of the wound. One patient was lost to follow up. After six years the patient re-presented with a complete recurrence. All patients had a major improvement in quality of life, and all were able to return to their pre-morbid activity (i.e. work, school). Eleven of the 13 patients who underwent excision followed by split-thickness skin grafting achieved complete wound closure without recurrence in the immediate post-op period. Two of the 13 patients required additional surgical treatment.

Conclusions: Although not a novel approach, the staged operative treatment for patients suffering with Beard Sycosis with and without the use of adjuvant radiation therapy has led to excellent results with minor recurrences. Patient appearance and satisfaction was markedly improved.

NOTES

IN VIVO MODEL OF ADIPOGENESIS USING A HYDROGEL BIOMATERIAL

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Background: Tissue-engineered biomaterial constructs will become a big asset to plastic and reconstructive surgery. We successfully created a sodium alginate based macroporous hydrogel bead. The bead features a cell adhesion molecule (arginine, glycine, aspartic acid peptide a.k.a. RGD) covalently bonded to the sodium alginate to promote cellular adhesion and proliferation. Our previously published in vivo studies have shown that this biomaterial causes minimal inflammatory reaction, minimal capsule formation, and supported vascular as well as cellular ingrowth into the material. These studies were conducted using animal (sheep) preadipocytes.

Purpose: This study focuses on human preadipocytes and evaluating their ability to adhere to alginate-RGD biomaterial and differentiate into adipose tissue.

Methods: Human pre-adipocytes (Zen Bio INC) were exposed to differentiation media in vitro and differentiation was determined by quantification of leptin protein production via ELISA, and visualization of lipid formation via Oil Red O staining. Human preadipocytes were seeded onto porous alginate-RGD beads using a roller bottle technique and evaluated for viability through confocal microscopy and a live-dead assay.

Results: The in vitro experiments demonstrated human pre-adipocyte differentiation into adipocyte tissue as evidenced by increased leptin production and lipid droplet formation when compared to undifferentiated controls. Confocal microscopy in conjunction with a live--dead assay revealed successful seeding of human preadipocytes onto alginate-RGD beads. In addition, Oil red O staining of pre-adipocyte seeded alginate beads demonstrated intracellular lipid droplet formation after exposure to adipocyte differentiation media.

Conclusions: Human preadipocytes show the ability to attach to the alginate-RGD biomaterial and differentiate into adipose tissue. The continuation of this project will evaluate the ability of the biomaterial to form and maintain adipose tissue in vivo. This will entail the subcutaneous implantation of the biomaterial construct into SCID mice. The implants will be harvested and evaluated for adipose tissue differentiation, vascular tissue formation, native tissue ingrowth, and inflammatory response.

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DISRUPTION OF TGF- β SIGNALING AND ITS RESTORATION MODULATED HEPATOCELLULAR CARCINOGENESIS IN HEPATOCELLULAR CANCER CELL LINES AND ELF+/- MUTANT TISSUES

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Background: Hepatocellular cancer (HCC) is the fifth most common solid malignancy worldwide and is increasing in the United States. Nearly 500,000 cases of HCC are diagnosed each year, prognosis remaining extremely poor. Recent studies in human HCCs reveal the emergence of transforming growth factor-beta (TGF- β) as a key signaling pathway in suppressing these cancers through Smad proteins and adaptor proteins such as the embryonic liver fodrin (ELF). TGF- β can induce antiproliferative gene responses by inhibiting cellular progression in G1 phase. Arrested cells in the G1 phase display downregulation in expression of Cdk2, Cdk4, cyclin D1, cyclin D2, cyclin D3 and cyclin A. Escape from this response is a hallmark of many cancer cells. Importantly, our lab has demonstrated that as many as 35% of elf+/- mice (7/20) develop HCC spontaneously and show additional phenotypic changes such as increased centrilobular steatosis and high grade dysplasia.

Methods: 1. To determine whether ELF and TGF- β signaling proteins are inactivated in human HCC cell lines SNU-182, SNU-398, SNU-449, and SNU-475. 2. To investigate ELF expression in human hepatocellular carcinoma samples by immunohistochemistry. 3. To analyze the changes in cell cycle regulation in wild type mouse embryonic fibroblasts (MEF) and elf-/- MEF cell lines by western blot analysis. 4. To investigate whether restoration of ELF can reverse the aberration of cell cycle regulation.

Results: 1. Expression of ELF and other proteins involved in the TGF- β signaling pathway, particularly Smad2, Smad4 and TGF-beta receptor II (TBR II), were examined in human HCC cell lines SNU-182, SNU-398, SNU-449, and SNU-475. ELF expression was lost in one human HCC cell line (SNU-398), and decreased in SNU-182 and SNU-475. Smad 2 expression was lost in all five

HCC cell lines. TBR II expression was lost in three cell lines (SNU-398, SNU-182, and SNU-475). 2. Further analysis of the role of ELF in human HCC confirms markedly reduced nuclear expression of ELF in 7 out of 9 human HCC samples by immunohistochemistry. 3. Immortalized elf-/- MEF cell lines showed a marked increase in Cdk4 level by 3-4 times. 4. Immunohistochemical labeling of elf+/- HCC tissue revealed markedly increased cyclin D1. 5. Restoration of ELF protein in human HCC cell line, SNU-182, results in a decrease in cyclin D1 protein expression via Western blot analysis.

Conclusions: Diminished or absent ELF expression in mouse and human HCC as well as cell cycle deregulation are due to disruption of TGF- β signaling. Loss of ELF can serve as a primary event in progression towards a fully transformed phenotype. Exploration of the mechanisms behind inactivation of the TGF- β signaling pathway and its restoration holds promise for new therapeutic approaches in human hepatocellular cancer.

NOTES

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PANCREATIC STELLATE CELLS PROMOTE MALIGNANT POTENTIAL IN PANCREATIC CANCER

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Background: Pancreatic adenocarcinomas contain a significant degree of tumor-associated stroma. In other solid tumors, such as breast and prostate cancer, carcinoma associated fibrosis contributes to the malignant phenotype by stimulating tumor proliferation, and increasing invasive and metastatic ability. However, the role of carcinoma-associated fibrosis in pancreatic cancer is not well understood. We hypothesize that pancreatic stellate cells in the stroma promote the malignant phenotype of pancreatic adenocarcinoma cells.

Methods: Rat pancreatic stellate cells (RPSCs) were isolated and established in primary culture. Conditioned media from either RPSCs or immortalized rat pancreatic stellate (LTC-7) cells was added to Panc-1, MiaPaCa, or MPanc96 pancreatic cells and proliferation was assessed using a standard MTS assay. A survival assay was performed to assess whether conditioned media improved pancreatic cancer cell survival in the presence of gemcitabine. The rates of migration and invasion of the tumor cells were evaluated using a Transwell culture system in the presence of either concentrated conditioned media, or RPSC or LTC-7 cells in the lower chamber for 48 hours.

Results: Panc-1 cells increased proliferation in the presence of conditioned media derived from RPSCs. Panc-1, MiaPaCa and MPanc96 cells showed statistically significant increased rates of migration in the presence of either RPSCs, LTC-7, or concentrated conditioned media (CCM) from LTC-7 cells.

Table. Migration Rates. (Mean number of cells in three non-overlapping high powered fields at 20x magnification + Standard Deviation.)

