

**THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY**



69th Annual Meeting

**Ritz-Carlton Lake Las Vegas
Henderson, Nevada**

October 31 – November 3, 2007



American
Association of
Neurological
Surgeons

Jointly Sponsored by AANS



FUTURE MEETINGS

2008

**September 10–13
Enchantment Resort
Sedona, Arizona**

2009

**November 4-7
The Breakers
Palm Beach, Florida**

Mark your calendars now!

GENERAL INFORMATION

HOTEL INFORMATION

The Ritz-Carlton, Lake Las Vegas
1610 Lake Las Vegas Parkway
Henderson, Nevada 89011

Telephone: 702-567-4700
Facsimile: 702-567-4777

MEETING REGISTRATION

Wednesday, October 31	Salons 3&4 Foyer	12:00 PM – 6:30 PM
Thursday, November 1	Salons 1&2 Foyer	6:00 AM – 11:30 AM
Friday, November 2	Salons 1&2 Foyer	6:00 AM – 11:30 AM
Saturday, November 3	Salons 1&2 Foyer	6:00 AM – 11:30 AM

PROGRAM SUMMARY

WEDNESDAY, OCTOBER 31

EVENT	TIME	LOCATION
Registration	12:00 PM – 6:30 PM	Salons 3&4 Foyer
Executive Committee Meeting	2:00 PM – 5:00 PM	Deserto
OPENING RECEPTION		
Cocktail Dinner <i>CASUAL ATTIRE</i>	6:30 PM – 8:30 PM	Florentine Gardens <i>(Back up: Salons 3&4)</i>

THURSDAY, NOVEMBER 1

EVENT	TIME	LOCATION
Registration	6:00 AM – 11:30 AM	Salons 1&2 Foyer

EVENT	TIME	LOCATION
Business Breakfast <i>MEMBERS ONLY</i>	6:00 AM – 7:00 AM	Salons 3&4
Spouse & Guest Breakfast	6:00 AM – 9:00 AM	Tuscany & Courtyard
Scientific Session	7:00 AM – 11:30 AM	Salons 1&2
Spouse Program Jane Drake <i>Writing Between the Lines: Engaging Children in Non-fiction</i> Delia Ray (Howard) <i>A Spoonful of Sugar: Bringing History Alive in Children's Literature</i>	9:00 AM – 10:30 AM	Tuscany

AFTERNOON ACTIVITIES (Require Prior Registration & Payment)

Golf (Tournament)	12:30 PM Shotgun Start	Reflection Bay
Hoover Dam Comedy Tour	1:00 PM – 5:00 PM	Meet in Lobby 10 minutes prior
Lake Las Vegas Guided Hike	1:30 PM – 4:00 PM	Meet in Lobby 10 minutes prior
Painting the Scenic Outdoors	1:30 PM – 4:30 PM	Meet in Lobby 10 minutes prior

Above may be cancelled

EVENING

Casino Night <i>GAMING ATTIRE</i>	6:30 PM – 10:00 PM	Salons 2–4
<i>Reception</i>	<i>6:30 PM – 7:30 PM</i>	
<i>Dinner</i>	<i>7:30 PM – 9:30 PM</i>	
<i>Gaming continues until 10:00 PM</i>		

FRIDAY, NOVEMBER 2

Registration	6:00 AM – 11:30 AM	Salons 1&2 Foyer
Business Breakfast <i>MEMBERS ONLY</i>	6:00 AM – 7:00 AM	Salons 3&4
Spouse & Guest Breakfast	6:00 AM – 9:00 AM	Tuscany & Courtyard

EVENT	TIME	LOCATION
Scientific Session	7:00 AM – 11:30 AM	Salons 1&2
Spouse Program <i>Book Discussion: <u>Deafening</u> by Francis Itani</i>	9:30 AM – 10:30 AM	Tuscany
Presidential Introduction	10:30 AM – 10:45 AM	Salons 1&2
Presidential Address	10:45 AM – 11:30 AM	Salons 1&2
AFTERNOON ACTIVITIES (Require Prior Registration & Payment)		
Golf	12:30 PM Shotgun Start	The Falls
Guided Kayaking	1:30 PM – 4:00 PM	Meet in Lobby 10 minutes prior
Fly Fishing	1:30 PM – 4:30 PM	Meet in Lobby 10 minutes prior
Cooking & Cocktails with the Chef	1:30 PM – 4:30 PM	Meet in Lobby 10 minutes prior
EVENING		
Presidential Reception	6:30 PM – 7:30 PM	Salons 1&2 Foyer
Dinner, Entertainment, & Dancing	7:30 PM – 10:30 PM	Salons 2–4
<i>OPTIONAL BLACK TIE</i>		

SATURDAY, NOVEMBER 3

Registration	6:00 AM – 11:30 AM	Salons 1&2 Foyer
Breakfast for Members, Spouses, & Guests	6:00 AM – 9:00 AM	Salon 2
Scientific Session	7:00 AM – 11:30 AM	Salon 1
Golf	Tee times from 12:20PM	Reflection Bay
Farewell Gathering <i>CASUAL ATTIRE</i>	5:00 PM – 6:30 PM	Presidential Suite

2007 OFFICERS

PRESIDENT

Richard B. Morawetz

PRESIDENT-ELECT

Robert F. Spetzler

VICE PRESIDENT

Griffith R. Harsh, IV

SECRETARY

Ralph G. Dacey, Jr.

TREASURER

James T. Rutka

EXECUTIVE COMMITTEE

Richard B. Morawetz

Robert F. Spetzler

Griffith R. Harsh, IV

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David G. Piepgras

James T. Rutka

L. Nelson Hopkins, III

Steven Papadopoulos

HISTORIAN

David G. Piepgras

ACADEMY COMMITTEES 2007

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Subcommittee on Corresponding

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Richard Morawetz
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Robert Harbaugh
Jim Markert

Round Robin Editor:

Ralph Dacey, Jr.

Local Arrangements:

L. Nelson Hopkins

AANS Joint Sponsorship Education Representative:

James Markert

WFNS Delegates:

Martin Camins - Senior
Volker Sonntag - Alternate

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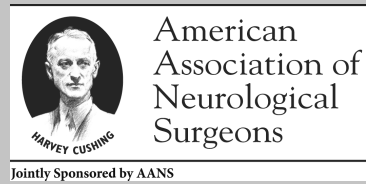
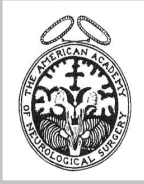
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in support of the

2007 Annual Meeting of the

American Academy of Neurological Surgery

American Academy of Neurological Surgery



SCIENTIFIC PROGRAM AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

2007 LEARNING OBJECTIVES

Learning Objectives

Upon completion of this CME activity, participants should be able to:

Evaluate current methodologies and outcomes of neurosurgical trauma management.

Discuss new applications of neuro-stimulation including psychiatric indications.

Critique the value of surgical and non-surgical options presented in the scientific papers.

Evaluate the relevance of research methodologies and the findings and potential usefulness in the practice of neurosurgical topics presented.

Accreditation/Continuing Medical Education

This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the

AANS and American Academy of Neurological Surgery. The AANS is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The AANS designates this educational activity for a maximum of 12 *AMA PRA Category 1 Credits™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

DISCLOSURE INFORMATION

The AANS and The American Academy of Neurological Surgery control the content and production of this CME activity and attempt to assure the presentation of balanced, objective information. In accord with the Standards for Commercial Support established by the Accreditation Council for Continuing Medical Education, speakers and paper presenters are asked to disclose any relationship they or their co-authors have with commercial companies that may relate to the content of their lecture.

Speakers and paper presenters/authors who have disclosed a relationship* with commercial companies whose products may have a relevance to their presentation are listed below.

<u>Name</u>	<u>Conflict of Interest</u>	<u>Company</u>
Aagaard-Kienitz, B	Industrial Grant Support Consultant Fee/Advisory Board	Micrus Micrus, Microvention/Terumo
Abdulrauf, S	Consultant Fee	Scanlan Inc
Albuquerque, FC	Consultant Fee/Advisory Board	Micrus Endovascular
Ashby, L	Consultant Fee Honorarium Speaker's Bureau	MGI Pharma MGI Pharma MGI Pharma, Schering Plough
Asher, A	Industrial Grant Support	MGI Pharma
Benzel, EC	Stock/Shareholder	DePuy, Orthomems, Abbott

Berger, MS	University Grant/Research Support	NIH
Brem, H	University Grant/Research Support Industrial Grant Support	NIH Protherics
Chiocca, EA	Consultant Fee	Ceregene Inc; Oxford Biomedica Inc
Colman, H	Honorarium	Investigator Advisory Board for Schering Plough, MGI Pharma (No conflict with presentation)
Etu, JJ	University Grant/Research Support	Dept of Anesthesiology-Columbia
Fiorella, D	Industrial Grant Support Honorarium	Boston Scientific Boston Scientific
Haacke, EM	University Grant/Research Support Industrial Grant Support	State of Michigan, Loma Linda University, Henry Ford Hospital, Wayne State University Siemens Medical Solutions
Haid, RW	Other Financial or Material Support	Medtronic
Hanley, D	University Grant/Research Support Industrial Grant Support Consultant Fee Fiduciary Position	NINDS, FDA Genentech Novo Nordisk National Stroke, NMT Medical
Harbaugh, R	University Grant/Research Support Industrial Grant Support Consultant Fee Stock or Shareholder	NIH ROI NSO49135, Commonwealth of PA Codman, Medtronic, Integra Neuroscience Micromechatronics, Inc Cortex Pharmaceuticals, MedCool, Inc

Hopkins, LN	Industrial Grant Support	Boston Scientific, Cordis, Micrus
	Consultant Fee/Advisory Board	Abbott, Access Closure, Bard, Boston Scientific, Cordis, Market Rx, Micrus
	Stock or Shareholder	APW Holding; Boston Scientific; Micrus
	Honorarium	Bard, Boston Scientific, Cordis, Medsn
Iskandar, BJ	University Grant/Research Support	Howard Hughes Med Investigator Grant, Amer College of Surgeons Faculty Res Fellowship, NIH/NICHHD RO1
Joshi, S	University Grant/Research Support	Dept of Anesthesiology-Columbia
Kirsch, WM	University Grant/Research Support	NIH
Lavine, SD	Consultant Fee	Cordis Endovascular, Microvention Terumo
Levy, AP	Consultant Fee	Altean Inc
Levy, EI	Industrial Grant Support	Boston Scientific
	Honorarium	Boston Scientific, Cordis
	Other Financial or Material Support	Boston Scientific-Wingspan devices; Abbott Vascular and ev3-funding for carotid training; Zimmer Spine-patent royalties
Liotta, LA	University Grant/Research Support	Perkin Elmer
	Stock or Shareholder	Theranostics Health
	Other Financial or Material Support	University patent royalties—currently none
Lozano, AM	Industrial Grant Support	Medtronic
Lunsford, LD	Consultant Fee	AB Elekta
	Stock or Shareholder	AB Elekta

Mayberg, H	University Grant/Research Support Consultant Fee Other Financial or Material Support	NIMH, Dana Foundation, Stanley Foundation, Woodruff Foundation. ANS, Inc. Licensing Fees, intellectual property, ANS, Inc
McDermott, MW	Other Financial or Material Support	Patent application for device with Integra Neuroscience
McDougall, CG	Consultant Fee/Advisory Board Honorarium	Cardiomind, Cordis Endovascular, Gore & Company, MTL/ev3. Boston Scientific
Moseley, M	University Grant/Research Support	NIH NINDS, NCI
Mummaneni, PV	University Grant/Research Support Consultant Fee	Medtronic, Depuy Spine Medtronic, Depuy Spine
Niemann, DB	Industrial Grant Support Consultant Fee/Advisory Board	ev3 Boston Scientific, ev3, Cordis;Micrus
Niranjan, A	Industrial Grant Support Other Financial or Material Support	AB Elekta Consultant with Elekta, Inc
Olivi, A	Speaker Bureau	MGI Pharma
Petersen, F	Stock/Shareholder Fiduciary Position Other Financial or Material Support	Amphastar Pharmaceuticals, Rancho Cucamonga, CA City of Loma Linda, CA (council member); Omnitrans (public transportation-member, board of directors) Member, board of directors, Amphastar Pharmaceuticals, Rancho Cucamonga, CA
Piegras, DG	University Grant/Research Support	NIH. ISUIA Steering Committee

Prados, MD	Industrial Grant Support	Clinical research grants: Genentech, Inc, Eli Lilly, NeoPharm, Inc
Pride, GL, Jr.	Honorarium Employee Any Industry	Boston Scientific ev3 Neurovascular (clinical proctor)
Purdy, PD	Honorarium Other Financial or Material Support	Cordis Cordis-royalties
Rasmussen, PA	Industrial Grant Support Stock or Sharehold Honorarium	Boston Scientific, ev3 Chestnut Medical Boston Scientific; Micrus
Rodts, GE	University Grant/Research Support Consultant Fee Other Financial or Material Support	Medtronic Medtronic Medtronic
Russell, S	Other Financial or Material Support	Intellectual property rights & inventor measles virus as a cancer therapy
Scott, EW	Stock or Shareholder Fiduciary Position	RegenMed Inc RegenMed Corp, Chief Scientific Officer
Spetzler, RF	Consultant Fee Stock or Shareholder Other Financial or Material Support	Zeiss. Anspach Synergetics Allegiance
Steinberg, GK	University Grant/Research Support Stock or Shareholder	NIH NINDS Stem Cells, Inc
Turk, AS	Industry Grant Support Consultant Fee	Biomerix Corp.; Boston Scientific; General Electric (GE) Biomerix; Boston Scientific; GE
Uchida, N	Employee Any Industry	StemCells Inc., Palo Alto, CA
Vogelbaum, MA	Consultant Fee Stock or Shareholder Honorarium Speaker's Bureau	NeoPharm Johnson & Johnson MGI Pharma, Schering Plough Schering Plough

Wang, M	University Grant/Research Support	Dept of Anesthesiology-Columbia
Weissman, IL	Consultant Fee Stock or Shareholder Employee Any Industry Other Financial or Material Support	Cellerant Therapeutics Amgen, Cellerant, Stem Cells Inc Stanford University Cellerant Therapeutics
Welch, BG	Industrial Grant Support	Boston Scientific
Yu, JS	University Grant/Research Support Industrial Grant Support: Consultant Fee Stock or Shareholder Fiduciary Position	NIH, California Institute for Regenerative Medicine MGI Pharma MGI Pharma Immunocellular Therapeutics Ltd Chair, Immunocellular Therapeutics, Ltd.

*Relationship refers to receipt of royalties, consultancy, funding by research grant, receiving honoraria for educational services elsewhere, or any other relationship to a commercial company that provides sufficient reason for disclosure.