	Serum Free Media (Negative Control)	RPSC	LTC-7	CCM from LTC-7
Panc-1	0.33 + 0.6	4.0 + 1.7*	38.67	62.33 + 2.5*
MiaPaCa	0	11.67 + 2.3*	19.33 + 3.1*	4.0 + 2.6
MPanc96	1.33 + 0.6	14.33 + 2.3*	6.67 + 4.1	5.0 + 0

* indicates p-value < 0.05

Survival rates of Panc-1, MiaPaCa and MPanc96 in gemcitabine were increased in the presence of conditioned media from RPSCs (all p-values <0.01).

Conclusion: Pancreatic stellate cells promote proliferation and migration in selected pancreatic cancer cell lines.

NOTES

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ESOPHAGEAL ADENOCARCINOMA IN AFRICAN-AMERICANS: RARE AND DEADLY

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Background: Esophageal adenocarcinoma (EAC) occurs much more commonly in men than women, and Caucasians than African-Americans (AA). Few reports have examined differences in clinicopathologic characteristics between AA and Caucasians with EAC since so few AA with EAC present to any single institution. We hypothesized that EAC in AA has less gender disparity, but poorer survival than in Caucasians.

Methods: Utilizing the Healthcare Costs and Utilization Project's in-patient database for the national and regional data, 7,541 EAC patients from 38 states (1999-2002) were compared with 2,016 EAC regional patients from Maryland (1994-2004). Using the Johns Hopkins Hospital (JHH)'s inpatient records and the institution's tumor registry, 659 EAC local patients treated from 1984 to 2002 were further compared to national and regional data. Although demographic data were available in all databases, survival, stage and risk factors for EAC were available only at the local level.

Results: Prevalence rates of EAC among AA are low compared to Caucasians (nationally 6.8% vs. 62.4%, regionally 15.4% vs. 82.6%, locally 3.8% vs. 96.2%). Three-year survival rates at JHH for AA are significantly worse than for Caucasians (4% vs. 28%, $p < 0.001$). AA presenting locally are also at a significantly more advanced stage of disease than Caucasians (Stages III/IV: 68% vs. 50%, $p < 0.001$) and this is the most likely explanation for their poor survival. The low rates of surgery with curative intent among AA compared to Caucasians at national (7.2% vs. 12.7%) and regional levels (10.6% vs. 19.5%) also imply a later stage of EAC presentation. Local data revealed no differences in risk factors associated with EAC such as prevalence of Barretts', gastroesophageal reflux disease, hiatal hernias, and cigarette smoking. There were, however, important age and gender differences. Both AA males and females were persistently younger at EAC presentation (nationally: 63 years vs. 68 years, $p < 0.01$) and EAC was significantly more prevalent in AA females than Caucasian females (nationally, 40% vs. 22%; $p < 0.001$; regionally, 50% vs. 20%, $p < 0.001$; locally, 50% vs. 14%, $p < 0.001$).

Conclusion: Overall, EAC is more common in males than females, but if stratified by race and ethnicity, EAC is less prevalent in male AA than Caucasian males, but more prevalent in female AA than Caucasian females. Furthermore, there is an age-of-onset discrepancy by ethnicity with AA being significantly younger. Finally, EAC in AA is associated both with more advanced disease and worse survival.

NOTES

EARLY-AGE-ONSET FAMILIAL PANCREATIC ADENOCARCINOMA MAY IMPACT LONG-TERM SURVIVAL

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Introduction: Familial pancreatic adenocarcinoma (FPAC) accounts for 5-10% of all pancreatic cancer cases. Tobacco use and early-age-of-onset have been associated features of FPAC. The purpose of this study is to evaluate the effect of these two risk factors on survival in this genetically predisposed population.

Methods: A retrospective review of a prospective FPAC database was performed. Patients were evaluated for age at diagnosis and history of tobacco use. The cohort was divided into patients with early-onset of disease (diagnosed before age 60) and those with late-onset of disease (diagnosed at age 60 or above). Additional analyses were performed based on history of tobacco use. Patients were divided as those with smoking histories (past or present) versus those who have never smoked. Survival estimates were calculated by the method of Kaplan and Meier.

Results: 31 patients were identified with a diagnosis of familial pancreatic cancer. The male to female ratio was 1.8:1. The median age at diagnosis for the entire cohort was 60 years (range 35-80). Smoking histories were obtainable for 26 patients. At the time of diagnosis, 20 (77%) patients had a past or present history of tobacco use, and 6 patients (23%) never smoked. Median survival was 7 months for the smoking group (CI 4,10-current smokers, CI 6,8-former smokers) and 4 months (CI 3,5) for patients who never smoked. Long-term survival was observed in 40% of the smoking group and in 17% of the non-smoking group (p=NS). 15 patients had early-onset of disease (<60 years of age) with a median survival of 6 months (CI 2,10), and 16 patients had late-onset of disease (>60 years of age) with a median survival of 7 months (CI 5,9). Long-term survival (>12 months) was observed in 20% of the early onset patients and in 44% of those with late onset of disease (p=NS).

Conclusion: Tobacco use is prevalent in patients with familial pancreatic carcinoma and continues to be a significant risk factor for this disease. However, no survival difference was noted in smokers versus non-smokers with FPAC. Fewer patients diagnosed at an early age of onset had long-term survival compared to those diagnosed at later ages.

SCREENING FOR BLUNT THORACIC AORTIC INJURIES: ARE CHEST X-RAYS ADEQUATE?

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Objective: Thoracic aortic injuries following blunt trauma mechanisms are associated with high mortality rates. For survivors, chest X-rays (CXR) have traditionally been used as the screening modality to decide on the need for further diagnostic testing. The liberal use of spiral chest CT angiography (CTA) following all high-speed crashes regardless of CXR findings has been advocated. We set to determine the adequacy of utilizing CXR alone as a screening tool for blunt thoracic aortic injury (BTAI) at a Level I Trauma Center.

Methods: All patients with BTAI diagnosed clinically or at autopsy admitted to a Level I Trauma Center between Jan 1998 and Dec 2004 were identified. The CXRs of these patients and those of a cohort of blunt trauma patients with an ISS > 15 (control group) in the same time period were retrospectively reviewed by 4 trauma surgeons who were blinded to the diagnosis. Each of the trauma surgeons decided, based on each film viewed, if they would have proceeded to chest CTA, conventional aortic angiography or required no further studies to rule out BTAI.

Results: In the 7-year period, there were 83 patients with BTAI. CXRs were available for 47 patients, all motor vehicle crash victims. The CXRs of these patients and those of 49 controls were reviewed. Of the 47 patients with BTAI, the surgeons chose to proceed to chest CTA in 38 patients (81%), conventional aortography in 2 patients (4%) and no further testing in 7 patients (15%). A widened mediastinum was the most common CXR feature chosen to decide the need for further studies - 34/47 (72%). Loss of AP window (38%), indistinct aortic knob (28%) and pleural capping (4%) were other reasons. Patients with BTAI were more likely to have a positive CXR (40/47) when compared with the controls (25/49), statistically significant by chi-square testing ($p < 0.001$). CXR sensitivity and specificity were 85.1% and 51%, respectively.

Conclusions: CXR is sensitive as a sole screening test for BTAI, but failed to identify up to 15% of patients with BTAI. Liberal use of chest CTA, especially following high-speed motor vehicle crashes, is highly recommended to minimize the incidence of missed thoracic aortic injuries. A widened mediastinum was the most frequent criterion used to indicate a positive CXR.

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A COMPREHENSIVE INJURY RISK ASSESSMENT SCREENING TOOL FOR SENIOR POPULATIONS: PRELIMINARY FINDINGS FROM A MINORITY COMMUNITY

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Background: Conducting injury prevention in elderly populations is more complex than in children due to the interactions of function, frailty, multiple comorbid conditions, and poly-pharmacy. While there are gaps in knowledge and a continuing need to improve interventions, there is a missed opportunity to apply existing knowledge in a comprehensive systematic approach to injury prevention in the elderly.

Objectives: To develop and test a comprehensive risk assessment tool suitable for identification of injury risk in seniors.

Methods: We developed and administered an abbreviated version of our Comprehensive Injury Risk Assessment and Reduction for Elderly Populations (CIRA-REP) instrument to assess individual, community, environmental, medical care and intrinsic comorbid disease risk in seniors (n=100). The full-length CIRA-REP contains 4 subscores for measuring baseline and follow-up modifiable risk from 4 sources: Community (Subscore A); Home (Subscore B); Individual intrinsic based on symptoms, health status, comorbid diseases, physical and cognitive profile (Subscore C); and a Medical Care and Rehabilitation Component (Subscore D). All injury risk assessments were administered in the Harlem community across a variety of settings (injury fair, commercial banks, and other public places).

Results: Nearly two-thirds of seniors reported a history of injury, 90.5% of whom had emergency department treatment and 54.0% hospitalization for injury. *Fall Risks:* Medical conditions associated with frailty, balance, gait and strength can contribute to falls as well as home and environmental conditions. The distribution of medical conditions/symptoms included dizziness (17.3%), seizures (5.0%), history of fainting (8.1%), difficulty sleeping (30.6%), difficulty with urination (32.0%), irregular heart beat (27.4%), previous stroke (13.0%), dementia (2.0%), diabetes (20.0%), hypertension (60.0%), and depression (19.4). At least one-third reported some functional limitation or difficulty with walking outdoors (28.0%), climbing/descending stairs (25.5%), walking indoors (23.0%), showering and/or bathing (78.0%).

Home and environmental conditions with potential to contribute to falls included lack of grab bars in bath/shower (71.7%), handrails on stairs (16.5%), night-lights (32.7%), and flashlights (28.3%). Mild, moderate, or severely low bone density was present in 60.7%, but only 47.1% of those were on calcium supplements. *Poisoning Risks:* Multiple risk factors were present for accidental medication poisonings. Poly-pharmacy was common with the mean number of medications taken by seniors at 4.6 (3.1 prescription and 1.5 nonprescription medications). Nearly one-third (31.9%) reported no pill box organizer and 7.6% reported having difficulty remembering to take their medications. During screening, 38.8% of patients could not see to read fine print on pill bottle either with or without glasses and 11.2% could not read contraindication and warning labels on a pill bottle with a lighted magnifying glass. Hearing is judged to be an important component in comprehension of medical and pharmaceutical instructions; however, of the 15 participants reporting difficulty hearing, only half reported having a prior hearing test and one-fourth used a hearing aid. *Fire/Burn, Heat and Cold Exposure:* The majority of senior homes lacked one or more safety devices. Although most had a smoke alarm (93.9%), only 71.0% of those reported having changed the batteries in the last 6 months. The majority had no room thermometer to measure temperature of their residence (81.6%). Nearly one-third lived in non-air conditioned residences, but only 3.4% reported having no fan or air conditioner for summer cooling. Approximately 40% had no iron with an automatic shut-off and few had or used a kitchen timer when cooking.

Conclusions: This comprehensive injury risk assessment instrument is a useful tool for identification of modifiable injury risk factors in minority community-dwelling seniors. It was instrumental in identifying risks associated with mechanisms identified during surveillance as important contributors to injury-related mortality, hospitalization, and/or emergency department visits.

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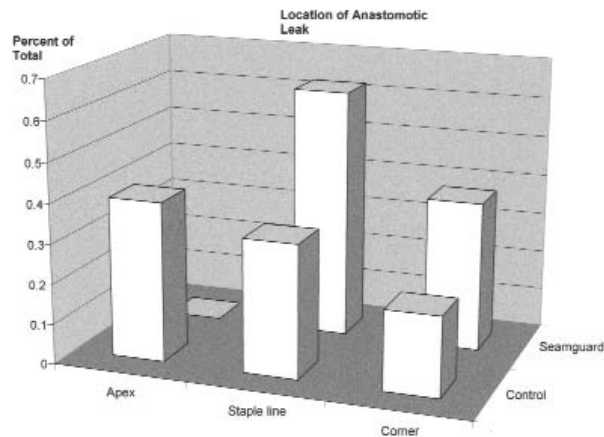
ANASTOMOTIC STAPLE LINES: DOES REINFORCEMENT MATTER?

RJ Howard, M LaSpina, K Snyder, R LaFollette, J DiRocco, V Panemanglore, IA Munshi. SUNY Upstate Medical University, Syracuse, NY.

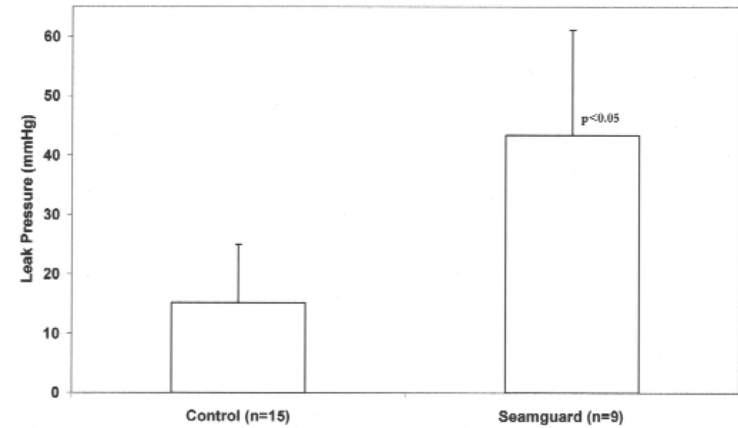
Background: Staple line disruption is a feared complication after gastrointestinal anastomosis. Staple line reinforcement is thought to decrease anastomotic leak rates, decrease staple line bleeding and increase the strength of the staple line. This study was designed to evaluate the strength of PGA/TCA (Seamguard, GORE) staple line reinforcement in a porcine model.

Methods: A side-to-side jejunojejunostomy is created in a porcine model using the ETHICON 6 row stapler (45mm blue cartridge) with and without PGA/TCA reinforcement material. The enteroenterostomy is then closed transversely using the same instrument with and without reinforcement materials. The anastomosis is subjected to increasing intraluminal pressure by instilling methylene blue dye until a leak is noted. Burst pressure is defined as the final pressure at which the dye is noted to first leak from the staple lines.

Results: Twenty-four side-to-side jejunojejunostomies were created in the mid-small bowel of the porcine model. Fifteen of these were without staple line reinforcement and nine were with PGA/TCA reinforcement. The bursting pressure data are listed below. Data were analyzed using Student's t test with a $p < 0.05$ level of significance.



Bursting Pressures



Conclusion: Staple line reinforcement significantly increases the strength of small bowel side-side anastomoses as determined by bursting pressure measurements. The areas of weakness with and without SeamGuard appear to be at the crossing staple lines and the apex, respectively.