§§§§§

Speakers and paper presenters/authors who have reported they do not have any relationships with commercial companies:

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Wiebers, DO
Wilmer, EN
Woo, H
Xi, G
Xu, Y
Zhan, X
Zipfel, G

SCIENTIFIC PROGRAM

**American Academy of Neurological Surgery Annual Meeting
Ritz-Carlton at Lake Las Vegas, Henderson, Nevada
October 31 – November 3, 2007**

THURSDAY, NOVEMBER 1

Moderator: Robert Harbaugh

7:00 – 7:45

POINT/COUNTERPOINT

**Neurosurgical Emergency
Coverage: Is There a Problem?
What Can We Do About It?**

7:00 – 7:20

POINT

Alex B. Valadka, MD, FACS

7:20 – 7:40

COUNTERPOINT

John Fildes, MD, FACS

7:40 – 7:45

Audience Participation

PAPER PRESENTATIONS

7:45 – 8:00

**Haptoglobin 2-2 Genotype
Predisposes to Chronic Vasospasm
after Experimental Subarachnoid
Hemorrhage**

*Rafael J. Tamargo, MD, FACS,
Andrew P. Levy, MD, PhD, Kaisorn
L. Chaichana, BS, Rachel Miller-
Lotan, PhD, and Sophia Shakur, BS*

8:00 – 8:15

**Academic Neurosurgery in
Developing Countries an
Achievable Responsibility**

Robert Dempsey, MD

8:15 – 8:30

Genetically Engineered T-cells for Treatment of Human Glioblastoma

Bob S. Carter M., PhD

8:30 – 8:45

MAGE and GAGE are Expressed in Medulloblastoma and Modulate Resistance to Chemotherapy

C. Kasuga, MD Taylor, C. Smith, and *James T. Rutka MD, PhD*

8:45 – 9:00

Tumor Formation by Malignant Human Glioma Cells Requires Activated Stat3

Michael A. Vogelbaum, MD, Atreyi Dasgupta, Baisakhi Raychaudhuri, Talat Haqqi, Richard Prayson, Erwin G. Van Meir, and S. Jaharul Haque

9:00 – 9:30

Beverage Break

9:30 – 9:45

Tentorial Dural Arteriovenous Fistulae: Operative Strategies and Microsurgical Results for Six Types

Michael T. Lawton, MD, Rene O. Sanchez-Mejia, Van V. Halbach

9:45 – 10:00

Physical Exercise and Ischemic Tolerance in Stroke

David F. Jimenez, MD, FACS

10:00 – 10:15

**Bone-Marrow Derived
Hematopoietic Stem Cells Migrate
to Sites of Aneurysm Formation by
an iNOS-Mediated but not eNOS-
Mediated Process in a Novel
Murine Saccular Aneurysm Model**

Brian L. Hoh, MD, Gregory J.
Velat, MD, Erin N. Wilmer, BA,
Edward W. Scott, PhD

10:15 – 10:30

**Influence of Patient Age and
Stenosis Location on Wingspan In-
Stent Restenosis (ISR)**

A.S. Turk, DO, *Elad I. Levy, MD*,
F.C. Albuquerque, MD, G.L. Pride Jr,
MD, H. Woo, MD, B.G. Welch, MD,
D.B. Niemann, MD, P.D. Purdy, MD,
B. Aagaard-Kienitz, MD,
P.A. Rasmussen, MD, L.N. Hopkins,
MD, T.J. Masaryk, MD, C.G.
McDougall, MD, D. Fiorella, MD, PhD

10:30 – 10:45

Secondary Stroke Prevention

Robert F. Spetzler, MD

10:45 – 11:00

**Enhancement of Brain Tumor
Immunotherapy by RNAi
mediated Down-regulation of Fas-
ligand (FasL)**

Alessandro Olivi, MD, Timothy
Jansen, John Laterra, MD, PhD

11:00 – 11:15

**Intracranial Hemorrhage
Following Treatment of
Unruptured Intracranial
Aneurysms**

ISUIA Investigators including
D.O. Wiebers, MD, J.C. Torner, MD,
R.D. Brown, Jr., MD, I. Meissner,
MD, and *David G. Piegras, MD*

11:15 – 11:30

**Effects of Protease-Activated
Receptor-1 on Thrombin-Induced
Glioma Cell Proliferation and
Vascular Endothelial Growth
Factor Secretion and Cell Invasion**

Karin M. Muraszko, MD, Ya Hua,
MD, Ying Xu, MD, Wenquan Liu, MD,
Richard F. Keep, PhD, Guohua Xi, MD

FRIDAY, NOVEMBER 2

Moderator: Henry Brem

7:00 - 7:30

GUEST SPEAKER
**DBS to Treat Depression:
Redefining Psychosurgery**

Helen S. Mayberg, MD
Emory University School of
Medicine

7:30 - 8:00

GUEST SPEAKER
**The Glioblastoma Genome:
Revealing Secrets for Improved
Therapy**

Gregory J. Riggins, MD, PhD
Johns Hopkins University School of
Medicine

PAPER PRESENTATIONS

8:00 - 8:15

Deep Brain Stimulation Increases Adult Hippocampal Neurogenesis

Andres M. Lozano, MD, PhD,
H. Toda, C. Hamani, A.P. Fawcett,
W.D. Hutchison

8:15 – 8:30

Vaccination with Tumor Lysate-Pulsed Dendritic Cells Elicits Correlated Immune and Survival Response in Patients with Glioblastoma Multiforme (GBM)

Keith L. Black, MD, Mia Wagenberg, Xiao-xue Zhang, Gentao Liu, Elina Mindlin, Laura Blascewicz, Chiayi Chen, Samuel Pepkowitz, Dennis Goldfinger, Hiushan Ng, Dwain Irvin, John S. Yu, Christopher J. Wheeler

8:30 – 8:45

Temozolomide (TMZ) can be Safely Administered in the Immediate Postoperative Period After Tumor Resection and Gliadel Wafer Placement in Patients with Newly Diagnosed Gliomas: Results of a Prospective, Multi-institutional, Phase I/II Trial

Anthony L. Asher, MD, FACS, Stuart Burri, MD, Wendy Brick, MD, Lynn Ashby, MD, Kevin Judy, MD

8:45 – 9:00

Lateral Hypothalamic Radiosurgery for Obesity

L. Dade Lunsford, MD, FACS and Ajay Niranjana, MCh

- 9:00 - 9:30 Beverage Break
- 9:30 - 9:45 **Facial Nerve Outcomes in Vestibular Schwannoma Surgery Following Failed Radiosurgery or Failed Microsurgery**
Michael J. Link, MD
- 9:45 - 10:00 **The Development of Oncolytic HSV-1 as a Potential Therapy for Malignant Glioma**
James M. Markert, MD, FACS
- 10:00 - 10:15 **ACADEMY AWARD RUNNER UP**
An siRNA Screen Revealed that PARP1 and CDK7 Inhibition Selectively Radiosensitized Glioma Cells Over-Expressing EGFRvIII
Clark C. Chen, MD, PhD, Richard Kennedy, MD, PhD, Alan D'Andrea, MD
- 10:15 - 10:30 **ACADEMY AWARD PAPER**
A Combined Epigenetic and Genetic Genome-Wide Screen Identifies SPINT2 as a Novel Tumour Suppressor Gene in Medulloblastoma
Paul N. Kongkham, MD, Michael D. Taylor, MD, PhD, James T. Rutka MD, PhD

10:30 - 10:45

PRESIDENTIAL INTRODUCTION
Griffith R. Harsh, IV, MD

10:45 - 11:30

PRESIDENTIAL ADDRESS
“Water”
Richard B. Morawetz, MD

SATURDAY, NOVEMBER 3

Moderator: James Markert

PAPER PRESENTATIONS

7:00 – 7:15

**Neurosurgery and Pay for
Performance**

Robert Harbaugh, MD

7:15 – 7:30

**Community Equipose and
Individual Surgeon Equipose in
Designing a Feasible Randomized
Clinical Trial**

***Fred G. Barker MD, Jean-Valery
Coumans MD, Edward C. Benzel
MD, Lauren Stabile BS, Zoher
Ghogawala, MD***

7:30 – 7:45

**Aggressive Correction of Cervical
Kyphotic Deformity Results in
Improved Neurological Outcomes**

***Praveen V. Mummaneni, MD,
Sanjay Dhall, MD, Regis W. Haid,
MD, Gerald E. Rodts, MD***

7:45 – 8:00

High-Resolution MRI Virtual Endoscopy for Preoperative Visualization of Neurovascular Anatomy in Trigeminal Neuralgia

Kim Burchiel, MD, and
Jonathan Miller, MD

8:00 – 8:15

A Progressive Increase in Brain Microhemorrhages Correlates With Sporadic Late-Onset Dementia Development

Wolff M. Kirsch, MD, W. Baqai, Mc Auley, J.P. Larsen, S. Bhaskerrao, W. Britt, III, F. Petersen, C. Dickson, D. Kido, M. Ayaz, E.M. Haacke, L. Liotta, H. Vinters

8:15 – 8:30

Modulation of Tumor Microenvironment to Enhance Oncolytic Virus Therapy

E. Antonio Chiocca, MD

8:30 – 8:45

Integrated Medical Learning: A New Concept for Neurosurgical Education

Douglas Kondziolka, MD, and
Anthony Asher MD

8:45 – 9:00

Automated, Compliant, High-Flow Common Carotid to Middle Cerebral Artery Bypass

Ralph G. Dacey, Jr., MD, Greg Zipfel, MD, William Ashley, MD, PhD., Michael Chicoine, MD, and Michael Reinert, MD

9:00 – 9:30

Beverage Break

9:30 – 9:45

**Neural Stem Cell Transplantation
for Stroke**

Gary K. Steinberg, MD, PhD,

Tonya M. Bliss, PhD, Raphael
Guzman, MD, Marcel Daadi, PhD,
Nobuko Uchida, PhD, Irving L.
Weissman, MD, Ben Barres, MD,
PhD, Michael Moseley, PhD, Theo
Palmer, PhD

9:45 – 10:00

**Bone Marrow-Derived
Mesenchymal Stem Cells are
Recruited to and Alter the Growth
of Human Gliomas**

Frederick F. Lang, MD, Kenneth

Aldape, Raymond Sawaya, Howard
Colman

10:00 – 10:15

**Thrombolysis for Intraventricular
Hemorrhage: Results of Phase II
Trials and Plans for Phase III**

Issam Awad, MD, and Daniel

Hanley, MD, for the CLEAR IVH
Investigators

10:15 – 10:30

The Role of Extent of Resection in the Long-Term Outcome of Low-Grade Hemispheric Gliomas

Justin S. Smith, MD, PhD, Edward F. Chang, MD, Kathleen R. Lamborn, PhD, Susan M. Chang, MD, Michael D. Prados, MD, Soonmee Cha, MD, Tarik Tihan, MD, Scott Vandenberg, MD, Michael W. McDermott, MD, Mitchel S. Berger, MD

10:30 – 10:45

High-Flow Extracranial to Intracranial (HF-EC-IC) Bypass for Giant Transitional Cavernous-Clinoidal Internal Carotid Artery (ICA) Aneurysms: Assessment of Surgical and Long Term Functional Outcomes

Saleem Abdulrauf, MD, Sonia Teufack, BS, Jeroen Coppens, MD, Raul Olivera, MD, Dana Oliver, MPH

10:45 – 11:00

Computer Numerical Control Machine Tool for Automated Drilling of the Skull Base

William T. Couldwell, MD, J.D. MacDonald, A.K. Balaji, C.L.Thomas

11:00 – 11:15

Intra-Arterial Calcium Channel Blockade for Cerebral Vasospasm, a Comparison of Intra-carotid Nicardipine and Verapamil: Augmentation of Cerebral Blood Flow and Reversal of Endothelin-1 Induced Vasospasm

Sean D. Lavine, MD, Mei Wang, MPH, Joshua J. Etu, BA, Philip M. Meyers, MD Shailendra Joshi, MD

11:15 – 11:30

Understanding the Role of the Folate Pathway in Repair of the Injured CNS

Bermans J. Iskandar, MD

THURSDAY PROGRAM

THURSDAY, NOVEMBER 1

7:00 – 7:20 AM
7:20 – 7:40 AM

POINT: Alex B. Valadka, MD
COUNTER POINT: John Fildes, MD, FACS

Neurosurgical Emergency Coverage: Is There a Problem? What Can We Do About It?

*Alex B. Valadka, MD, FACS and
John Fildes, MD, FACS*

Is there a problem?

No: Without question, there are parts of this country with unsatisfactory coverage for neurosurgical emergencies. Many of these regions have major holes in their entire emergency medical care systems, not just in neurosurgery. Because these problematic areas have received a disproportionately large share of attention in discussions about the neurosurgery workforce, the overall extent of the problem may be overstated. Survey data indicate that 93% of neurosurgeons participate in their local emergency care systems, and more than 50% take call at least two or three times per week.

Yes: Although most parts of this country enjoy ready access to emergency care (including neurosurgical emergencies), there are many places in which this type of care is unavailable. Survey data support these anecdotal reports. Most hospitals report difficulty in securing subspecialty coverage, and neurosurgery is at or near the top of those lists. This problem is getting worse. Most neurosurgeons may participate in their emergency call systems, but the sheer volume of emergency work seems to overwhelm the available neurosurgeons.

What can we do about it?

Neurosurgeon: The system is not efficient in its use of available resources. Emergency care must be regionalized, especially for scarce specialties like neurosurgery. EMTALA should be modified to facilitate regionalization. Tort reform and appropriate

compensation would go a long way towards eliminating disincentives for participation. It is even possible to structure emergency care in a way that makes it a worthwhile and attractive practice option. The numbers of applicants being accepted into neurosurgery training programs is increasing, and the eventual growth in the neurosurgery workforce will help to offset this problem.

Trauma surgeon: These solutions are important, but in many parts of the country, they are not enough. Many published reports describe good results when neurotrauma patients are managed in the absence of neurosurgeons. Trauma surgeons should receive basic neurosurgical training that would enable them to support and assist neurosurgeons in caring for these patients.

THURSDAY, NOVEMBER 1

7:45 – 8:00 am

Haptoglobin 2-2 Genotype Predisposes to Chronic Vasospasm after Experimental Subarachnoid Hemorrhage

Rafael J. Tamargo, MD, FACS, Andrew P. Levy, MD, PhD, Kaisorn L. Chaichana, BS, Rachel Miller-Lotan, PhD, and Sophia Shakur, BS

Introduction - Patients who develop severe, symptomatic vasospasm after aneurysmal subarachnoid hemorrhage (SAH) may be genetically predisposed to this complication because of their haptoglobin (Hp) genotype. Inflammation (specifically leukocyte-endothelial cell interactions) appears to be critical in the pathogenesis of vasospasm, and Hp, the serum protein that binds extracorporeal hemoglobin (Hgb) and limits inflammation after a hemorrhagic event, may determine vasospasm. Humans are the only mammals with two alleles of the Hp gene, namely Hp 1 and Hp 2; therefore an individual can be Hp 1-1, Hp 2-1, or Hp 2-2. The Hp 1-1 protein dimer is far superior to the Hp 2-2 cyclical polymer in its immunomodulatory and antioxidant effects.

Methods - 30 wild-type C57Bl/6J Hp 1-1 mice and 30 genetically-modified C57Bl/6J Hp 2-2 mice (Technion, Haifa, Israel) underwent injection of either autologous blood or normal saline solution into the cisterna magna. An additional 30 mice (15 per genotype) served as controls. At 24 hours after SAH, the extent and manifestations of vasospasm were assessed by (1) measuring lumen patency of the basilar artery, (2) quantifying activity levels using a 3-level scale, and (3) counting the number of peri-adventitial macrophages/neutrophils in the subarachnoid space. Statistical comparisons were done using one-way, non-parametric ANOVA (Kruskal-Wallis test and Student-Newman-Keuls multiple comparison test).

Results – Genetically-modified Hp 2-2 mice with SAH had significantly lower basilar artery lumen patencies (mean \pm SEM) (52.9 \pm 1.9% vs. 82.3 \pm 1.3%, $p < 0.01$), reduced activity levels (0.8 \pm 0.3 vs. 2.4 \pm 0.2, $p < 0.01$), and increased macrophage/neutrophil counts in the subarachnoid space (31.2 \pm 6.3 vs. 8.8 \pm 1.7, $p < 0.01$) as compared to wild-type Hp 1-1 mice.

Conclusions - These findings suggest that the Hp 2-2 genotype is critical for the development of severe, symptomatic vasospasm. Vasospasm research should be conducted in Hp 2-2 animals to fully understand the pathobiology of this complication. Aneurysmal SAH patients should be screened for the Hp 2-2 genotype.