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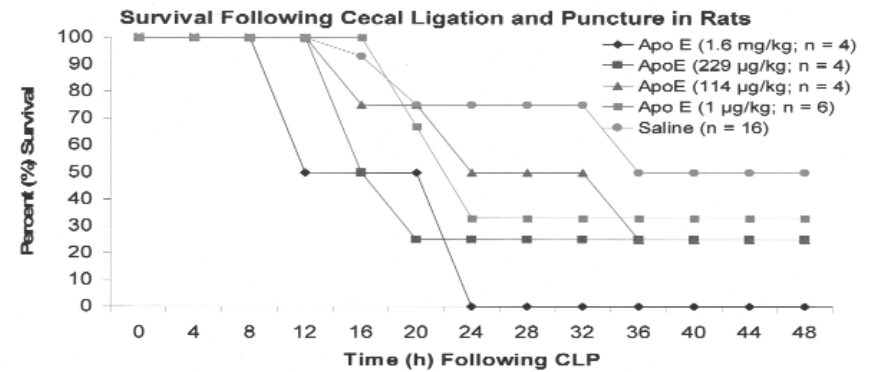
APO E IS ASSOCIATED WITH INCREASED MORTALITY IN SEPTIC RATS

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Background: In addition to Apolipoprotein E (Apo E) functioning as a mediator of extracellular lipid metabolism, recent studies have suggested Apo E may have an additional role in immunity. Apo E has been shown to bind lipid antigens and present them to endosomal compartments of antigen-presenting cells (APC) containing CD1 via low density lipoprotein receptors (LDLR) and LDL-like receptor proteins (LRP). APC then present these lipid antigens as a mechanism to recognize foreign antigens and stimulate an immunologic response. Consequently, we hypothesized that increased concentrations of Apo E during sepsis could hyperstimulate the host innate immune response, leading to septic shock and increased mortality.

Methods: Male Sprague-Dawley rats weighing approximately 300 kg were used. A catheter was placed in the external jugular vein for serial dosing and blood sampling. After the rats recovered for 24 h following the catheter placement, cecal ligation and puncture (CLP) was performed. Various concentrations of Apo E (1.6 mg/kg, 229 μ g/kg, 114 μ g/kg, or 1 μ g/kg), or equivalent volume of saline in controls, was injected via the catheter immediately after CLP (time = 0 h) and every 4 h for 24 h. Survival was assessed at 48 h.

Results: Saline control rats (n = 14) had a survival rate of 33% (Figure). We observed an inverse relationship between survival and Apo E concentration. Rats that received Apo E at a concentration of 1 μ g/kg (n = 6) had a survival rate of 33% compared to a 0% survival rate among rats that received Apo E at a concentration of 1.6 mg/kg (n = 4). Interestingly, none of the Apo E groups had a survival rate comparable to saline controls.



Conclusion: During sepsis, Apo E may act to further stimulate the innate immune response through increased lipid antigen presentation, leading to septic shock and higher mortality.

NOTES

MURINE BURN WOUND INFLAMMATION THAT INCREASES BURN DEPTH MAY BE CAUSED BY LOCAL REPERFUSION INJURY

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Background: Specific antibodies that produce reperfusion injury in antibody-deficient mice also produce marked deepening of burn wounds in the deficient mice. Likewise, a peptide that mimicks the antibody binding sites can prevent both reperfusion injury and deepening of burn wounds in mice with no defined deficiencies (C57/BL6). To test whether a cutaneous burn might manifest a loss of perfusion and then a time of reperfusion, we studied mice in a scald burn model.

Methods: A 2.5% BSA intrascapular wound was created in anesthetized and shaved C57BL/6 and Balb-C mice and titrated to produce a distinct 1cm² wound that heals by secondary intent. At certain times (10 min, 20 min, 30 min, 60 min, and 90 min) before and after burn, 100 µl of 1% Evans blue (MW=960) was administered i.v. to assess permeability changes and subsequent edema. Photos or biopsies were taken of either intact wounds or the underneath wound and vasculature. Permeability was quantitated by measuring the absorbance of proteinase K extracts of Evans blue dye at 630 nm with a spectrophotometer. Edema was measured by determining the wet weight of burn skin sections at 60 min after burn and comparing them to control skin sections.

Results: Mice given Evans blue dye after the burn demonstrated a temporal pattern of skin blanching, suggesting vasospasm seen more at the early time points, followed by accumulation of dye limited to the burn area, indicating reperfusion of leaky vessels. In mice given Evans blue dye before the burn, direct visualization of the vasculature showed extravasation of dye limited to the burn area. This effect was not seen in mice that were pretreated with the peptide antigen mimick that blocks binding of the IgM clone that initiates reperfusion injury. There was a 250% increase in permeability in the burned skin compared to the unburned skin. Edema was increased by 60% in burned skin.

Conclusion: These data indicates that scald burns have intense vasospasm followed by reperfusion and capillary leakage. This element of reperfusion may explain why agents designed to prevent reperfusion injury also control damage to burns caused by host inflammatory elements.

HOSPITAL-BASED VIOLENCE INTERVENTION PROGRAMS WORK

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Purpose: Hospital-based violence prevention programs have emerged at trauma centers nationwide; however, none has been thoroughly evaluated for effectiveness. Our Violence Intervention Program (VIP) conducted a prospective randomized control study to evaluate the effectiveness of intervention for repeat victims of violence.

Methods: Patients admitted between 1999 and 2002 for treatment of injuries inflicted by a violent act were identified. Repeat victims of violence on parole/probation were invited to join the study. Participants were given a history-gathering questionnaire and randomized into two groups. Cases (intervention [n=56]) received intensive psychosocial follow-up services, substance abuse treatment and family/group therapy. Controls (non-intervention [n=44]) received standard medical treatment and follow-through in accordance with standard parole/probation procedures.

Results: There was no significant difference in the number of arrests in the two groups. The control group was three times more likely to be arrested for a violent crime, two times more likely to be convicted of any crime, and four times more likely to be convicted of a violent crime. The projected time of incarceration is significantly longer for the control group. Repeat violent criminal activity was significantly more evident in the control group.

Conclusion: Significant differences exist between the VIP intervention/non-intervention groups in terms of the quantity and severity of criminal activity.

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EARLY ENTERAL NUTRITION IMPROVES GUT EDEMA-ASSOCIATED ILEUS

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Background: Damage control laparotomy is associated with intra-abdominal hypertension (IAH), gut edema and ileus, but surprisingly we have shown that patients tolerate early enteral nutrition (EEN). Abdominal closure can be complicated by IAH with concomitant elevation of abdominal venous pressures. In our lab we have shown that gut edema created by elevating mesenteric venous pressure and fluid resuscitation impairs intestinal transit without imitating an inflammatory response. We therefore hypothesized that EEN would improve intestinal transit and down-regulate pro-inflammatory gene expression.

Methods: At laparotomy, rats had a duodenal catheter placed and were given an 80 cc/kg bolus of 0.9% saline and superior mesenteric venous pressure elevation (IAH) + EEN (12 h continuous infusion via duodenal catheter) or sham surgery. At 12 hrs, EEN was discontinued and FITC-Dextran was instilled into the gut via the duodenal catheter to evaluate intestinal transit expressed as the mean geometric center (MGC). Small intestine was harvested for evaluation of tissue water weight and histological injury and isolation of total RNA to analyze gene expression using cDNA microarray.

Results: Rats subjected to IAH without EEN had markedly depressed intestinal transit, very mild mucosal injury and more genetic alterations. In comparison, rats subjected to IAH + EEN had markedly improved intestinal transit, minimal mucosal damage and less genetic alterations. Microarray analysis revealed genetic alterations in cell structure/integrity, cellular development, protein synthesis and degradation, apoptosis, and metabolic enzymes. Minimal changes were noted in the inflammatory and stress response genes. Wet to dry ratios were the same in both IAH groups.

	Tissue Water (Wet-to-Dry)	Mucosal Injury (Chiu Score)	Intestinal Transit (MGC)	Microarray (genes up-regulated)	Microarray (genes down-regulated)
Sham	3.2 + 0.1*	0.3 + 0.2	4.2 + 0.3	--	--
IAH	3.7 + 0.1	1.3 + 0.5	3.6 + 0.1**	774	1061
IAH + EEN	3.7 + 0.1	0.5 + 0.2	4.5 + 0.2	44	55

Data are expressed as mean \pm SEM, n=5-6 and *, ** = p < 0.05 vs. all groups using ANOVA.

Conclusion: Enteral feeding improves gut edema-induced delayed intestinal transit. In-depth analysis of the transcriptional alterations induced by gut edema and changed by feeding may provide a foundation for identifying novel therapeutics to improve intestinal transit.

NOTES

CYCLOSPORIN A INHIBITS HEMORRHAGIC SHOCK-INDUCED HYPERPERMEABILITY AND MITOCHONDRIAL TRANSITION PORES

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Introduction: Cyclosporin A (CSA), a commonly used immunosuppressive agent, is a known inhibitor of the mitochondrial transition pore (MTP). Recent data have suggested that the release of reactive oxygen intermediates from the mitochondria may alter microvascular hyperpermeability following hemorrhagic shock (HS). The purpose of this study was to determine if CSA given before, during, and after HS could attenuate microvascular permeability, and if this attenuation was related to depolarization of the mitochondrial transition pores (MTP) and inhibition of cytochrome c release.

Methods: Following a control period, blood was withdrawn to reduce the mean arterial pressure to 40 mmHg for one hour in urethane-anesthetized rats. Mesenteric postcapillary venules in a transilluminated segment of small intestine were examined to measure changes in permeability utilizing intravital microscopy. The rats received an intravenous injection of FITC-albumin during the control period. The fluorescent light intensity emitted from FITC-albumin was recorded within the vessel lumen and in the adjacent extravascular space to determine albumin flux. The images were analyzed to determine changes in light intensity.

Results and Conclusion: Our results demonstrated a marked increase in albumin leak following HS that was significantly attenuated with CSA given before and during HS ($p < 0.05$). In addition, CSA attenuated the release of the mitochondrial sensitive fluorescent probe, JC-1 (5,5',6,6'-tetra chloro-1,1',3,3'-tetraethylbenzimidazolyl carbocyanine iodide) and cytochrome c from the mitochondria, suggesting inhibition of the MTP as a possible mechanism.

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AN EVALUATION OF OUTCOMES AFTER CAROTID ENDARTERECTOMY WITH EXTRA- AND INTRA-CRANIAL TANDEM LESIONS

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Introduction: The North American Symptomatic Carotid Endarterectomy Trial (NASCET) in 1991 described the benefit of carotid endarterectomy (CEA) over medical treatment in symptomatic patients with carotid artery stenosis greater than 70%. The recommendations outlined from this trial did not include the association of intra-cranial atherosclerotic disease. The incidence of tandem lesions has been reported to be between 10-84%. There are several reports in the literature describing successful treatment of tandem lesions through either a combined approach or simultaneous stenting. However these reports remain largely anecdotal and the question regarding the outcomes of treated tandem lesions remains unanswered.

Methods: Literature review and meta-analysis.

Results: Studies prior to and since NASCET have attempted to look at peri-operative stroke, morbidity and mortality rates in patients with extra- and intra-cranial tandem lesions and those with carotid artery bifurcation disease only. To date, there is no statistical evidence that there is an increased risk of morbidity and mortality following open CEA in patients with tandem lesions. We found that there were only 5 studies which attempted to look at open repair and tandem lesions and evaluate the outcomes. Of the five studies, only three had similar endpoints. Meta-analysis yielded 816 patients with bifurcation disease and 337 patients with tandem disease. The 30-day mortality was 1.48% for tandem lesions and 0.98% for bifurcation lesion ($p=0.26$). The 30-day stroke rate was 4.45% versus 2.45% for tandem and bifurcation lesions, respectively ($p=0.26$). Late death rates (over 30 days) were statistically significant for tandem versus bifurcated lesions (23.29% versus 18.10%, respectively, $p=0.025$). Further evaluation of the causes of late death shows cardiac causes for those with tandem lesions (10.44%) and bifurcation alone (8.22% $p=0.14$). Evaluation of stroke as a late death was not statistically significant.

Conclusions: Carotid endarterectomy (CEA) is an established treatment for carotid disease. CEA does appear to benefit patients with tandem lesions. Our meta-analysis suggests that those patients with tandem lesions have more severe cardiac disease at presentation than those with bifurcation disease alone. This may account for the increased rates of late deaths in patients with tandem lesions.

NOTES

ISCHEMIC PRECONDITIONING IN MYOCARDIAL HYPERTROPHY INDUCES A DISTINCT PATTERN OF JAK-STAT ACTIVATION

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Objective: The Janus Activated Kinase (JAK) and Signal Transducers and Activators of Transcription (STAT) pathway is activated in the early and late phases of ischemic preconditioning (IPC) in normal myocardium. We have previously shown that STAT-3 activation is associated with effective preconditioning in myocardial hypertrophy. The purpose of this study was to determine if the pattern of JAK-STAT activation in myocardial hypertrophy is similar to that identified in non-hypertrophied hearts.

Methods: Male Sprague-Dawley rats eight weeks following thoracic aortic constriction (TAC) or sham operation underwent Langendorff perfusion. Randomized hearts were subjected to 30 minutes global ischemia, 120 minutes reperfusion with or without IPC in the presence or absence of the JAK-2 inhibitor AG490. Nuclear fractions from left ventricular samples were separated on 10% SDS-PAGE and probed with primary antibody against total and phosphorylated STAT-1, STAT-3 and STAT-5. Cut gels were probed with primary antibody to GAPDH to normalize protein loading.

Results: In the absence of preconditioning, hearts from TAC rats had increased pSTAT-1 and pSTAT-3 expression compared to sham. Ischemic preconditioning further increased only nuclear pSTAT-3 but had no additional effect on pSTAT-1 or pSTAT-5 expression. Hearts from sham operated animals showed an increase in both pSTAT-1 and pSTAT-3 expression with no significant increase in pSTAT-5 in response to preconditioning. Treatment with AG490 in hearts from both TAC and sham operated animals decreased only nuclear pSTAT-3.

Conclusions: Ischemic preconditioning in hypertrophied myocardium is associated with activation of the JAK-STAT pathway and a pattern of STAT activation that is different from that identified in non-hypertrophied myocardium. Gene-targeted manipulation of this pathway may potentially benefit patients with myocardial hypertrophy subjected to ischemia/reperfusion stress.

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THE ROLE OF TOLL-LIKE RECEPTORS IN THORACIC AORTIC ISCHEMIA REPERFUSION

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Introduction: Toll-like receptors (TLRs) are critical in early innate immunity to invading pathogens. Recently, TLR-4 has been identified as an important mediator of the inflammatory response to ischemia/reperfusion (I/R) injury in isolated mouse organ systems such as the brain, heart, lung, liver, and kidney. In these models, the absence of TLR-4 reduces I/R injury. The thoracic aorta ischemia reperfusion (TAR) model in mice is unique in that it serves as both a systemic model for I/R injury as well as a model for I/R injury of the spinal cord. These experiments were designed to evaluate whether the absence of TLR-4 confers survival benefit or spinal cord protection in response to TAR.