THURSAY, NOVEMBER 1

8:00 – 8:15 AM

Academic Neurosurgery in Developing Countries an Achievable Responsibility

Robert Dempsey, MD

Over the past 40 years, the Foundation for International Education in Neurological Surgery (FIENS) has worked to achieve the establishment and maintenance of academic neurosurgical training programs in developing countries. The author recounts a personal 16 year experience of teaching neurosurgery in developing countries as an evolution from naïve volunteerism to focused curricula development under the direction of FIENS. A particular example is the four nation East Africa training program, which is now being launched, where a service population of 300 million had no neurosurgical training programs. The region is now served by a handful of physicians trained in other countries. The steps necessary to develop such a program including international cooperation, local certification, peaceful national security and sufficient funding to retain graduates will be emphasized. The FIENS program participation by academic programs in this country with programs overseas will be addressed through the pairing program of FIENS, pairing U.S. and Canadian programs with those in developing countries. Emphasis will be placed on the central infrastructure needed to establish and maintain academic neurosurgery education. The talk will present our experience including up to 40,000 pounds of donated equipment shipped in one year, 16 sites presently being supported and the need for continued coordination of donations of equipment, time and expertise.

THURSDAY, NOVEMBER 1

8:15 – 8:30 AM

Genetically Engineered T-cells for Treatment of Human Glioblastoma

Bob S. Carter MD, PhD

Introduction: The identification of specific molecular genetic changes in glioma affords the promise of “targeted” molecular and genetic therapies. Our laboratory hypothesized that human T-cells could be genetically to specifically attack the EGFRvIII mutation detected in a substantial proportion of human gliomas, with resultant tumor growth control.

Methods: Human T-cells were nucleofected with a novel chimeric T-cell receptor protein (MRI-CIR) comprised of an extracellular signaling moiety to bind to EGFRvIII, a flexible protein hinge, and the zeta chain of the T-cell receptor. Cells were expanded on feeder layers in IL-2 and anti-CD3 under selection to clinically relevant numbers of T-cell effectors and were used for in vitro and in vivo assays of efficacy.

Results: After nucleofections, cells readily expressed the MRI-CIR by RT-PCR, Western blotting and FACS. Furthermore, the genetically engineered T-cells demonstrated a highly specific and sensitive cytotoxic activity against EGFRvIII expressing glioma cells. Time-lapse video microscopy demonstrated rapid clearing of tumor cells in admixing experiments. Finally, a single intracranial treatment of U87-EGFRvIII expressing tumors doubled the survival time of mice.

Conclusions: Human T-cells, genetically engineered to express a novel chimeric T-cell receptor against EGFRvIII show specific and sensitive activity against human glioma cells expressing this antigen. This study provides a framework for human trials of highly specific anti-glioma immunotherapies using genetic engineering techniques. Our laboratory is currently preparing clinical studies based on the MRI-CIR approach.

THURSDAY, NOVEMBER 1

8:30 – 8:45 AM

MAGE and GAGE are Expressed in Medulloblastoma and Modulate Resistance to Chemotherapy

C. Kasuga, MD Taylor, C. Smith, and
James T. Rutka MD, PhD

Cancer testis antigens (CTAs) are expressed in the testis and many malignant tumors, but not in other normal tissues. As a result, CTAs have the potential to provide specific antitumor immunity to malignant cells without affecting normal cells. MAGE and GAGE are examples of CTAs that have been associated with malignant melanoma. The functions of most MAGE proteins remain unknown, however some studies show correlations between MAGE expression and tumor development, aggressive clinical course, or resistance to chemotherapeutic agents. From a transcriptional profiling study examining 47,000 genes in medulloblastoma cell lines, we identified members of the MAGE and GAGE family as being highly expressed. We examined a series of medulloblastoma tumors by both immunohistochemistry and western blot analysis with antibodies to MAGE-A family, MAGE-A1, and GAGE proteins. Western blot analysis showed expression in 62%, 46%, and 84% of medulloblastoma specimens examined respectively. In addition we observed a correlation between the expression of MAGE and GAGE genes with drug-resistance of medulloblastoma cells to chemotherapeutic agents. We examined the functional significance of this correlation in MAGE knockdown studies and observed increased drug-induced cytotoxicity in UW426 medulloblastoma cells following treatment with chemotherapeutic drugs. Cleaved caspase-3 was found in UW426/MAGE siRNA cells treated with cisplatin, but not in UW426 cells treated with cisplatin alone at the same concentration. Our data show that MAGE and GAGE family members are expressed in medulloblastoma cell lines and specimens and that inhibition of MAGE and GAGE genes by siRNA increases apoptosis of medulloblastoma cells, and sensitizes them to certain chemotherapeutic agents such as cisplatin and etoposide.

THURSDAY, NOVEMBER 1

8:45 – 9:00 AM

Tumor Formation by Malignant Human Glioma Cells Requires Activated Stat3

Michael A. Vogelbaum, MD, Atreyi Dasgupta, Baisakhi Raychaudhuri, Talat Haqqi, Richard Prayson, Erwin G. Van Meir, and S. Jaharul Haque

Previous work published by our group has shown that more than 90% of GBM tumors and all GBM cell lines examined contain the aberrantly activated Stat3 transcription factor. We have also demonstrated that activated Stat3 promotes the growth of GBM cells in vitro by inducing the bcl-2 family of pro-survival genes. To understand the role of activated Stat3 in the growth of GBM in vivo, we have generated U87 clones that express varying levels of a dominant negative mutant (DN)-Stat3 protein in response to tumor hypoxia. We selected several clones that exhibited tight regulation of DN-Stat3 expression under hypoxia in vitro to evaluate their tumorigenic potential in nude mice. We found that palpable tumors were formed within two weeks but further growth of the tumors from the DN-Stat3 clones were halted once they reached ~2mm in thickness, whereas the parental and vector derived tumors maintained a steady and rapid growth rate. This restriction of tumor growth was associated with hypoxia-induced expression of DN-Stat3 which was confirmed by immunohistochemistry. Kaplan-Meier analysis showed the survival time of the mice with orthotopic tumors derived from the DN-Stat3 clones to be significantly longer than the control groups. Ki-67 staining showed limited numbers of proliferative cells in the DN-Stat3 clones compared with control group. These data suggest that activated Stat3 may play an essential role in the tumorigenesis of malignant gliomas. Further efforts are focused on understanding the interactions between Stat3 and other regulatory elements, and on the development of pharmacological inhibitors of Stat3 function.

Supported by NIH R01 grants CA095006 to SJH/MAV and CA87830 and CA86335 to EGVM.

THURSDAY, NOVEMBER 1

9:30 – 9:45 AM

Tentorial Dural Arteriovenous Fistulae: Operative Strategies and Microsurgical Results for Six Types

Michael T. Lawton, MD, Rene O. Sanchez-Mejia, Van V. Halbach

OBJECTIVE: Tentorial dural arteriovenous fistulae (DAVF) are rare, have a high risk of hemorrhage, often cannot be obliterated endovascularly, and frequently require microsurgical interruption of the draining vein. We differentiated these fistulae into 6 types and developed specific operative strategies based on these types.

METHODS: During a 9-year period, 31 patients underwent microsurgical treatment for tentorial fistulae: 7 Galenic DAVFs, 8 straight sinus DAVFs, 3 torcular DAVFs, 3 tentorial sinus DAVFs, 8 superior petrosal sinus DAVFs, and 2 incisural DAVFs.

RESULTS: The posterior interhemispheric approach was used with Galenic DAVFs; the supracerebellar-infratentorial approach was used with straight sinus DAVFs; a torcular craniotomy was used with torcular DAVFs; the supratentorial-infraoccipital approach was used with tentorial sinus DAVFs; the extended retrosigmoid approach was used with superior petrosal sinus DAVFs; and a pterional or subtemporal approach was used with incisural DAVFs. Angiographically, 94% of fistulae were obliterated completely. Four patients had transient neurological morbidity; none had permanent neurological morbidity; and there was no operative mortality (mean follow-up, 4.2 years).

CONCLUSION: Tentorial DAVFs can be differentiated based on fistula location, dural base, associated sinus, and direction of venous drainage. The operative strategy for each type is almost algorithmic, with each type having an optimum surgical approach and an optimum patient position that allows gravity to retract the brain, open subarachnoid planes, and shorten dissection times. No matter the type, the fistula is treated microsurgically by simple interruption of the draining vein.

THURSDAY, NOVEMBER 1

9:45 – 10:00 AM

Physical Exercise and Ischemic Tolerance in Stroke

David F. Jimenez, MD, FACS

Prior studies have indicated that tumor necrosis factor-alpha (TNF-alpha) plays a major roll in ischemia/reperfusion (I/R) injury following ischemic stroke. The study's goals were to further elucidate the relationship between TNF-alpha, heat shock protein-70 (HSP-70) and the extracellular signal-regulated kinases-1 and -2 (ERK1/2) pathways in ischemic/reperfusion injury.

Adult male Sprague Dawley rats were subjected to 30 minutes of exercise on a treadmill each day for 3 weeks. Stroke was induced by 2 hour middle cerebral artery (MCA) occlusion using an intraluminal filament followed by reperfusion. Infarct volume in ischemic injury was determined by Nissl staining in cortex and striatum. Neuronal apoptosis was detected using TUNEL staining. Blood brain barrier (BBB) dysfunction was determined by brain edema and Evans blue extravasation for BBB permeability. Expression of TNF-alpha and HSP-70, as well as MMP-9, and collagen IV, the major protein component in basal lamina of BBB, was determined at transcription and translation levels by real-time PCR and Western blot. Activation of ERK1/2 was determined by examining phosphor-ERK1/2 with Western blot. MMP-9 enzyme activity was determined by Zymography. In order to determine the causative role of pre-ischemic induction of TNF-alpha and HSP-70 in exercise-induced neuroprotection, as well as their signal transduction by ERK1/2, each of the 3 molecules were blocked using neutralizing antibodies for TNF- alpha and HSP-70 or U0126 (phosphorERK1/2 inhibitor). To determine the ERK1/2 mediated MMP-9 pathway in exercise-reduced neuronal damage, we co-administered U0126 and Doxycycline (MMP-9 inhibitor).

Our studies show that physical exercise prior to the ischemic event, down-regulates expression of MMP-9 and collagen IV in association with reduction in brain infarction, apoptosis, and BBB disruption after I/R injury in stroke. Physical exercise up-regulates TNF-alpha and HSP-70. Inhibition of either TNF-alpha or HSP-70 prior to I/R injury blocked exercise-induced neuroprotection and altered ERK1/2 activation. When ERK1/2 was blocked, protein and

activity levels of MMP-9 were increased, in association with increases in infarct volume, apoptosis and BBB permeability. Co-administration of U0126 and Doxycycline suppressed brain injury in association with reduction in MMP-9 gene, protein expression, and enzyme activity. This data elucidates that ERK1/2 signaling function in exercise induces neuronal protection and the ERK-MMP-9 pathway in neuronal damage.

In conclusion, pre-ischemic induction of TNF-alpha and HSP-70 by physical exercise regulates ERK1/2 activation in response to I/R injury, leading to the reduction in brain damage.

THURSDAY, NOVEMBER 1

10:00 – 10:15 AM

Bone-Marrow Derived Hematopoietic Stem Cells Migrate to Sites of Aneurysm Formation by an iNOS-Mediated but not eNOS-Mediated Process in a Novel Murine Saccular Aneurysm Model

Brian L. Hoh, MD, Gregory J. Velat, MD,
Erin N. Wilmer, BA, Edward W. Scott, PhD

Introduction: The pathophysiology of aneurysm formation is not fully-understood. Hematopoietic stem cells (HSCs) are believed to migrate to sites of vascular injury. We developed a novel murine aneurysm model and demonstrate that HSCs participate in aneurysm formation via an inducible nitric oxide synthase (iNOS)-mediated but not an endothelial nitric oxide synthase (eNOS) process.

Methods: Right common carotid artery (RCCA) aneurysms were created in C57BL6 mice by a novel elastase technique. Aneurysms and control left common carotid arteries (LCCA) were harvested at weeks 1,2,3,4 and immunohistochemistry for stem cell markers performed. Bone marrow from DsRed^{+/+} mice, GFP^{+/+},eNOS^{-/-} mice, and GFP^{+/+},iNOS^{-/-} mice were transplanted into irradiated C57BL6 mice, aneurysms created, and migration of DsRed⁺ or GFP⁺ HSCs to aneurysms tracked.

Results: Aneurysms were confirmed by ultrasonography, demonstrated loss of elastin and progressive enlargement over four weeks (P<0.01 vs. LCCA). There was progressive infiltration of CD45⁺,MECA-32⁺,CD31⁺, and F480⁺ HSCs in the aneurysms. Abundant DsRed⁺ CD45⁺,MECA-32⁺ HSCs were tracked to the aneurysms. There was significant reduction in GFP⁺,iNOS⁻ HSCs, but no difference in GFP⁺,eNOS⁻ HSCs, tracked to the aneurysms compared to control animals.

Conclusions: We demonstrate progressive infiltration of bone marrow-derived HSCs with an endothelial progenitor cell profile (CD45⁺,MECA-32⁺) in aneurysms that corresponds with aneurysm enlargement in a novel murine aneurysm model. There was marked reduction in migration of iNOS⁻ HSCs, and no difference in eNOS⁻ HSCs, to aneurysms compared to control animals. This suggests that bone marrow-derived HSCs participate in aneurysm formation via an iNOS-mediated but not an eNOS-mediated process.

THURSDAY, NOVEMBER 1

10:15 - 10:30 AM

Influence of Patient Age and Stenosis Location on Wingspan In-Stent Restenosis (ISR)

A.S. Turk, DO, *Elad I. Levy, MD*,
F.C. Albuquerque, MD, G.L. Pride Jr, MD,
H. Woo, MD, B.G. Welch, MD, D.B. Niemann,
MD, P.D. Purdy, MD, B. Aagaard-Kienitz, MD,
P.A. Rasmussen, MD, L.N. Hopkins, MD,
T.J. Masaryk, MD, C.G. McDougall, MD,
D. Fiorella, MD, PhD

Purpose: To discuss the effect of patient age and lesion location on in-stent restenosis (ISR) rates after percutaneous transluminal angioplasty and stenting (PTAS) with the Wingspan stenting system.

Methods: Clinical and angiographic follow-up results were recorded for all patients from five participating institutions. ISR was defined as >50% stenosis within or immediately adjacent (within 5mm) to the implanted stent(s) AND >20% absolute luminal loss. For the present analysis, patients were stratified into younger (≤ 55 years) and older (>55 years) age groups.

Results: ISR occurred at a rate of 45.2% (14/31) in the younger group and 24.2% (15/62) in the older group (odds ratio 2.6, 1.03-6.5; 95% confidence interval). In the younger group, ISR occurred after treatment of 13/26 (50%) anterior circulation (AC) lesions versus only 1/5 (20%) posterior circulation (PC) lesions. In the older group, ISR occurred in 9/29 (31.0%) AC lesions and 6/33 (18.2%) PC lesions. In young patients, internal carotid artery (ICA) lesions (10/17 treated, 58.8%), especially those involving the supraclinoid segment (SCICA) (8/9; 88.9%), were very prone to ISR. When patients of all ages were considered, SCICA lesions had much higher rates of both ISR (66.6% vs. 24.4%) and symptomatic ISR (40% vs. 3.9%) in comparison to all other locations.

Conclusion: Post-Wingspan ISR is more common in younger patients. This increased risk can be accounted for by a high prevalence of AC lesions in this population, specifically those affecting the SCICA, which are much more prone to ISR and symptomatic ISR than all other lesions.

THURSDAY, NOVEMBER 1

10:30 – 10:45 AM

Secondary Stroke Prevention

Robert F. Spetzler, MD

Objective: Secondary stroke prevention treating vertebral artery origin stenosis is not as well established as internal carotid revascularization at its origin (i.e., carotid endarterectomy). Different therapeutic modalities have been used to treat such conditions and include medical treatment, angioplasty and stenting and surgical revascularization. Recent reports of high restenosis rates after stenting for vertebral artery origin disease revitalized the interest in microsurgical revascularization. The present study was conducted to analyze the results of microsurgical revascularization methods on the treatment of proximal vertebral artery occlusive disease.

Methods: The authors retrospectively reviewed the medical records of twenty-nine patients who underwent microsurgical revascularization for proximal vertebral artery disease between 1986 and 2007. Patient records were analyzed for symptoms at presentation, presence of comorbidities, target lesion characteristics, contralateral vertebral artery findings, procedure-related morbidity, as well as clinical and radiographic outcomes.

Results: Twenty-nine patients (19 male) with age ranging from 35 years to 93 years were operated during the study period. Thirteen, seven and six patients were submitted to vertebral to carotid transposition, endarterectomy or both, respectively. Two patients had subclavian endarterectomy plus vertebral endarterectomy and 1 patient had carotid endarterectomy associated with vertebral transposition. There were no periprocedural strokes or mortality in the present series. Permanent procedure-related neurological complications included 1 case of Horner's Syndrome and 1 case of hoarseness. Two of 27 patients had new neurological symptoms attributable to the vertebral basilar system at 29 months (mean follow up). Although imaging follow up was restricted to 14 of 27 cases, only 1 patient presented early restenosis, most likely related to progression of disease at the site of implantation.

THURSDAY, NOVEMBER 1

10:45 – 11:00 AM

Enhancement of Brain Tumor Immunotherapy by RNAi mediated Down- regulation of Fas-ligand (FasL).

Alessandro Olivi, MD, Timothy Jansen, John
Lattera, MD, PhD

Immunotherapy is emerging as a promising strategy to treat brain tumors. Recently, a number of clinical experimental protocols utilizing vaccines to treat recurrent malignant gliomas have been introduced and enthusiastically embraced by numerous Neurosurgical Centers.

A recognized limitation of this approach has been identified in the active production by the neoplastic cells of immunosuppressive agents that can induce a pronounced apoptosis of the recruited tumor-infiltrating T-cells. Fas-Ligand is a protein over-expressed by malignant brain tumors that indeed appears responsible for T-cell apoptosis.

The hypothesis of this work is that immunotherapy of experimental gliomas can be enhanced by inhibiting FasL expression by glioma cells. We first characterized the expression of FasL in a series of rat glioma cell lines and in human glioma specimens using immunoblot analysis and real-time polymerase chain reaction (real time RT-PCR). Each cell line tested and all human specimens were positive for FasL protein and mRNA expression. Inhibition of FasL expression in 9L, C6, and F98 glioma cell lines was then accomplished in vitro using siRNA. FasL expression inhibition had no effect on tumor growth in T-cell deficient athymic rats and caused a ~50% reduction in subcutaneous and intracranial tumor volumes in immune competent rats indicating that this phenomenon is immune cell dependent. In addition, we have recently been able to generate a FasLsiRNA expressing adenovirus toward developing an effective delivery system for treatment of pre-established wild type gliomas.

These findings suggest that inhibiting FasL in gliomas may be used to enhance the efficacy of already existing immunotherapies such as interleukin delivery and vaccines.

THURSDAY, NOVEMBER 1

11:00 – 11:15 AM

Intracranial Hemorrhage Following Treatment of Unruptured Intracranial Aneurysms

ISUIA Investigators including
D. O. Wiebers, MD, J. C. Torner, MD,
R. D. Brown, Jr., MD, I. Meissner, MD,
and *David G. Piepgras, MD*

Purpose: This study attempts to definite the risk for intracranial (subarachnoid and intracerebral) hemorrhage after treatment of unruptured intracranial aneurysms.

Methods: All patients were participants in the NIH sponsored Study of Unruptured Intracranial Aneurysms involving 61 centers, and were enrolled in the prospective cohort of the study. Mean follow-up was 4 years.

Hemorrhage was determined by clinical, radiographic, and pathologic observations with central adjudication of endpoints. Intracranial hemorrhage occurring within 24 hours of treatment or those clearly associated with the original treatment procedure were excluded from the analysis.

Results: Four hundred fifty one patients underwent endovascular treatment of an unruptured aneurysm. Among these 10 (2.2%) experienced post treatment hemorrhage. Five (1.1%) were from the treated aneurysm, 1 from an untreated aneurysm, 1 from a de novo aneurysm, and 3 of uncertain etiology. Amongst the hemorrhages confirmed as arising from the treated aneurysm, two occurred within 30 days of the procedure and 3 more within the 2nd year post treatment.

Among 1917 patients treated with craniotomy and direct aneurysm repair (clipping in 95%), 11 (0.6%) had post treatment hemorrhage. Seven (0.4%) occurred from the treated aneurysm, 1 from a de novo aneurysm, and 3 of uncertain etiology. Of the hemorrhages arising

from the treated aneurysm, 3 occurred within 30 days of operation and 3 within the first postoperative year.

In all cases with confirmed hemorrhage from the treated aneurysm (both endovascular and clipping) aneurysm size was greater than 15 mm at baseline and in 10 of the 12 cases, giant or near giant in size.

Conclusion: There exists a low but long term risk for intracranial (subarachnoid and intracerebral) hemorrhage that is not eliminated by treatment of unruptured intracranial aneurysms. Nearly half of these hemorrhages occur in the first month post treatment with the treated (index) aneurysm being the most likely source. Large aneurysm size (> 15 mm diameter) is the most significant predictor of this risk.

THURSDAY, NOVEMBER 1

11:15 – 11:30 AM.

Effects of Protease-Activated Receptor-1 on Thrombin-Induced Glioma Cell Proliferation and Vascular Endothelial Growth Factor Secretion and Cell Invasion

Karin M. Muraszko, MD, Ya Hua, MD, Ying Xu, MD, Wenquan Liu, MD, Richard F. Keep, PhD, Guohua Xi, MD

Object: Our previous studies found that thrombin contributes to glioma growth and thrombin inhibition reduces glioma mass and prolongs survival time in rats. The present study was to investigate the role of protease-activated receptor-1 (PAR-1), one of thrombin receptors, in cell proliferation, vascular endothelial growth factor (VEGF) upregulation, and cell migration and invasion induced by thrombin *in vitro*.

Methods: Rat C6 glioma cells were treated with vehicle, thrombin (1 unit), a PAR-1 agonist (TRAP, 50 μ M) or thrombin plus a PAR-1 antagonist (RPPGF, 500 nM). Cell proliferation was measured by a 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-tetrazolium bromide (MTT) assay, VEGF levels were examined using an enzyme-linked immunosorbent assay (ELISA) kit, and cell migration and invasion were determined by a 24-multiwell insert system.

Results: Both thrombin and the PAR-1 agonist induced C6 cell proliferation (% of vehicle: thrombin 1 U, 147 \pm 45%, $p < 0.01$; PAR-1 agonist, 132 \pm 20%, $p < 0.05$) and increased VEGF levels in the culture medium (thrombin 1U, 29 \pm 2 ng/ml; PAR-1 agonist, 20 \pm 2 ng/ml vs 15 \pm 1 ng/ml in the vehicle, $p < 0.05$). Thrombin and PAR-1 agonist also significantly increased cell migration and invasion ($p < 0.05$). The effects of thrombin were blocked by the PAR-1 antagonist.

Conclusion: Thrombin increases glioma cell proliferation, VEGF secretion, the ratio of C6 cell migration and invasion, at least in part, through PAR-1 activation suggesting that PAR-1 may be a new therapeutic target of gliomas.

FRIDAY PROGRAM

FRIDAY, NOVEMBER 2

7:00 – 7:30 AM

GUEST SPEAKER

DBS to Treat Depression: Redefining Psychosurgery

Helen S. Mayberg, MD

Emory University School of Medicine

Critical to development of deep brain stimulation (DBS) as a novel therapy for treatment resistant depression (TRD), has been the evolving understanding of brain systems mediating normal and abnormal mood states and the ongoing, systematic characterization of neural substrates mediating successful and unsuccessful response to other antidepressant interventions. Based on previous work implicating the subgenual cingulate (sACC) and its functional connections to specific paralimbic, cortical and subcortical regions in the pathophysiology of depression and antidepressant response mechanisms, we tested the use of bilateral, chronic high frequency DBS to modulate sACC connectivity in patients with TRD. Initial open studies of Cg25WM DBS for TRD suggest the procedure is safe and effective with sustained remission in initially responsive patients now exceeding 1 year. Replication of the initial findings using a placebo controlled design is ongoing. The theoretical and data-driven foundation for developing this new procedure as well as clinical, neuropsychological and imaging findings from these first experimental patient cohorts will be presented.

FRIDAY, NOVEMBER 2

7:30 – 8:00 AM

GUEST SPEAKER

The Glioblastoma Genome: Revealing Secrets for Improved Therapy

Gregory J. Riggins, MD, PhD,
Johns Hopkins University, Department of
Neurosurgery

Oncogenomics technology has rapidly advanced, and it is now possible to comprehensively analyze cancer genomes. Genome projects are completed or underway to gain a nearly complete understanding of the genomic DNA changes in glioblastoma. What has been learned so far about these molecular changes that lead to brain cancer? In our work, we have analyzed glioblastoma genomes in high resolution using techniques such as Digital Karyotyping and Illumina 550K SNP bead arrays. Although the frequency of amplified oncogenes or deleted tumor suppressor genes is often surprisingly different than thought, these genomic alterations cluster into well studied pathways, such as those that overcome checkpoint control, and receptor tyrosine kinase activation to suppress apoptosis and promote invasion. Our large-scale mutation screens show similar results with new mutations being found in certain functionally (and therapeutically) important receptor tyrosine kinases genes.

Better therapy has been the promise of genome projects since the planning of the human genome project- can this knowledge be translated into therapeutic advances? Although it is certainly an ambitious undertaking, targeted therapy of brain tumors can be achieved. Using targeted small molecule screens; we have identified new kinase inhibitors for glioblastoma that extends survival as a single agent in animal trials. These screens are a possible example of how to translate genomic information into therapy. Success in this area from investigators targeting genomic changes in cancer will likely transform the practice of neurosurgical and medical treatment of brain cancer.

FRIDAY, NOVEMBER 2

8:00 – 8:15 AM

Deep Brain Stimulation Increases Adult Hippocampal Neurogenesis

Andres M. Lozano MD, PhD, H. Toda,
C. Hamani, A.P. Fawcett, W.D. Hutchison

Deep brain stimulation (DBS) is actively used to treat Parkinson's disease, epilepsy and psychiatric disorders, but the cellular and molecular consequences of this therapy are largely unknown. Here we show in adult rodents that, in addition to modulating the activity of brain circuits, DBS also strikingly increases the production of neurons in the hippocampus and reverses the suppression of neurogenesis with steroids. The coupling between stimulation and neurogenesis occurs only with high frequency stimulation. Since increasing hippocampal neurogenesis may lead to functional improvement, it may be possible to use electrical stimulation to treat conditions associated with impairment of hippocampal function.

Key Words: deep brain stimulation, adult neurogenesis, hippocampus, anterior thalamic nuclei

FRIDAY, NOVEMBER 2

8:15 – 8:30 AM

Vaccination with Tumor Lysate-Pulsed Dendritic Cells Elicits Correlated Immune and Survival Response in Patients with Glioblastoma Multiforme (GBM)

Keith L. Black, MD, Mia Wagenberg, Xiaoxue Zhang, Gentao Liu, Elina Mindlin, Laura Blasczewicz, Chiayi Chen, Samuel Pepkowitz, Dennis Goldfinger, Hiushan Ng, Dwain Irvin, John S. Yu, Christopher J. Wheeler

Glioblastoma has a dismal prognosis, and survival is at best improved by a few months with existing therapies. Immune-correlated clinical improvements observed in animal models have not previously been reported in vaccinated glioma or other cancer patients. This has hampered insight into how therapeutic vaccines afford clinical benefits to patients, limited rational approaches to the improvement of clinical vaccines, and fueled controversy over the utility of both animal models and human cancer vaccines. Type I cytokine responsiveness and post-treatment survival was analyzed in 33 patients with grade IV glioma (glioblastoma multiforme; GBM). 55% of GBM patients exhibited a 1.5 fold or higher vaccine-enhanced cytokine response (vaccine responders). Similar endogenous anti-tumor responses were evident in 28% of GBM patients prior to vaccination, and these endogenous responses were stronger in younger patients. Endogenous responsiveness was inversely correlated with post-vaccine anti-tumor responsiveness. Vaccine responders exhibited significantly longer survival and post-vaccine time to progression (TTP) relative to post-vaccine TTP of non-responders. Survival was increased in vaccinated GBM patients relative to comparable patients receiving non vaccine standard or experimental treatments, with vaccine responders contributing exclusively to this increase. Analysis of tumor TTP suggested that vaccination incurred a substantial benefit by sensitizing tumors to post-vaccine chemotherapy. This is the first report demonstrating that therapeutic vaccination enhances survival in proportion to T cell responsiveness.

FRIDAY, NOVEMBER 2

8:30 – 8:45 AM

Temozolomide (TMZ) can be Safely Administered in the Immediate Postoperative Period After Tumor Resection and Gliadel Wafer Placement in Patients with Newly Diagnosed Gliomas: Results of a Prospective, Multi-institutional, Phase I/II Trial

Anthony L. Asher, MD, FACS, Stuart Burri, MD, Wendy Brick, MD, Lynn Ashby, MD, Kevin Judy, MD

Introduction: TMZ and BCNU have demonstrated anti-tumor synergism in pre-clinical models. Kinetics of BCNU dispersion in brain after Gliadel wafer placement mandates that potentially synergistic systemic agents be administered in the early post-operative period. We report interim data from a prospective, multi-institutional, study of Gliadel wafers and TMZ in patients with newly diagnosed malignant gliomas.

Methods: After resection and Gliadel wafer placement, first cycle TMZ began on day four postoperatively. Next, involved-field radiation to 59.4 Gy with concomitant TMZ (75 mg/m²/d) was administered. Two weeks after RT, monthly TMZ was initiated. TMZ cycle doses for the first and second cohorts were 200 mg/m²/d x 5 days and 150 mg/m²/d x 5 days, respectively.

Results: 40 (38 GBM, 2 AA) patients enrolled. In 26 patients with >12 month follow up (to date) median and twelve month survivals were 533 days and 85%, respectively. Non-hematologic toxicities were minimal. In cohort 1 (median TMZ cycles and days follow-up 5 and 220 respectively) 9/29 patients experienced one or more episodes of grade 3/4 thrombocytopenias (6/29 patients after initial cycle TMZ). Only one patient removed from study secondary to HT. In a second cohort of 11 patients receiving TMZ cycles 150 mg/m²/d x 5 days (median TMZ cycles and days follow-up, 6 and 165, respectively) no hematologic toxicities have yet been observed.

Conclusions: We conclude that peri-operative TMZ can be safely administered as an adjunct to Gliadel wafers in the early postoperative period after glioma resection and that further studies are warranted to examine the efficacy of this combination.