Methods: Fourteen TLR-4 knockout (TLR-4^{-/-}) mice and 8 control mice (C) were subjected to 11 minutes of normothermic TAR and 24 hours of reperfusion. The thoracic aorta was clamped at the level of the left subclavian artery. Distal corporal and hindlimb ischemia were qualitatively confirmed using laser Doppler imaging. Mice were evaluated by blinded observers at 0, 6 and 24 hours using an established rodent paralysis scoring system, score 0-6, with 0 indicating no injury and 6 indicating complete paralysis. The mice were sacrificed at 24 hours and the tissues were collected for analysis. Results were normalized per mg total protein, and reported as the mean \pm standard error (SE).

Results: TLR-4^{-/-} mice demonstrated improved survival and spinal cord protection as compared to control mice following TAR. 13 of 14 (93%) TLR-4^{-/-} mice were alive at 24 hours versus 4 of 8 (50%) control mice ($p=0.02$). Similarly, the TLR-4^{-/-} mice demonstrated a statistically significant improvement in paralysis score at 24 hours as compared to controls (2.3 ± 0.77 vs. 6 ± 0 ; $p<0.01$). A trend towards a decrease in the protein levels of keratinocyte-derived cytokine (KC), a murine equivalent of human interleukin 8, was observed in both the liver and kidneys of the TLR-4^{-/-} mice (Liver: $-/-$: 11.35 ± 3.44 pg/ml vs. C: 48.37 ± 17.02 pg/mg, $p=0.093$; Kidney: TLR-4 $-/-$: 25.2 ± 8.23 pg/mg vs. C: 125.58 ± 46.2 pg/mg SE, $p=0.17$).

Conclusions: The absence of TLR-4 improved survival and ameliorated spinal cord injury after TAR in mice. To our knowledge, this is the first time that TLR-4 has been shown to be an important mediator of spinal cord protection in a model of spinal cord ischemia/reperfusion. Further investigation is needed to determine the mechanism by which the absence of TLR-4 confers neurological protection and survival benefit in this model of spinal cord I/R injury.

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RACIAL DISPARITY IN PATIENT PRESENTATION FOR CAROTID ENDARTERECTOMY

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Introduction: It is not known if racial differences in patient outcome can be explained by differences in income between racial groups. It is hypothesized that racial disparity may persist even after controlling for patient income. Differences in patient presentation for carotid endarterectomy (CEA), as a marker of the quality of care that the patients receive in the pre-surgical health care system (in terms of their primary evaluations and workups and/or referrals), are examined to determine whether there is a difference by race, and whether such difference can be explained by differences in patient income.

Methods: Retrospective analysis of 10 years (1994-2003) of Maryland state-wide hospital discharge database and 5 years (1999-2003) of California state-wide hospital discharge database. Patients with International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) procedure code 38.12 (endarterectomy of vessels of the head and neck other than intracranial vessels) in the primary but not in any secondary position, the presence of ICD-9 diagnosis code 433-433.91 (occlusion/stenosis, precerebral artery), and the Diagnosis-Related Group (DRG) 5 (extracranial vascular procedure) were included in the analysis. Symptomatic patients were identified by history of previous stroke (ICD-9 codes 342.xx or 438.xx), TIA (435.xx or 781.4), or amaurosis fugax (362.34 or 368.12).

Results: A total of 23,237 CEA cases were identified in the Maryland data. Among them, 3,526 (15.17%) were symptomatic patients on presentation. African-Americans are found to come from lower-income neighborhoods than White patients (\$52,000 vs \$62,000, $p < 0.01$). However, controlling for differences in income level, African-Americans are still more likely than White patients to present symptomatically (OR 1.61, $p < 0.001$). Furthermore, even among high-income patients (i.e., patients with household income greater than \$60,000), African-Americans are still more likely to present symptomatically (OR 1.70, $p < 0.001$).

Conclusions: African-American patients are more likely than White patients to present symptomatically before their carotid endarterectomy. Such differences cannot be explained by income alone, and persist even in high-income patients. Racial disparity in the pre-surgical health care system that delays the access to surgical care for African-American patients, in terms of primary evaluations and workups and/or referrals, needs to be further examined. Outreach programs specifically targeting African-Americans may be beneficial.

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360-DEGREE FEEDBACK SURVEY TO ASSESS FACULTY COMPETENCY IN A CARDIOTHORACIC SURGERY PRACTICE

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Purpose: The 360-degree feedback or multisource surveys have been utilized in corporate and academic settings to facilitate self-awareness and professional development. We recently developed a 360-degree survey to assess the six competency categories outlined by the ACGME in our residency training program. It has become apparent that resident trainees often incorporate the attitudes, perspectives and behaviors of their faculty mentors and as such, it would seem appropriate to assess these competencies (leadership of patient care team, medical knowledge, inter-personal and communication skills, professionalism, systems-based and practice-based learning, integrity) among the faculty of a program.

Methods: A 46 item survey, specifically tailored for faculty, was distributed to six to ten evaluators of each faculty member including the chairperson, peers, direct report and support staff. Faculty members were allowed to select three evaluators of their own choice and the program chair selected the additional evaluators. The surveys were returned to the Workplace Initiatives Program for evaluation and correlation. A comprehensive report was provided to the participants, which provided overall and individual assessment of performance on the survey.

Results: Eight faculty members participated in the survey. All but one faculty member completed the self-evaluation. Each faculty member received a comprehensive report and a transcript of the responses to the open-ended questions and a summary of data highlighting areas of excellence, areas for improvement and the suggested goals and recommendations. Faculty members as a whole were rated above average in medical knowledge, patient-based learning, patient care and leadership of the patient care team. Some faculty members scored below average in interpersonal and communication skills and professionalism (especially around conflict). These scores were observed in areas commonly identified as suboptimal in residents as well.

Conclusion: We believe that the 360-degree assessment tool provides valuable feedback for faculty members in cardiothoracic surgery. It also underscores our belief that resident physicians may model behavior after that of the faculty and as such, it emphasizes the importance of faculty role models in the education of young cardiothoracic surgeons.

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BRACHIAL AND INFRA-BRACHIAL ARTERIAL RECONSTRUCTION FOR CHRONIC ISCHEMIA

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Objective: Chronic ischemia of the upper extremity requiring surgical revascularization is an uncommon condition. We analyzed modes of presentation, methods of operative repair, and follow-up in all consecutive patients with chronic ischemia of the upper extremity requiring arterial bypass.

Methods: Data prospectively entered into a vascular registry were retrospectively analyzed for all patients undergoing upper extremity arterial bypass from January 1, 1990 to June 30, 2003. Simple thromboembolectomy procedures and bypasses to an outflow target more proximal than the brachial artery were excluded.

Results: Twenty patients were identified. Mean age was 57; eleven (55%) were female. Eight (40%) had diabetes and 5 (25%) had renal insufficiency. Indications included exercise intolerance in 11 patients (55%), tissue loss in 6 (30%), and rest pain in 3 (15%). The etiology of ischemia was atherosclerosis in 7 patients (35%) and complications of iatrogenic or civilian trauma in 13 patients (65%). The brachial artery was used as the inflow in 13 patients (65%), the axillary in 6 (30%), and the ulnar in one patient (5%). Conduits used included the great saphenous vein in 11 (55%), arm vein in 7 (35%), and prosthetic in 2 patients (10%). Outflow targets included the brachial artery in 12 patients (55%), the radial in 5 (25%), and the ulnar in 3 patients (15%). There were no perioperative deaths. There was one (5%) early graft occlusion (within 30 days of surgery). Mean follow-up was 12 months. Mean survival after bypass was 62 months. Patency at 1 and 3 years was 90%. Two patients had associated minor amputations: 1 finger and 1 partial hand amputation. Limb salvage rate was 100%.

Conclusion: Although rare, results for upper extremity bypass are excellent and superior to those reported for lower extremity ischemia. These results may reflect the indications, which differ considerably from those for lower extremity bypass, with the majority being performed for complications of trauma.

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EXTRACORPOREAL PHOTOPHERESIS CAUSES DELETION OF CARDIAC ALLOGRAFT SPECIFIC T CELLS IN VIVO

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Background: Extracorporeal photopheresis (ECP) is an immunomodulatory therapy used for treatment of a variety of diseases. It is proposed that this treatment induces an autologous suppressor response, and it has been shown to have therapeutic benefits in a wide variety of T-cell mediated diseases including cardiac allograft rejection. In this treatment, leukocytes are enriched by leukapheresis, treated with a UV sensitizing agent, 8-methoxypsoralen, and UV irradiated. The treated cells are then infused into the patient. Currently, ECP is involved in clinical trials as an adjunct to chemotherapy to reduce the incidence of rejection in heart transplant patients. Using a mouse model of heart transplantation, we addressed the hypothesis that ECP treatment modulates cardiac allograft rejection by causing deletion of graft specific T-cells.

Methods: Heterotopic cardiac allografts from C57BL/6 mice were transplanted into CBA/CA mice the day after injecting them with CFSE labeled cells from a BM3 TCR transgenic mouse. In the BM3 mice, 80% of CD8+ T cells are specific for the K^b alloantigen expressed by the donor heart. On the day of transplant, the recipient mice were injected with ECP-treated splenocytes from syngenic CBA/Ca mice. The hearts, lymph nodes, and spleens were removed from the recipient mice on post-op days 1, 2, 3, and 5. The cells were then analyzed by flow cytometry for CFSE fluorescence and expression of T cell surface antigens, including the K^b-specific T cell receptor present on the CFSE labeled T cells adoptively transferred from the BM3 mice.

Results: See Table. From days 1 through 5, there is little discernable difference between ECP-treated and non-treated recipients' peripheral lymphoid tissue T cell infiltration. However, in the cardiac allografts of the untreated mice, there is a sharp increase in the amount of total T cell and K^b-specific T cell infiltration. Specifically on day 3, the proportion of CD3+ T cells rose from 2.77% on day 2 to 35.30% by day 3 in the allografts of the untreated mice. In contrast, CD3+ in the ECP treated mice allografts only increased from 4.3% to 9.47%. A similar reduction in graft infiltration of K^b-specific T cells was also observed in treated

mice. This reduction in the proportion of CD3+ cells was reflected in the absolute numbers of T cells recovered from the transplanted hearts. In the ECP treated mice, the number of recovered viable CD3+ cells is reduced by two orders of magnitude.

Time	ECP	Spleen		Lymph Node		Heart	
		%CD3	%Ti98	%CD3	%Ti98	%CD3	%Ti98
1 Day	-	23.52	.07	76.44	.24	8.03	ND
	+	35.17	.15	87.94	.195	2.38	ND
2 Days	-	28.18	.03	80.47	.01	2.77	ND
	+	25.69	.07	73.93	.01	4.30	ND
3 Days	-	28.2	5.04	78.72	0.58	35.03	9.81
	+	29.0	1.12	62.87	0.32	9.47	ND
5 Days	-	25.0	5.43	82.9	2.53	45.33	93.4
	+	27.9	5.79	88.3	0.98	5.46	4.57

Conclusions: Infusion of ECP-treated cells causes deletion of HOST graft-specific T-cells in vivo, resulting in reduced infiltration of graft-specific T cells, CD3+ T cells and CD8+ T cells in cardiac allografts.

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CONSTITUTION

CONSTITUTION OF THE SOCIETY OF BLACK ACADEMIC SURGEONS

ARTICLE I: Designation

The name of the organization shall be the Society of Black Academic Surgeons (SBAS). It shall be incorporated as a non-profit organization and have no capital stock or shareholders. The address of the President will be the official address of the Society.

ARTICLE II: Objective

The paramount objectives of the Society of Black Academic Surgeons shall be supportive of and consistent with the enhancement of the academic surgical community both nationally and internationally. The specific objectives are as follows:

- A. Identify and promote professional and intellectual exchange among surgeons and scientists involved in their related fields.
- B. Promote the participation of minority surgeons and scientists in the activities of all academic surgical organizations.
- C. Stimulate and assist government, private industry and voluntary organizations to develop and promote programs to increase the participation of minority surgeons in the academic community.
- D. Encourage and assist minority surgeons to conduct original research in both the basic and clinical sciences.
- E. Support and strengthen the surgical section programs of the National Medical Association.

ARTICLE III: Members

Active members will be designated as Fellows of the Society of Black Academic Surgeons and will be comprised of reputable surgeons. All Fellows will be elected to membership according to the Constitution and Bylaws. Termination of a member by resignation, death, or any other manner will end all rights and privileges in the Society. None of the assets or privileges will be transferable to any representative of a member's estate.

ARTICLE IV: Officers/Council

The Officers of the Society shall be President, President-Elect, Secretary and Treasurer. The President and President-Elect shall be elected for a one-year term; the President-Elect shall automatically become President. The Secretary and the Treasurer shall be elected for three-year terms. This slate of officers, along with two Fellows (appointed by the President) will be designated as the Executive Council.

ARTICLE V: Organization Structure

- A. The Society's organizational structure will consist of General Membership, Officers, Executive Council, and Standing Committees. The span of authority, rights and privileges shall be based on the Constitution and Bylaws.
- B. The duties, powers and regulations governing the Society's organizational structure shall be defined and delineated in the Society's Bylaws.

ARTICLE VI: Meetings

- A. The Society shall hold an annual scientific and business meeting, the time and place determined by the Executive Council at least two years in advance of the meeting. Only members of the Society may attend the business meeting.

ARTICLE VII: Rules

The conduct of all Society meetings including those of the Executive Council shall be governed by the Bylaws of the Society and Robert's Rules of Order.

ARTICLE VIII: Governance

- | | |
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| Section 1 | The Society shall be governed by this Constitution and Bylaws, the latter document to provide specific direction for the organization, administration and services of the Society. |
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CONSTITUTION OF THE SOCIETY OF BLACK ACADEMIC SURGEONS

(continued)

Section 2 The Society's Constitution and Bylaws shall be consistent with provisions and content of any organizational charter or certificate of incorporation the Society may propose and/or execute.

ARTICLE IX: Certificate of Incorporation

Section 1 The Society may propose and execute an organizational charter or certificate of incorporation in accordance with all local, state and federal (U.S.) regulations, codes and laws.

Section 2 The certificate of incorporation shall not vitiate any provision of this Constitution or the Society's Bylaws, unless a court of competent jurisdiction expressly rules, orders or directs otherwise. If any such provision or the certificate, in whole or part, is held to be unlawful, only the unlawful provision or certificate will be null and void. The remaining provisions and/or certificate, in whole or part, will continue in effect as valid.