FRIDAY, NOVEMBER 2

8:45 – 9:00 AM

Lateral Hypothalamic Radiosurgery for Obesity

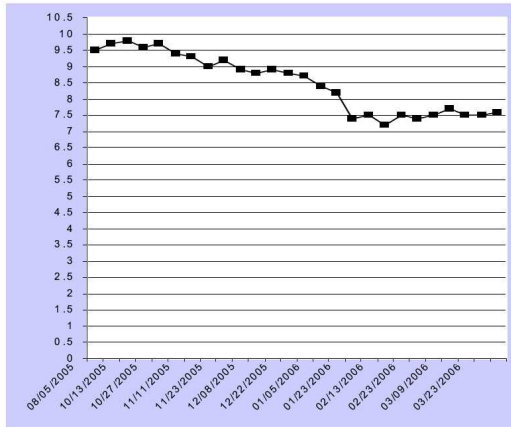
L. Dade Lunsford, MD, FACS and
Ajay Niranjana, MCh

Background:

Obesity is a growing and major U.S. health problem associated with morbidities leading to early death. Currently, gastric bypass is the most effective way to induce long lasting weight loss, but has significant potential side effects. We assessed the feasibility of lateral hypothalamic radiosurgery in a primate model prior to introduction of a Phase I clinical trial in appropriately selected patients.

Methods and Results:

The initial pilot project developed a protocol for identification of the monkey lateral hypothalamic target using high resolution MRI fused to a standard stereotactic MRI. In 4 obese macaca mulata monkeys we created a lateral hypothalamic lesion using the 4 mm Leksell Gamma Knife® collimator. In 2 monkeys, we were able to demonstrate a gradual loss of total body weight and to observe changes in feeding behavior.



Gradual weight reduction leading to 21% weight loss within 6 months of radiosurgery was noted in primate treated with 100Gy bilateral hypothalamic radiosurgery

Discussion:

We confirmed that lateral hypothalamotomy can result in appetite suppression and weight loss in morbidly obese animals. Radiosurgery has been used effectively in the management of pain, movement disorders, and epilepsy in humans. High resolution MR imaging is an effective way to delineate the lateral hypothalamus and to avoid injury to the optic apparatus. In the future, radiosurgical hypothalamotomy may improve weight management without the long-term risks of more invasive treatment strategies such as gastric bypass.

FRIDAY, NOVEMBER 2

9:30 – 9:45 AM

Facial Nerve Outcomes in Vestibular Schwannoma Surgery Following Failed Radiosurgery or Failed Microsurgery

Michael J. Link, MD

Introduction: Microsurgery (MS) to remove vestibular schwannomas (VS) and stereotactic radiosurgery (SRS) to prevent future growth can rarely result in tumor recurrence or progressive tumor enlargement, respectively, necessitating further intervention.

Material and Methods: Patients with VS treated with SRS receiving subsequent salvage MS were compared with a similar cohort of patients that developed tumor recurrence after prior surgery requiring additional MS. No patient had NF2. There were 8 patients in each group.

Results: The mean age of the patients in the failed SRS group was 62.6 years. Mean tumor diameter at subsequent MS was 2.1 cm. The mean tumor growth following failed SRS was 10.5 mm. 7/8 patients had normal (HB gr 1) VII nerve function preoperatively. At mean f/u of 13 months after surgery only 3/7 patients kept HB gr 1 – 2 VII n function. In 5/8 patients, a gross total resection (GTR) could be performed.

The mean age of the patients in the failed MS group was 49.7 years. Mean tumor size was 2.6 cm. Only 5/8 patients had HB gr 1 – 2 VII n function preoperatively. At f/u 6/8 patients had HB gr 1 – 2 VII n function with one patient improving from grade 5 preoperatively to a grade 2. A GTR could be performed in 7/8 patients.

Conclusions: MS and SRS both can be associated with treatment failures. Subsequent salvage MS after failed SRS is associated with a higher likelihood of poor VII n outcome and less than GTR, compared to patients requiring repeat MS.

FRIDAY, NOVEMBER 2

9:45 – 10:00 AM

The Development of Oncolytic HSV-1 as a Potential Therapy for Malignant Glioma

James M. Markert, MD, FACS

Oncolytic Herpes Simplex Virus type 1 (HSV) vectors have undergone significant development as potential therapeutics for malignant glioma since their initial description for such in 1991. Two North American clinical trials using an engineered oncolytic HSV, G207 have been completed without dose limiting toxicity. Positive responses to treatment have been demonstrated in some patients, with some long-term survivors. Biologic data generated from the second study has increased our understanding of how oncolysis proceeds in human tumors, and the potential relationship of the immune response to therapeutic response. A third study, combining G207 with an immediate fraction of post-inoculation radiation, is underway. The rationale for the current study is the synergistic antiglioma effect of oncolytic HSV-1 that occurs in conjunction with ionizing radiation, particularly when administered within 24 hours of treatment. A low dose radiation cohort is nearing the completion of enrollment. Results from these initial studies have led to the development of second and third generation viruses in the laboratory, as well as novel approaches for adjunctive treatment with oncolytic HSV-1. Future trials are planned that will build on the findings from these initial clinical and laboratory studies, utilizing viruses that express foreign genes and thus function as gene therapy vectors as well as oncolytic viruses.

FRIDAY, NOVEMBER 2

10:00 – 10:15 AM

ACADEMY AWARD RUNNER UP

An SiRNA Screen Revealed that PARP1 and CDK7 Inhibition Selectively Radiosensitized Glioma Cells Over-Expressing EGFRvIII

Clark C. Chen, MD, PhD, Richard Kennedy, MD, PhD, Alan D'Andrea, MD

Radiotherapy is a critical component of malignant glioma treatment, however, its efficacy is limited by tumor radioresistance. This resistance is, in large part, due to cellular changes related to oncogene over-expression. We carried out a SiRNA screen to identify genes involved in this process. In the initial screen, a glioma cell line over-expressing the EGFRvIII oncogene (U87+EGFRvIII) and another line harboring an oncogenic version of its downstream effector, H-Ras (THV+ H-Ras^{V12G}) were used. Distinct cell lines and oncogenes were used to minimize experimental artifacts. We identified SiRNAs that radiosensitized both the U87+EGFRvIII and the THV+H-Ras^{V12G} cell line. Five top scoring SiRNAs (directed against PARP1, CDK7, RPA3, FANCF, and HDAC1) were then tested to determine whether they radiosensitized the oncogene over-expressing cell lines (U87+EGFRvIII and THV+H-Ras^{V12G}) more than isogenic control cell lines without such over-expression (U87 and THV, respectively). These experiments revealed that silencing of PARP1 and CDK7, but not of RPA3, FANCF, or HDAC1, preferentially radiosensitized gliomas over-expressing EGFRvIII or H-Ras^{V12G} relative to their isogenic control counterparts. To exclude “off-target effects”, we showed that treatment with a specific PARP1 inhibitor, recapitulated the selective radiosensitizing effects of PARP1 silencing. These results demonstrate the utility of SiRNA/isogenic cell line use in drug discovery and suggest EGFRvIII overexpression and H-ras activity as potential biomarkers for PARP1 inhibitor sensitivity.

FRIDAY, NOVEMBER 2

10:15 – 10:30 AM

ACADEMY AWARD PAPER

A Combined Epigenetic and Genetic Genome-wide Screen Identifies *SPINT2* as a Novel Tumour Suppressor Gene in Medulloblastoma

Paul N. Kongkham, MD

Michael D. Taylor, MD, PhD,

James T. Rutka MD, PhD

Medulloblastomas (MB) are the most common pediatric nervous system malignancy. Despite treatment, mortality rates remain 30%. Known mutations account for only a small subset of MB cases. We hypothesize that promoter CpG island methylation-mediated tumour suppressor gene (TSG) silencing contributes to MB pathogenesis – either alone, or in combination with genetic events such as loss of heterozygosity (LOH). Screening for genes silenced by methylation, and regions where methylation converges with LOH, may pinpoint novel TSGs previously not described in MB. To this end, we performed a genome-wide screen of MB cell lines treated with 5-aza 2’ deoxycytidine using Affymetrix HG U133 plus 2.0 cDNA expression arrays, finding genes with increased expression following treatment with this methylation inhibitor. We identified regions where methylation and LOH converge by comparing expression array data with LOH data generated on the Affymetrix 100K single nucleotide polymorphism mapping array set. This strategy identified *SPINT2* – an inhibitor of the HGF/cMET signaling pathway. We confirmed methylation of *SPINT2* using bisulfite sequencing, and found aberrant methylation in 17/51 primary tumours by methylation-specific PCR. TSG function was assessed through re-expression studies in vitro, demonstrating a reduction in cell proliferation rate by MTS assay following *SPINT2* re-expression. In addition, re-expression of *SPINT2* lead to a reduction in cell migratory ability, as evidenced by impaired healing of an artificial wound using a scratch assay. This study is the first to identify *SPINT2* as a TSG in human MB, and further implicates aberrant HGF-cMET oncogenic signaling in the pathogenesis of this disease.

SATURDAY PROGRAM

SATURDAY, NOVEMBER 3

7:00 – 7:15 AM

Neurosurgery and Pay for Performance

Robert Harbaugh, MD

In an effort to control the costs and, ostensibly, improve the quality of medical care, the Center for Medicare and Medicaid Services (CMS) has initiated a “pay for performance” program. The background information regarding this evolving process for physician reimbursement and the numerous government and private organizations involved in its development will be reviewed. The response of organized neurosurgery, via the Washington Committee’s Quality Improvement Workgroup (QIW) and the American Board of Neurological Surgery (ABNS) will be delineated.

To improve the quality of neurosurgical care, it will be necessary to prospectively collect process and outcomes indicators of quality, analyze this data and feed it back to individual surgeons in a secure, non-punitive environment. To accomplish this, the QIW and ABNS have developed procedure specific, secure, on-line outcomes modules that will allow neurosurgeons to collect baseline clinical, demographic and functional health status data, track process measures, surgical procedures, adverse events and patient outcomes. The system allows individual surgeons to compare their risk-adjusted data to benchmarks developed from the universal dataset. Data analysis will allow determination of best practices and dissemination of this information to all neurosurgeons. Such a system could be used as a tool for resident training in outcomes assessment, data collection for board eligible neurosurgeons prior to oral board examination, part of the ABNS maintenance of certification program, state and hospital mandated quality improvement efforts and pay for performance.

SATURDAY, NOVEMBER 3

7:15 – 7:30 AM

Community Equipoise and Individual Surgeon Equipoise in Designing a Feasible Randomized Clinical Trial

Fred G. Barker MD, Jean-Valery Coumans MD, Edward C. Benzel MD, Lauren Stabile BS, Zoher Ghogawala, MD

Introduction: Randomized clinical trials (RCTs) can fail because of poor enrollment, or if completed, can fail to convince clinical practitioners. Both problems can reflect a lack of equipoise, or clinical uncertainty, for the trial’s clinical question. When surgeons lack equipoise between the two competing treatments, trial enrollment suffers; when enrollment includes patients unsuitable for one treatment, trial results may be unconvincing. We used spatial voting theory to study equipoise quantitatively in the design of a neurosurgical RCT.

Methods: A proposed RCT of anterior vs. posterior decompression for multilevel cervical spondylotic myelopathy (CSM) was modeled using a survey. 91 spine surgeons each assessed 10 CSM cases randomly drawn from a panel of 20 actual cases (804 individual case/surgeon decisions analyzed). For each case, surgeons stated preferred surgical approach (anterior/posterior) and willingness to randomize the decision. Surgeon demographics and interest in RCT participation were captured.

Results: Using item-response theory, both surgeons and cases could be “scored” on a single quantitative scale that predicted anterior/posterior choice for individual decisions. For some extremely-“anterior” or “posterior” cases, all surveyed surgeons chose the same approach; for others, many surgeons were predicted to have difficulty deciding. Cases approximating a 50/50 split between approaches were most likely to be randomized ($P=0.04$). Cases close to each surgeon’s “uncertainty point” were most likely to be randomized by that surgeon. Surgeons soonest after training were significantly more likely to randomize ($P=0.04$).

Conclusions: This novel method adapted from theoretical political science allowed identification of case eligibility criteria and surgeon-investigator characteristics likely to maximize RCT enrollment.

SATURDAY, NOVEMBER 3

7:30 – 7:45 AM

Aggressive Correction of Cervical Kyphotic Deformity Results in Improved Neurological Outcomes

Praveen V. Mummaneni, MD, Sanjay Dhall, MD, Regis W. Haid, MD, Gerald E. Rodts, MD

Introduction: Few prior reports have examined combined anterior/posterior correction of cervical kyphotic deformity. We present our long-term results with cervical kyphosis correction.

Methods: 25 patients underwent surgery at one institution from 2002 to 2006 for cervical kyphotic deformity. Anterior procedures included discectomies and corpectomies on one or more levels. Posterior operations included fixation and arthrodesis with decompression and/or osteotomies. Preoperative and postoperative Ishihara kyphosis indices, modified JOA scores and Nurick grades were assigned as well as Odom's outcome measures. Arthrodesis was assessed via dynamic radiographs. CT scans were used to assess fusion in questionable cases.

Results: Two patients were lost to follow-up. Of the 23 remaining, 3 died postoperatively. 20 patients were available for follow-up with a range from 3-44 months (Mean 15 months). Ishihara indices improved from a preoperative mean of -14 to a postoperative mean of +10. The mean improvement in Nurick score after surgery was 1.9 grades. The mean improvement in modified JOA scores after surgery was 3.8. Odom's scores were: Excellent 30%, Good 35%, Fair 35%. All patients with follow-up fused postoperatively except one with renal failure (95% fusion rate).

Conclusions: In cervical kyphosis cases, aggressive management with decompression, osteotomies, and stabilization from both anterior and posterior approaches can restore cervical lordosis. Such aggressive management techniques can provide measurable improvements in neurological function (Nurick grades and modified JOA scores) and achieve high fusion rates. Our experience is the largest in the literature examining combined anterior and posterior correction of cervical kyphotic deformity using standardized outcomes.

SATURDAY, NOVEMBER 3

7:45 – 8:00 AM

High-Resolution MRI Virtual Endoscopy for Preoperative Visualization of Neurovascular Anatomy in Trigeminal Neuralgia

Kim Burchiel, MD, Jonathan Miller, MD

We report a novel technique to identify neurovascular compression in trigeminal neuralgia. Using three-dimensional reconstructed high-resolution balanced-fast-field-echo (BFFE) images fused with 3D-TOF MR-angiography and gadolinium-enhanced MR images, it is possible to objectively visualize the trigeminal nerve and nearby arterial and venous structures at an unprecedented level of anatomic detail. Forty consecutive patients with unilateral trigeminal neuralgia underwent three prospective 3-Tesla MRI examinations: high-resolution BFFE, 3D-TOF MRA, and post-contrast 3D-spoiled-gradient-recalled sequence. Images were imported to a Macintosh laptop computer running the open-source OsiriX imaging software. After fusion, a three-dimensional false-color reconstruction was produced using surface rendering. The reconstructed image objectively differentiates nerves and vessels and can be viewed from any angle, including the anticipated surgical approach. Patients then underwent microvascular decompression. Twenty-two patients were predicted to have neurovascular compression on the symptomatic side (14 arterial and 8 venous). Among patients taken to surgery, fifteen nine patients with predicted to have compression (9 arterial and 5 venous), and all of these were found to have a vascular structure identical in location and configuration to that predicted by preoperative analysis. Three patients without predicted compression nevertheless underwent exploration because of classic symptoms. Surgical exploration in two of these revealed no offending vessel, as was predicted. The other patient was found to have a small vein embedded into the nerve that was below the resolution of the 3D-Gad study. By combining BFFE with MRA and gadolinium-enhanced MRI, we have been able to capitalize on the advantages of both techniques, allowing for MRA and post-contrast MRI discrimination of vascular structures at BFFE resolution. This results in an unambiguous three-dimensional image that can be used for diagnosis and surgical planning.

SATURDAY, NOVEMBER 3

8:00 – 8:15 AM

A Progressive Increase In Brain Microhemorrhages Correlates With Sporadic Late-Onset Dementia Development

Wolff M. Kirsch, MD, W. Baqai, Mc Auley, J.P. Larsen, S. Bhaskerrao, W. Britt, III, F. Petersen, C. Dickson, D. Kido, M. Ayaz, E.M. Haacke, L. Liotta, H. Vinters

Our goal is to define the pathogenesis of sporadic late-onset dementias studying brain microvasculature with new minimally invasive technologies. For the past 4.0 years we have monitored the cognitive course of 76 mildly cognitively impaired (MCI) and 28 cognitively normal elderly individuals applying new MR contrast (Susceptibility Weighted Imaging, SWI) and proteomic tools (“carrier protein stripping”). Our aim was to determine regional brain iron accumulation in the course of cognitive loss by SWI. SWI at 1.5T revealed an unanticipated association of cognitive loss with increasing cerebral microhemorrhages (MH) typical for “cerebral amyloid angiopathy (CAA).” SWI is superior to conventional gradient echo T2* (GE-T2*) for MH detection. Six of 15 MCI participants followed to dementia have significant MH and unique serum proteins. Our human experiment is the first prospective evidence for CAA microvasculopathy in the pathogenesis of late onset dementia.

As a result of the association between cerebral MH and progression to dementia, 3 key endpoints have been added to our study; (a) quantifying the location of MH by 3T SWI during the course of dementia, (b) proteomic studies of peripheral blood to complement SWI MH detection to develop a clinical test for CAA and (c) determine the role of CAA hypoxic and apoptotic mechanisms responsible for neuronal and cognitive loss in an appropriate transgenic mouse model.

Defining the role of microvasculopathies in the pathogenesis of sporadic late-onset dementia may result in new therapeutic strategies.

This research was funded by NIH grant AG20948.

SATURDAY, NOVEMBER 3

8:15 – 8:30 AM

Modulation of Tumor Microenvironment to Enhance Oncolytic Virus Therapy

E. Antonio Chiocca, MD

Background: Tumor microenvironment is increasingly recognized as an important determinant for tumor progression and also its response to therapeutics. We investigated changes induced in tumor blood vessels after oncolytic-viral (OV) therapy, and the impact of modulating tumor vasculature to enhance OV therapy. *Methods:* Glioma cells U87 (human) or D74/HveC (rat) implanted intracranially in nude mice or immune-competent rats were treated with oncolytic viruses (rHSVQ or hrR3). Changes in tumor circulation were evaluated by MRI imaging of i.v injected USPIO particles, and by leakage of injected fluorescent-dye, imaged by confocal fluorescence microscopy. Changes in inflammatory cell infiltrate, vasculature, and presence of virus in tumor was evaluated by immunohistochemistry staining. Changes in gene expression after oncolysis were measured by real time QPCR. Therapeutic efficacy was determined by comparing survival of tumor bearing animals using Kaplan-Meier analysis. *Results:* OV treatment of experimental gliomas resulted in a significant increase in vascular permeability. This vascular hyperpermeability correlated with increased iNOS and infiltration of host leucocyte cells in tumors. Vascular hyperpermeability was suppressed in immune suppressed animals. Normalization of tumor blood-vessels significantly reduced OV-induced vascular permeability and inflammation. Reduced inflammation limited viral clearance by host immune response, thereby increasing therapeutic efficacy. *Conclusions:* This article is the first study describing changes in host vasculature upon OV treatment of tumors, and how these can be manipulated to limit host inflammatory responses, to enhance oncolysis. This study can lead to the development of treatment strategies combining drugs that are already in clinical trials allowing for their rapid translation.

SATURDAY, NOVEMBER 3

8:30 – 8:45 AM

Integrated Medical Learning: A New Concept for Neurosurgical Education

Douglas Kondziolka MD and Anthony Asher MD

Multiple forces are redefining surgical education as a life-long process designed to meet individual needs by utilizing on-going self-assessment. Novel programs emphasize learner-driven and learner-centered activities that are comprehensive in scope, and utilize a variety of learning formats and venues. Recent data suggests that learner-centered programs, which encourage interaction between students and teachers, are superior in effecting transfer of knowledge. We created a new educational concept called Integrated Medical Learning (IML). IML transforms previously isolated, short-term educational experiences (such as medical meetings, lectures, publications, or teaching modules), into a unified series of dynamic interactions between teacher and student. IML based-education integrates the knowledge and practice patterns of both teacher and student. IML focuses not only on the learner but also on continual interactive feedback that improves the learning system over time.

A Scientific Committee (SC) comprised of senior medical educators coordinates the IML process. The process begins with a preliminary identification of essential topics and faculty members along with a selection of scientific *questions* that are valid, current, and important to the learning experience. These questions are submitted to the student body (e.g., CNS membership) in the form of a web-based questionnaire, together with educational content (relevant material). Over a 1-2 month period, learners answer questions relevant to the essential topics covering a variety of areas including individual practice patterns, treatment biases, baseline knowledge, socioeconomic factors, sample case management etc. Responses are then sent to the lecturers so that the data can be incorporated into their presentations. Lecturers are requested to specifically address these core issues and to respond to learner comments and answers. Learners then participate in an interactive session at a meeting, facilitated through a web-based format using a variety of digital devices. After real-time review of audience feedback by a subset of the SC, the moderator then can pose novel or

frequently asked questions or comments to the speakers. Data regarding practice patterns, treatment biases, impact of the sessions on opinions regarding patient care, and other factors is collected during the interactive sessions and from the initial survey. The data is screened for information that could help clinician/educators formulate ideas for new clinical protocols, identify deficient areas in a specialty's collective knowledge base, and create new educational programs for the society's members. Following the meeting, the collected data is then analyzed, written in manuscript form and submitted to peer-reviewed journals or organization publications as appropriate. The educational experience is then evaluated and refined as needed. IML was first used during the 2007 Congress of Neurological Surgeons meeting, focusing on topics related to brain metastases, lumbar spondylolisthesis, and cerebral aneurysm. The data obtained from this process will first be presented at this Academy meeting.

IML will allow us to measure the effects of educational techniques on the knowledge and behavior of participating neurosurgeons (both learners and teachers). In this manner, we transform the scientific program into an investigative enterprise, in which learners collectively participate in advancing the science of both patient care and surgical education. IML creates synergistic relationships between a number of novel learner-centered educational approaches in order to provide for a coherent, continuous, overall approach to medical learning.

SATURDAY, NOVEMBER 3

8:45 – 9:00 AM

Automated, Compliant, High-Flow Common Carotid to Middle Cerebral Artery Bypass

Ralph G. Dacey, Jr., MD, Greg Zipfel, MD,
William Ashley, MD, PhD, Michael
Chicoine, MD, and Michael Reinert, MD

High flow extracranial – intracranial (EC-IC) arterial bypass is often required in the surgical or combined surgical – endovascular management of complex cerebral aneurysms and skull base tumors. Here we report for the first time the use of the Cardica C-Port device for automated cerebral EC-IC anastomosis.

METHODS:

The Cardica C-Port Anastomotic System has been developed and tested in coronary artery bypass surgery to perform rapid, compliant anastomosis. The device is hand held and powered by a CO₂ cartridge in the handle. The anvil contains a microknife, which simultaneously makes an arteriotomy in the recipient vessel, and inserts 12 microclips through the adventitia of the saphenous vein conduit into the recipient middle cerebral artery vessel. The anastomosis is created by depressing the trigger on the handle of the device, which makes the arteriotomy and deploys the cartridge assembly within seconds. Minimal temporary occlusion is required for the procedure.

RESULTS:

The use of the device was simulated in cadavers prepared for microdissection. M2 branches of the middle cerebral artery with diameters between 1.75 and 2.5 mm were noted to be excellent recipient vessels. The anvil of the device has a length of 9mm and the overall cartridge anvil assembly requires a space of about 14mm for insertion.

The device was used to create an EC-IC bypass in a patient with a traumatic fusiform aneurysm with a proximal internal carotid artery. The bypass extended from the common carotid artery to the inferior division of the middle cerebral artery on the left. The patient made a

good recovery after some cerebral swelling related to a hyper perfusion syndrome. Follow-up angiography revealed excellent patency of the bypass.

DISCUSSION:

The use of this anastomosis device is a new development, which will facilitate the technically predictable creation of compliant (non-running suture) cerebral anastomosis. The device facilitates the extremely rapid completion of the bypass and obviates long periods of temporary occlusion of the recipient vessels. A detailed description of the design of the device and a video presentation of the surgical procedure will be presented. The experience with the device in the cardiac surgery literature will be reviewed.

SATURDAY, NOVEMBER 3

9:30 – 9:45 AM

Neural Stem Cell Transplantation for Stroke

Gary K. Steinberg, MD, PhD, Tonya M. Bliss, PhD, Raphael Guzman, MD, Marcel Daadi, PhD, Nobuko Uchida, PhD, Irving L. Weissman, MD, Ben Barres, MD, PhD, Michael Moseley, PhD, Theo Palmer, PhD

Currently there is no treatment for restoring neurological function after stroke. Neural stem cell transplantation offers a potential new therapy for stroke patients. We studied the use of human fetal neural stem cells (hCNS-SC), human embryonic derived neural stem cells (hES), human post-mitotic neuronal progenitor cells (hNT) and rodent neural stem cells in experimental ischemic rodent models. hCNS-SCs transplanted to ischemic rat cortex survive robustly, migrate in a targeted fashion to the stroke and differentiate primarily into immature neurons (49% b-tubulin+). The dependence of targeted migration on the chemokine receptor CXCR4 was evaluated using neural progenitors from CXCR4 KO mice. Transplanted hCNS-SCs also induce neovascularization following stroke. hCNS-SCs improve neurologic function when transplanted into the ischemic NOD-*scid* mouse striatum. hES cells survive, migrate, differentiate (61% immature neurons) and improve motor deficits 2 months after stroke in immunosuppressed rats, without tumor formation. hNT neurons transplanted into the ischemic rat cortex survive, extend processes and differentiate into neurons, but do not improve behavior. Grafts of rat medial ganglionic eminence (MGE) neural precursors into ischemic rat striatum differentiated into neurons, express synaptic markers and improve motor behavioral deficits. Mouse neuronal progenitor cells enhance synapse formation when co-cultured with retinal ganglion cells, due to thrombospondin expression. Enrichment of mouse neural stem cells by FACS for the surface integrin CD49d and intracarotid delivery promotes transendothelial migration, cell homing to the stroke and improves behavioral recovery. Superparamagnetic iron oxide nanoparticle labeling allowed in vivo tracking of the hCNS-SCs in stroke models, without altering their biology.

Cell transplantation therapy for stroke holds great promise. Investigation of transplantation for stroke is still in the early stages. Many fundamental issues related to optimizing therapy need to be resolved in pre-clinical studies.

SATURDAY, NOVEMBER 3

9:45 – 10:00 AM

Bone Marrow-Derived Mesenchymal Stem Cells are Recruited to and Alter the Growth of Human Gliomas

Frederick F. Lang, MD, Kenneth Aldape, Raymond Sawaya, Howard Colman

Bone marrow-derived human mesenchymal stem cells (BM-hMSCs) are stem/progenitor cells that differentiate into stromal elements and are important for marrow homeostasis. We have shown that BM-hMSCs localize to gliomas after intravascular delivery (*Nakamizo et al. Cancer Res 65:3307-3318, 2005*), suggesting that circulating BM-hMSCs may be recruited into brain tumors. Thus, we tested whether BM-hMSCs can be isolated from gliomas and whether they alter tumor growth.

Surgical specimens from 41 glioma patients were cultured in “BM-hMSC media”. In 29 cases (71%) we isolated cells identical to BM-hMSCs (i.e. CD34-, CD38-, CD45-, CD133-, CD105+, CD44+, CD166+). Cultures contained 1.1% - 89.7% BM-hMSC-like cells. Single clones could be differentiated into osteocytes, chondrocytes, and adipocytes, identical to BM-hMSCs. Additionally, the median percentage of circulating BM-hMSCs (CD105+ cells) in blood samples from 19 patients was 34.9% (range 17.2%-69.8%), whereas in normal controls (N=5) it was 19.6% (range 11.1-23.0, $P < 0.05$). When tumor-derived BM-hMSC-like cells (10^6) were implanted in mice brains, 28/29 samples were not tumorigenic. However, when mixtures (5:1 ratio) of U87 glioma cells and BM-hMSCs, fibroblasts, or normal astrocytes (NHA) were implanted, the median survival of mice with U87/NHA or U87/ fibroblasts was the same as unmixed U87 controls (65 days), whereas the survival of mice with U87/hMSCs was significantly shorter (55 days, $P < 0.05$).

Therefore, BM-hMSCs-like cells can be isolated from human gliomas and may be recruited from the circulation. Although not tumorigenic, BM-hMSCs-like cells promote tumor growth. Thus, the glioma micro-environment includes recruited bone marrow-derived cells that contribute to the aggressiveness of these tumors.

SATURDAY, NOVEMBER 3

10:00 – 10:15 AM

Thrombolysis for Intraventricular Hemorrhage: Results of Phase II Trials and Plans for Phase III

Issam Awad MD (Evanston Northwestern Healthcare, Evanston, IL) and Daniel Hanley MD (Johns Hopkins Medical Center, Baltimore, MD) for the CLEAR IVH Investigators

Intraventricular hemorrhage (IVH) is estimated to afflict 45,000 cases in the United States each year, associated with 40% of intracerebral hemorrhages (ICH), and is an independent determinant of mortality (outcome) in this disease (NIH Stroke databank, STICH and *a*VII trials). There are no validated efficacious treatments in man, but animal models demonstrate a dramatic physiologic and functional impact of thrombolytic enhanced rapid clearance of IVH. We present results of Phase II multi-institutional randomized studies (CLEAR A and B), with feasibility, safety, dose optimization, and preliminary trends on improving outcome in patients with obstructive IVH, external ventricular drain (EVD) placement, small ICH, and no underlying vascular lesion.

CLEAR A (16 subjects) tested dose-response comparing placebo, 0.3 mg, 1 mg and 3 mg rt-PA doses q12hours for four days or until clearance of 3rd and 4th ventricles. CLEAR B (24 subjects) is ongoing, testing shorter dose intervals (Q8⁰ vs. Q12⁰) Primary outcome was total clot lysis rate (C_T) and safety; secondary outcomes were modified Rankin Scale (mRS) and Glasgow Outcome Scale (GOS) at days 30, 90, and 180.

There was a dose dependant decrease in the time until clearance of the 3rd and 4th ventricles (P< 0.02). Interestingly, there was no dose-response relationship between rt-PA and overall IVH CT, but a significant regional dose-response relationship emerged for the treated 3rd and 4th ventricular clot near the catheter delivery site (p<0.001), but not at sites distant from the EVD catheter. Symptomatic bleeding occurred in 23% of cases at the 3mg dose, but in none of the 1 or 0.3 mg doses, or placebo group. Bacterial ventriculitis occurred in 8% of patients. ICP and CPP were controlled (<30 mm Hg) within the targeted normal range 98% of the time

during active treatment, and duration of EVD was shorter in rt-PA cases. All 30 day outcome data pooled from the cases to date demonstrate greater proportion of normal subjects, mRS 0-3 subjects, and mRS 0-4 subjects in treated versus placebo cases. In the Clear B protocol improved functional gain, with 30% of subjects achieving mRS 0-3 at day 180 was observed. Analyses of the longer follow-up and CLEAR B dose comparisons are ongoing.

The safety of IVH thrombolysis, blood clearance effects, and trends of improved outcome as compared to placebo are encouraging. Results of Phase II studies reveal a most optimal IVH clearance-safety profile at 1mg rt-PA dose, with ongoing optimization of dose frequency. A Phase III trial is currently being planned, aiming to enroll 500 subjects from about 50 centers, with intention-to-treat principle, powered to test the primary hypothesis that a practice of early control of ICP with EVD coupled with rt-PA treatment produces improvements in outcome as measured by a dichotomized mRS (defined as a mRS score of ≤ 3 at 180 days post ictus) by a 15% absolute difference (expressed as an odds ratio of 1.8). Such a trial is needed to determine clinical efficacy of IVH thrombolysis as compared to EVD alone. If positive this trial may alter neurosurgical practice, with the potential to save up to 10,000 American lives per year.