Section 3 The certificate of incorporation shall not govern the application and administration of the Constitution or the Society's Bylaws.

Section 4 Notwithstanding any other provisions of these articles, the organization is organized exclusively for one or more of the purposes as specified in Section 501C (3) of the Internal Revenue Code of 1954, and shall not carry on any activities not permitted to be carried on by an organization exempt from Federal income tax under IRC 501C(3) or corresponding provisions of any subsequent Federal tax laws.

Section 5 No part of the net earnings of the organization shall inure to the benefit of a member or any private

individual (except that reasonable compensation may be paid for services rendered to or for the organization), and no member of the organization or any private individual shall be entitled to share in the distribution of any of the organization's assets on dissolution of the organization.

Section 6 No substantial part of the activities of the organization shall be carrying on propaganda, or otherwise attempting to influence legislation (except as otherwise provided by IRC 501C(h) and does not participate in, or intervene in (including the publication or distribution of statements), and political campaign on behalf of any candidate for public office.

Section 7 In the event of dissolution, all of the remaining assets and property of the organization shall after payment of necessary expenses thereof be distributed to such organizations as shall qualify under section 501(c)(3) of the Internal Revenue Code of 1986 and approved by the Executive Committee.

Section 8 In any taxable year in which the corporation is a private foundation as described in IRC 509(a), the organization shall distribute its income for said period at such time and manner as not to subject it to tax under IRC 4942, and the organization shall not (a) engage in any act of self-dealing as defined in IRC 4941(d), retain any excess business holdings as defined in IRC 4943(c), (b) make any investments in such a manner as to subject the organization to tax under IRC 4944, or C, make any taxable expenditures as defined in IRC 4945(d) or corresponding provisions of any subsequent Federal tax laws.

ARTICLE X: Funds and Expense

Funds for the Society may be raised by approved dues and/or in any manner approved initially by the Executive Committee and the organization. Funds may be appropriated by the Executive Council to

CONSTITUTION OF THE SOCIETY OF BLACK ACADEMIC SURGEONS

(continued)

defray the expense of the Society to carry out the necessary functions, and for any other purpose approved by the Council; provided, however, that no funds or assets shall be used to inappropriately benefit one member of the unit.

ARTICLE XI: Amendments

This Society, at any annual business meeting of the Fellows, may amend any Article of this Constitution by a two-thirds majority of the Fellows present, provided that a copy of the proposed Amendment has been furnished to each active Fellow at least thirty days in advance of the meeting.

ARTICLE XII: Effective Date

These revised Bylaws shall take effect immediately upon acceptance by a simple majority of the membership and extend indefinitely, subject to alteration, amendment or repeal in whole or part, as specifically provided in the Constitution.

BYLAWS: SOCIETY OF BLACK ACADEMIC SURGEONS

Section 1 Annual Meeting

The Society of Black Academic Surgeons shall meet annually at such time and place as designated by the Executive Council.

Section 2 Quorum

The Fellows present shall constitute a quorum for business. All questions before the Society shall be determined by the vote of the majority of those present at any regular business meeting.

Section 3 Fiscal Year

The fiscal year shall begin on January first. The annual dues of each member shall be determined by the Executive Council with approval of the membership, payable on January first of each year. Each member of the Society who reaches the age of sixty-five years shall automatically have his dues rescinded.

Section 4 Parliamentary Procedure

Robert's Rules of Order shall govern the sessions of the Society.

Section 5 Membership

A. Eligibility

1. An individual who occupies a faculty position in a university department of surgery or its affiliated hospitals.
2. An individual who occupies a faculty position in a free-standing surgical residency program.
3. An investigator or teacher in an academic department of surgery or an ACGME-approved surgery program.
4. An individual in a surgical specialty (Neurosurgery, Orthopedics, Urology, Otorhinolaryngology, Plastic and Reconstructive) shall be eligible for membership.

B. Membership Certification

Membership in the Society shall include the following categories: Active, Senior, Associate and Honorary.

1. Active Fellow: Any person who is a Doctor of Medicine (M.D.) or the equivalent, a Doctor of Philosophy (Ph.D.) Degree or the equivalent who shares an interest in the purpose of the Society and is approved by the Fellowship Committee. Only active members have the right to vote and hold office.
2. Senior Fellow: Any active Fellow upon reaching the age of seventy years shall become a Senior Fellow. Senior Fellows are exempt from paying dues, and shall continue to vote, but shall not have the privilege of holding office.

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(continued)

3. Associate Fellow: Any surgical resident in good standing in an ACGME-approved residency program who desires to pursue an academic surgical career.
4. Honorary Fellow: Any person who is a Doctor of Medicine (M.D.) (or equivalent) or Doctor of Philosophy (Ph.D.) degree (or equivalent) and has distinguished himself/herself by outstanding achievement and dedication to the objectives of the Society. Honorary Fellows shall pay no due or initiation fees and may not vote or hold elected office.

Section 6 Responsibilities of the Officers

- A. It shall be the duty of the President to (1) preside at all meetings of the Society, (2) give the deciding vote, (3) ensure that Robert's Rules of Order and decorum are properly enforced in all deliberations of the Society, and (4) sign the approved proceedings of each meeting.
- B. In the absence of the President, the President-Elect shall preside, and in his absence the Secretary.
- C. It shall be the duty of the Secretary to (1) keep a true and correct record of the proceedings of the Meetings, (2) preserve all books, papers, and articles belonging to the Society, (3) keep an account of the Society with its Fellows, and (4) keep a register of the Fellows with the dates of their admission and places of residence. The Secretary shall report unfinished business at previous meetings requiring action, and attend to such other business as the Society may direct. The Secretary shall assist with the correspondence of the Society.
- D. It shall be the duty of the Treasurer to collect the dues of the Society and make disbursements for expenses. The Treasurer shall present an annual report of the financial condition of the

Society. The accounts of the Treasurer shall be audited once yearly by a committee appointed by the President.

Section 7 Vacancies, Resignations and Removal from Membership

A. Vacancies

Vacancies occurring in the offices of the Society, other than that of the President, shall be filled by appointment by the President until the next meeting. The President shall appoint members to all Committees.

B. Resignations

Any Fellow may resign from the Society by delivering a written resignation to the President or Secretary.

C. Expulsions

The removal of a Fellow from the society shall be based on gross negligence or poor character as determined by the Executive Council and a majority of the full membership.



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- 1991 Harvard University, Boston, MA
- 1993 UC Davis-East Bay (Meeting held in Napa Valley, CA)
- 1994 UTMB, Galveston, TX
- 1995 University of North Carolina, Chapel Hill, NC
- 1996 University of Colorado, Denver, CO
- 1997 State University of New York, Buffalo, NY
- 1998 Howard University College of Medicine, Washington, DC
- 1999 University of Louisville, Louisville, KY
- 2000 Charles R. Drew University, Los Angeles, CA
- 2001 Harvard University, Boston, MA
- 2002 Morehouse School of Medicine, Atlanta, GA
- 2003 University of Alabama at Birmingham, Birmingham, AL
- 2004 Howard University, Washington, DC
- 2005 University of Pittsburgh, Pittsburgh, PA

FUTURE SBAS MEETINGS

- 2007 Chicago - Rush University Medical Center, April 12-15
- 2008 Cleveland - Cleveland Clinic
- 2009 Seattle - University of Washington