SATURDAY, NOVEMBER 3

10:15 – 10:30 AM

The Role of Extent of Resection in the Long-Term Outcome of Low-Grade Hemispheric Gliomas

Justin S. Smith, MD, PhD, Edward F. Chang, MD, Kathleen R. Lamborn, PhD, Susan M. Chang, MD, Michael D. Prados, MD, Soonmee Cha, MD, Tarik Tihan, MD, Scott Vandenberg, MD, Michael W. McDermott, MD, and Mitchel S. Berger, MD

BACKGROUND: The prognostic role of extent of resection (EOR) of low-grade gliomas (LGG) is a major controversy. We designed a retrospective study to assess the influence of EOR on long-term outcomes of LGG.

METHODS: The study population (n=216) included adults undergoing initial resection of hemispheric LGG. Region-of-interest analysis was performed to measure tumor volumes based on FLAIR imaging.

RESULTS: Median pre-operative and post-operative tumor volumes and EOR were 36.6 cm³ (range: 0.7-246.1), 3.7 cm³ (range: 0-197.8) and 88.0% (range: 5-100), respectively. There was no operative mortality. New post-operative deficits were noted in 36 (17%) patients, however all but four had complete recovery. There were 34 (16%) deaths (median follow-up: 4.4 years). Progression and malignant progression were identified in 95 (44%) and 44 (20%) cases, respectively. Patients with ≥90% EOR had 5- and 8-year overall survival (OS) rates of 97% and 91%, respectively, while patients with <90% EOR had 5- and 8-year OS rates of 76% and 60%, respectively. After adjusting each measure of tumor burden for age, KPS, tumor location and tumor subtype, OS was predicted by EOR (HR=0.972, 95%CI=0.960-0.983, P<0.001), log of pre-operative tumor volume (HR=4.442, 95%CI=1.601-12.320, P<0.001), and post-operative tumor volume (HR=1.010, 95%CI=1.001-1.019, P=0.03), progression-free survival was predicted by log of pre-operative tumor volume (HR=2.711, 95%CI=1.590-4.623, P=<0.001) and post-operative tumor volume (HR=1.007, 95%CI=1.001-1.014, P=0.035) and malignant progression-free survival was predicted by

EOR (HR=0.983, 95% CI=0.972-0.995, P=0.005) and log of pre-operative tumor volume (HR=3.826, 95% CI=1.632-8.969, P=0.002).

CONCLUSIONS: Improved outcome among adult patients with hemispheric LGG is predicted by greater EOR.

SATURDAY, NOVEMBER 3

10:30 – 10:45 AM

High-Flow Extracranial to Intracranial (HF-EC-IC) Bypass for Giant Transitional Cavernous-Clinoidal Internal Carotid Artery (ICA) Aneurysms: Assessment of Surgical and Long Term Functional Outcomes

Saleem Abdulrauf, MD, Sonia Teufack, BS, Jeroen Coppens, MD, Raul Olivera, MD, Dana Oliver, MPH

Introduction: Direct microsurgical clipping and endovascular coiling of giant (>2.5cm) transitional cavernous-supraclinoidal ICA aneurysms have inherent limitations due to low efficacy (low angiographic obliteration rates) and/or relatively high treatment associated morbidity and mortality rates. The aim of this project is to assess the use of HF-EC-IC bypass for the treatment of these lesions with specific aims at delineating surgical morbidity (procedure related neurological deficits), mortality, efficacy (angiographic obliteration), and long-term functional outcomes (modified Rankin and Glasgow Outcome Scales).

Methods: IRB approved analysis of 55 consecutive patients with giant transitional cavernous-clinoidal ICA aneurysms treated with HF-EC-IC bypass over 8 years (June 1999 to June 2007) was undertaken.

Results: Mean age: 46.35 years (SD= 8.13). Mean aneurysm size: 34mm (range 25 to 70mm, SD= 6.72). Presentation was categorized into: 1.) cranial neuropathy or documented aneurysmal enlargement (n=23, 42%); 2.) subarachnoid hemorrhage (n=10, 18%); or 3.) headaches or incidental finding (n=22, 40%). WFNS classification for SAH grades: grade 0 (n=45, 81.8%), grade 1 (n=8, 14.5%), grade 2 (n=1, 1.8%), grade 3 (n=1, 1.8%), and grades 4 and 5 (n=0). Mean follow up period: 34.6 months (SD= 21.0). Immediate angiographic graft patency was achieved in 51 patients (93%). Four patients (7%) had acute (within 24 hours of surgery) graft occlusions. Late (>6 months post surgery) graft occlusions were documented in 2 patients (3.6%), for a total long-term graft occlusion rate of 10.9%. One patient (1.8%) died 5 weeks post surgery from a pulmonary embolus. Hospital discharge status: home (n=49, 89.1%), inpatient rehab (n=4,

7.3%), long-term care facility (n=2, 3.6%). Fifty four (98%) patients had complete angiographic aneurysmal occlusion. One patient (2%) had partial aneurysmal recurrence (treatment failure) and underwent endovascular distal ICA occlusion. Modified Rankin Scale grades: grade 0 (n=24, 43.6%), grade 1 (n=26, 47.3%), grade 2 (n=1, 1.8%), grade 3 (n=1, 1.8%), grade 4 (n=2, 3.6%), and grade 5 (n=1, 1.8%). GOS: 5 (n=51, 92.7%), 4 (n=1, 1.8%), 3 (n=1, 1.8%), 2 (n=1, 1.8%), and 1 (n=1, 1.8%).

Conclusions: 55 consecutive patients with giant cavernous-clinoidal ICA aneurysms were treated with HF-EC-IC bypass using radial artery grafts. 54 (98%) patients had angiographic aneurysmal occlusion. Procedure related stroke occurred in 4 patients (7%). One patient died (1.8%). Long term graft patency rate was 89%. Decision making protocols and potential mechanisms contributing to graft occlusions (n=6) will be discussed. Forty nine (89%) patients were discharged directly home. Fifty (91%) patients had modified Rankin Scale grades of 0 or 1 (non-symptomatic, able to carry out usual activities). GOS of 5 (good) was achieved in 51 (93%) patients. HF-EC-IC bypass for transitional proximal ICA giant aneurysms appears to provide high efficacy, relatively low morbidity and mortality, and good quality of life measurement scores for the majority of the patients.

SATURDAY, NOVEMBER 3

10:45 – 11:00 AM

Computer Numerical Control Machine Tool for Automated Drilling of the Skull Base

William T. Couldwell, J.D. MacDonald,
A.K. Balaji, C.L. Thomas

Drilling of the skull base, for example, the temporal bone for transtemporal procedures, may be a time-consuming process. Because of the complex anatomy of this region, this bone-drilling operation is labor intensive and facilitated by surgeon experience. Using existing technology that translates medical image data to computer-automated design (CAD) models and prototypes, the authors have developed a CAD-integrated machine tool to automate drilling of the convexity in the skull or temporal bone during neurosurgery. Software has been developed for generating the tool path of the drill bit from simple planning software using preoperative computed tomography images.

Reduced operative costs will be realized because of the decrease in time required to perform the bone removal. The accuracy of the semi-automated drilling system is within 1 mm, and its employment reduces drilling time of a translabyrinthine approach to less than 5 minutes. The design and use of the system will be discussed.

SATURDAY, NOVEMBER 3

11:00 – 11:15 AM

Intra-Arterial Calcium Channel Blockade for Cerebral Vasospasm, a Comparison of Intra-carotid Nicardipine and Verapamil: Augmentation of Cerebral Blood Flow and Reversal of Endothelin-1 Induced Vasospasm

Sean D. Lavine, MD, Mei Wang, MPH,
Joshua J. Etu, BA, Philip M. Meyers, MD
Shailendra Joshi, MD

Objective: Local intra-arterial infusions of verapamil and nicardipine have been used to treat human cerebral vasospasm. Only a few reports of early clinical experience with these medications are currently available, and limited data is available on their cerebral physiological activity. We assessed the efficacy of intracarotid verapamil and nicardipine on augmenting cerebral blood flow (CBF) of New Zealand White rabbits and compared the ability of these drugs to reverse topical endothelin-1 (ET-1) triggered vasospasm.

Methods: In the first group of New Zealand white rabbits, CBF (laser Doppler) and systemic hemodynamic measurements were recorded at baseline and with increasing intracarotid doses of verapamil and nicardipine. In the second group, topical ET-1 (10^{-4} M) was applied in an acutely implanted cranial window. Dose responses to non-specific reversal of ET-1 induced vasospasm were evaluated with intra-arterial nicardipine and verapamil.

Results: The dose-response studies revealed that intracarotid nicardipine, compared to verapamil, was more effective in augmenting CBF. Topical ET-1 induced vasospasm was reversed completely by nicardipine, and partially by verapamil.

Conclusion: This study suggests that intraarterial nicardipine is a more potent cerebral vasodilator and is superior to verapamil for treating ET-1 induced experimental cerebral vasospasm, and supports further investigation of these agents in SAH induced vasospasm.

SATURDAY, NOVEMBER 3

11:15 – 11:30 AM

Understanding the Role of the Folate Pathway in Repair of the Injured CNS

Bermans J. Iskandar, MD

Introduction: Folic acid supplementation decreases the occurrence of congenital anomalies such as neural tube defects. This implies that folic acid plays an important role in central nervous system (CNS) growth and development. Since some of the same signals needed for growth of the embryonic CNS are thought to be used for regeneration and repair of the adult CNS after injury, we investigated the relationship between the folate pathway and the adult CNS in animals.

Methods: Over the past 5 years, we have used anatomical, biochemical, and molecular techniques to study the response of the folate pathway to CNS injury, and the extent to which folate can enhance neuronal and neurological recovery in the spinal cord and optic pathway. Some of these results are summarized here, some of which have been published.

Results: We have shown the following statistically significant ($p < 0.05$) results: **1.** Folic acid treatment improves *in vivo* neuronal regeneration in the adult rat optic pathway after optic nerve injury, and the spinal cord after dorsal column injury. The latter shows a parabolic dose response curve, with maximal spinal regeneration occurring at 80 μ g/kg of folic acid. These results are superior to all other published therapeutic interventions in the injured CNS. **2.** Folic acid treatment improves recovery of locomotion in a rat spinal cord contusion model (1 and 2: Iskandar, et al. *Ann Neurol* 2004). **3.** The high affinity folate receptor (folbp1) but not the low affinity reduced folate carrier (RFC-1) mRNA is upregulation after spinal cord injury. **4.** In turn, spinal regeneration is significantly decreased in the heterozygous folbp1 knockout compared to wild-type mice. **5.** Shutting down DHFR (bioactivation step) and methionine synthase (methionine methylation part of the folate pathway) activities *in vivo* decreases the ability of the optic pathway and spinal cord to regenerate after injury. **6.** Global DNA methylation measurements in the injured spinal cord with increasing doses of folic acid show a

biphasic dose response curve that corresponds inversely to the biphasic curve seen with regeneration (see 1). **7.** The methylation response of the CNS to injury is confirmed through significant changes in the levels of methylation metabolites, and the expression and activity of the main methylation enzymes, the methyltransferases.

Conclusions: The folate pathway plays a crucial role in the regeneration and repair of the adult CNS after injury, and is not restricted to the embryonic period. Such a response is manifest at the level of folate entry into the cell (receptors), bioactivation (DHFR), and methylation, and can be shut down by turning off specific enzymes in the folate and methionine-methylation pathway. These are novel findings not previously reported in the literature, and which are important in determining the mechanism of CNS repair, and optimizing its therapy.

SPECIAL GUESTS

GUESTS

Saleem Abdulrauf
St. Louis, MO

Kazuhide Adachi (Resident)
Tokyo, Japan

Anthony Asher
Charlotte, NC

Fred Barker
Boston, MA

Paul Camarata
Overland Park, KS

Bob Carter
Boston, MA

Chun Siang Chen
New York, NY

Clark Chen (Resident)
Boston, MA

John Duncan
Barrington, RI

John Fildes
Las Vegas, NV

Brian Hoh
Boston, MA

Bermans Iskandar
Madison, WI

David Jimenez
San Antonio, TX

Paul Kongkham (Resident)
Toronto, Ontario, Canada

SPONSORS

Albert Rhoton

Takashi Kawase

Douglas Kondziolka

Larry Borges

Jaques Morcos

Nick Zervas

Martin Camins

Academy Award
Runner-up

James Rutka

Academy

William Friedman

Robert Dempsey

Jim Story

Academy Award
Winner

Frederick Lang
Houston, TX

Raymond Sawaya

Sean Lavine
New York, NY

Robert Solomon

Elad Levy
Buffalo, NY

Nick Hopkins

Michael Link
Rochester, MN

Bruce Pollock

Helen Mayberg
Atlanta, GA

Academy

Rajiv Midha
Calgary, Alberta, Canada

Chris Wallace

Praveen Mummaneni
San Francisco, CA

Michael Lawton

Karin Muraszko
Ann Arbor, MI

William Chandler

Alessandro Olivi
Baltimore, MD

Don Long

Troy Payner
Indianapolis, IN

John Tew

Zach Ray (Resident)
St. Louis, MO

Ralph Dacey

Charles Rich
Salt Lake City, UT

J. Charles Rich

Gregory Riggins
Baltimore, MD

Academy

Mitesh Shah
Indianapolis, IN

Paul Nelson

Nathan Simmons
Lebanon, NH

David Roberts

Justin Smith (Resident)
Charlottesville, VA

Mitch Berger

Rafael Tamargo
Baltimore, MD

Henry Brem

Nitin Tandon
Houston, TX

Dennis Vollmer

Phillip Tibbs
Lexington, KY

Russell Travis

Bruce Tranmer
Burlington, VT

Johnny Delashaw

Yong-Kwang Tu
Taipei, Taiwan

Roberto Heros

Alex Valadka
Houston, TX

Academy

Michael Vogelbaum
Cleveland, OH

Gene Barnett

ACADEMY AWARD WINNERS

Paul M. Lin	1955
Hubert L. Rosomoff	1956
Byron C. Pevehouse	1957
Norman Hill	1958
Jack Stern	1959
Robert Ojemann	1960
Lowell E. Ford	1962
Charles H. Tator	1963
Earle E. Crandall	1964
Stephen Mahaley, Jr.	1965
Chun Ching Kao.....	1966
John P. Kapp	1967
Yoshio Hosobuchi	1968
Gary G. Ferguson	1970
Richard L. Pressley	1971
David G. McLone.....	1972
Arden F. Reynolds, Jr.....	1973
Richard L. Rapport.....	1974
Andrew G. Shetter	1975
John R. Howe	1976
Howard W. Blume	1977
Howard J. Senter	1978
Elisabeth M. Post	1979
David Dubuisson.....	1980
Dennis A. Turner.....	1981
Marc R. Mayberg	1982
David S. Baskin.....	1983
Kevin J. Kiwak.....	1984
Terry Lichtor	1985
Michael G. Nosko	1986
Joseph R. Madsen.....	1987
James T. Rutka	1988
Christopher D. Heffner.....	1989
Scott I. Gingold	1990
Mary Louise Hlavin	1991
Adam P. Brown	1992
Michael Tymianski.....	1993
David Garrett, Jr.....	1994
L. Brannon Thomas.....	1995
John S. Yu.....	1996
Gregory W. Canute.....	1997

Nathan R. Selden.....1998
Robert M. Friedlander.....1999
Tien T. Nguyen.....2000
Peng Chen.....2001
Ganesh Rao.....2002
Gelareh Zadeh.....2003
Eric C. Leuthardt.....2004
Manish Aghi.....2005
Alfred T. Ogden.....2006
Paul Kongkham.....2007

MEETINGS OF THE ACADEMY

Hotel Netherland Plaza, Cincinnati, Ohio	October 28-29, 1938
Roosevelt Hotel, New Orleans, Louisiana	October 27-29, 1939
Tudor Arms Hotel, Cleveland, Ohio	October 21-22, 1940
Mark Hopkins Hotel, San Francisco and Ambassador Hotel, Los Angeles, California.....	November 11-15, 1941
The Palmer House, Chicago, Illinois	October 16-17, 1942
Hart Hotel, Battle Creek, Michigan.....	September 17-18, 1943
Ashford General Hospital, White Sulphur Springs, West Virginia	September 7-9, 1944
The Homestead, Hot Springs, Virginia	September 9-11, 1946
Broadmoor Hotel, Colorado Springs, Colorado	October 9-11, 1947
Windsor Hotel, Montreal, Canada	September 20-22, 1948
Benson Hotel, Portland, Oregon	October 25-27, 1949
Mayo Clinic, Rochester, Minnesota	September 28-30, 1950
Shamrock Hotel, Houston, Texas	October 4-6, 1951
Waldorf-Astoria Hotel, New York City, New York	September 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California	October 12-14, 1953
Broadmoor Hotel, Colorado Springs, Colorado	October 21-23, 1954
The Homestead, Hot Springs, Virginia	October 27-29, 1955
Camelback Inn, Phoenix, Arizona	November 8-10, 1956
The Cloister, Sea Island, Georgia	November 11-13, 1957
The Royal York Hotel, Toronto, Canada	November 6-8, 1958
Del Monte Lodge, Pebble Beach, California.....	October 18-21, 1959
Copley Sheraton Plaza, Boston, Massachusetts	October 5-8, 1960
Royal Orleans, New Orleans, Louisiana	November 7-10, 1962
El Mirador, Palm Springs, California	October 23-26, 1963
The Key Biscayne, Miami, Florida	November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio	October 14-16, 1965
Fairmont Hotel & Towers, San Francisco, California	October 17-19, 1966
The Key Biscayne, Miami, Florida	November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado	October 6-8, 1968
St. Regis Hotel, New York City	September 21, 1969
Camino Real, Mexico City, Mexico.....	November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada	September 26-30, 1971
New College, Oxford, England	September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California...	November 14-17, 1973
Southampton Princess Hotel, Bermuda.....	November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona ...	November 5-8, 1975
Mills Hyatt House, Charleston, South Carolina	November 10-13, 1976

Mauna Kea Beach Hotel, Kamuela, Hawaii November 2-5, 1977
 Hotel Bayerischer Hof, Munich, Germany October 22-25, 1978
 Hyatt Regency, Memphis, Tennessee November 7-10, 1979
 Waldorf-Astoria Hotel, New York City, New York October 1-4, 1980
 Sheraton Plaza, Palm Springs, California November 1-4, 1981
 Ritz-Carlton Hotel, Boston, Massachusetts October 10-13, 1982
 The Lodge at Pebble Beach, California October 23-26, 1983
 The Homestead, Hot Springs, Virginia October 17-20, 1984
 The Lincoln Hotel Post Oak, Houston, Texas October 27-30, 1985
 The Cloister, Sea Island, Georgia November 5-8, 1986
 Hyatt Regency, San Antonio, Texas October 7-10, 1987
 Omni Netherland Plaza, Cincinnati, Ohio September 13-17, 1988
 Loews Ventana Canyon, Tucson,
 Arizona September 27-October 1, 1989
 Amelia Island Plantation, Amelia Island, Florida October 2-7, 1990
 Salishan Lodge, Gleneden Beach, Oregon September 22-26, 1991
 Ritz-Carlton Hotel, Naples, Florida October 21-25, 1992
 The Wigwam, Phoenix, Arizona October 27-30, 1993
 The Cloister, Sea Island, Georgia November 3-6, 1994
 Loews Ventana Canyon Resort, Tucson, Arizona November 1-5, 1995
 The Greenbrier, White Sulphur Springs,
 West Virginia September 18-22, 1996
 Rimrock Resort, Banff, Alberta, Canada September 10-14, 1997
 Four Seasons Biltmore, Santa Barbara, California November 4-7, 1998
 Ritz-Carlton, Amelia Island, Florida November 10-13, 1999
 The Broadmoor, Colorado Springs, Colorado October 11-14, 2000
 The Breakers, Palm Beach, Florida November 14-17, 2001
 The Phoenician, Scottsdale, Arizona October 16-19, 2002
 Colonial Williamsburg, Williamsburg, VA .October 29-November 1, 2003
 Four Seasons Berlin and
 Taschenbergpalais Dresden, Germany October 3-8, 2004
 Ritz Carlton Half Moon Bay, California September 21-24, 2005
 Ritz Carlton Reynolds Plantation, Greensboro, GA October 18-21, 2006
 Ritz Carlton Lake Las Vegas, NV October 31-November 4, 2007

PAST PRESIDENTS

Dean H. Echols..... 1938-39	Sidney Goldring 1983
Spence Braden..... 1940	Russel H. Patterson, Jr..... 1984
Joseph P. Evans 1941	Thomas Langfitt 1985
Francis Murphey 1942	Phanor L. Perot, Jr..... 1986
Frank H. Mayfield..... 1943	Shelley N. Chou 1987
A. Earl Walker 1944	James T. Robertson 1988
Barnes Woodhall..... 1946	Thoralf M. Sundt, Jr. 1989
William S. Keith..... 1947	Robert Ojemann 1990
Howard A. Brown 1948	Nicholas Zervas..... 1991
John Raaf..... 1949	Henry Garretson 1992
E. Harry Botterell..... 1950	George Tindall 1993
Wallace B. Hamby 1951	William A. Buchheit 1994
Henry G. Schwartz 1952	David L. Kelly, Jr. 1995
J. Lawrence Pool 1953	John M. Tew, Jr..... 1996
Rupert B. Raney..... 1954	Julian T. Hoff 1997
David L. Reeves 1955	Edward Connolly 1998
Stuart N. Rowe 1956	J. Charles Rich 1999
Arthur R. Elvidge..... 1957	George A. Ojemann.....2000
Jess D. Herrmann 1958	Roberto C. Heros.....2001
Edwin B. Boldrey..... 1959	Donald O. Quest.....2002
George S. Baker 1960	David G. Piepgras.....2003
C. Hunter Shelden 1961-62	Volker K.H. Sonntag.....2004
Samuel R. Snodgrass 1963	Martin B. Camins.....2005
Theodore B. Rasmussen..... 1964	L. Nelson Hopkins.....2006
Edmund J. Morrissey 1965	Richard Morawetz.....2007
George Maltby 1966	
Guy L. Odom 1967	
James G. Galbraith 1968	
Robert H. Pudenz 1969-70	
William B. Scoville 1971	
Robert L. McLaurin 1972	
Lyle A. French 1973	
Benjamin B. Whitcomb..... 1974	
John R. Green..... 1975	
William H. Feindel 1976	
William H. Sweet..... 1977	
Arthur A. Ward 1978	
Robert B. King 1979	
Eben Alexander, Jr. 1980	
Joseph Ransohoff II 1981	
Byron C. Pevehouse 1982	

PAST VICE-PRESIDENTS

Francis Murphey	1941	Robert G Fisher	1978
William S Keith.....	1942	H Thomas Ballantine, Jr	1979
John Raaf.....	1943	George Ehni.....	1980
Rupert B Raney	1944	Courtland H Davis, Jr	1981
Arthur R Elvidge.....	1946	John F Mullan.....	1982
F Keith Bradford	1949	Hugo V Rizzoli.....	1983
David L Reeves	1950	James W Correll	1984
Henry G Schwartz	1951	E Bruce Hendrick	1985
J Lawrence Pool	1952	Griffith R Harsh, III.....	1986
Rupert B Raney	1953	Ellis B Keener	1987
David L Reeves	1954	Robert Grossman	1988
Stuart N Rowe	1955	Jim Story	1989
Jess D Hermann.....	1956	John Jane, Sr.....	1990
George S Baker	1957	Stewart Dunsker	1991
Samuel R Snodgrass.....	1958	Burton M Onofrio.....	1992
C Hunter Shelden	1959	Martin H Weiss	1993
Edmund Morrissey	1960	John M Tew, Jr.....	1994
Donald F Coburn	1961-62	John C VanGilder.....	1995
Eben Alexander, Jr	1963	Edward Connolly.....	1996
George L Maltby	1964	George Ojemann.....	1997
Robert Pudenz	1965	Charles H Tator	1998
Francis A Echlin.....	1966	Donald O Quest	1999
Benjamin Whitcomb	1967	Howard M. Eisenberg.....	2000
Homer S Swanson	1968	Richard B Morawetz.....	2001
Augustus McCravey.....	1969-70	Martin B Camins.....	2002
Edward W Davis	1971	Arthur L Day.....	2003
John R Green.....	1972	William F. Chandler.....	2004
George J Hayes	1973	Steven L. Gianotta.....	2005
Richard L DeSaussure.....	1974	Robert F. Spetzler.....	2006
Ernest W Mack.....	1975	Griffith R. Harsh IV.....	2007
Frank E Nulsen.....	1976		
Robert S Knighton.....	1977		

PAST SECRETARY-TREASURERS

Francis Murphey	1938-40	Eben Alexander	1954-57
A. Earl Walker	1941-43	Robert L. McLaurin.....	1958-62
Theodore C. Erickson.....	1944-47	Edward W. Davis	1963-65
Wallace B. Hamby	1948-50	Robert G. Fisher	1966-68
Theodore B. Rasmussen....	1951-53	Byron C. Pevehouse	1969-72

PAST SECRETARIES

Byron C. Pevehouse	1973	William A. Buchheit.....	1990-92
Russel H. Patterson, Jr.	1974-76	Julian T. Hoff	1992-95
Phanor L. Perot, Jr.....	1977-80	Roberto C. Heros	1995-98
John T. Garner.....	1981-83	David G. Piepgras.....	1999-01
James T. Robertson	1984-86	L. Nelson Hopkins.....	2002-04
Nicholas T. Zervas	1987-89	Ralph G. Dacey, Jr.....	2004-07

PAST TREASURERS

Russel H. Patterson, Jr.	1973	Julian T. Hoff	1990-92
Phanor L. Perot, Jr	1974-76	Roberto C. Heros	1992-95
John T. Garner	1977-80	David G. Piepgras	1995-98
James T. Robertson	1981-83	L. Nelson Hopkins.....	1999-01
Nicholas T. Zervas	1984-86	Ralph G. Dacey, Jr.....	2002-04
William A. Buchheit	1987-89	James T. Rutka.....	2004-07

HONORARY MEMBERS

- GUY LAZORTES** (Annick)..... Elected 1973
5 Allee Charles Martel
31300 Toulouse
FRANCE
05 3451 3215
- KEIJI SANO** (Yaeko)..... 1975
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SENIOR MEMBERS

(Elected)

- MICHAEL APUZZO**.....1988
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323-226-7421, fax 323-226-5897, apuzzo@usc.edu
- JAMES AUSMAN (Carolyn)** 1979
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Rancho Mirage CA 92270
760-779-8253, fax 760-779-8254, j.ausman@verizon.net
- DONALD BECKER (Maria)**1990
Division of Neurosurgery
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10833 LeConte Avenue
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- GILLES BERTRAND (Louise)**.....1967
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- KJELD VAERNET**1970
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- SYDNEY ERIC WATKINS** (Susan)1975
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CORRESPONDING MEMBERS

Elected

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- EDWARD MEE** (Jane Elliott).....2005
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 649-520-9672, fax 649-520-9673, Edward.mee@xtra.co.nz
- MICHAEL MORGAN** (Elizabeth)1999
 Neurosurgery
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 193 Macquarie Street
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- M. NECMETTIN PAMIR** (Feriha).....2006
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 TURKEY
 902165714483, fax 902166588456, pamirn@yahoo.com
- GABRIEL SCHACKERT** (Hans)2003
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DECEASED MEMBERS

	Elected	Deceased
EBEN ALEXANDER, JR.	1950	2004
Winston-Salem, North Carolina		
(Senior)		
JAMES R. ATKINSON	1970.....	1978
Phoenix, Arizona		
(Active)		
PERCIVAL BAILEY	1960.....	1973
Evanston, Illinois		
(Honorary)		
GEORGE BAKER	1940.....	1993
Litchfield Park, Arizona		
(Senior)		
H. THOMAS BALLANTINE, JR.	1951	1996
Boston, Massachusetts		
(Senior)		
WILLIAM F. BESWICK	1959.....	1971
Buffalo, New York		
(Active)		
EDWIN B. BOLDREY	1941	1988
San Francisco, California		
(Senior)		
E. HARRY BOTTERELL	1938.....	1997
Kingston, Ontario, CANADA		
(Senior)		
ROBERT BOURKE	1983.....	1996
Rockville, Maryland		
(Senior)		
SPENCER BRADEN	Founder	1969
Cleveland, Ohio		
(Active)		

- F. KEITH BRADFORD**..... 1938.....1971
Houston, Texas
(Active)
- JEAN BRIHAYE**..... 1975.....1999
Bruxelles, BELGIUM
(Senior Corresponding)
- KARL-AUGUST BUSHE**..... 1972.....1999
Wurzburg, GERMANY
(Senior Corresponding)
- HOWARD BROWN** 1939.....1990
San Francisco, California
(Senior)
- JUAN CARDENAS**..... 1966.....1996
Mexico City, MEXICO
(Senior Corresponding)
- HARVEY CHENAULT**.....1949.....2006
Lexington, KY
(Senior)
- SHELLEY CHOU** 1974.....2001
Rio Verde, Arizona
(Senior)
- JUAN CARLOS CHRISTENSEN** ... 1970.....2003
Buenos Aires, ARGENTINA
(Senior Corresponding)
- GALE CLARK**..... 1970.....1996
Oakland, California
(Senior)
- DONALD COBURN** 1938.....1988
Wilmington, Delaware
(Senior)
- JAMES CORRELL** 1966.....2004
Hampstead, North Carolina
(Senior)

WINCHELL McK. CRAIG	1942.....	1960
Rochester, Minnesota (Honorary)		
EDWARD DAVIS	1949.....	1988
Portland, Oregon (Senior)		
PEARDON DONAGHY	1970.....	1991
Burlington, Vermont (Senior)		
CHARLES DRAKE	1958.....	1998
London, Ontario, CANADA (Senior)		
FRANCIS ECHLIN	1944.....	1988
New Paltz, New York (Senior)		
DEAN ECHOLS	Founder	1991
New Orleans, Louisiana (Senior)		
GEORGE EHNI	1964.....	1986
Houston, Texas (Senior)		
ARTHUR ELVIDGE	1939.....	1985
Montreal, Quebec, CANADA (Senior)		
THEODORE ERICKSON	1940.....	1986
Madison, Wisconsin (Senior)		
JOSEPH EVANS	Founder	1985
Kensington, Maryland (Senior)		
ROBERT FISHER	1955.....	2003
Granada Hills, California (Senior)		

JOHN FRENCH	1951.....	1989
Los Angeles, California (Senior)		
LYLE FRENCH	1954	2004
Scottsdale, Arizona (Senior)		
JAMES GALBRAITH	1947.....	1997
Birmingham, Alabama (Senior)		
SIDNEY GOLDRING	1964.....	2004
St. Louis, Missouri (Senior)		
EVERETT GRANTHAM	1942.....	1997
Louisville, Kentucky (Senior)		
JOHN GREEN	1953.....	1990
Phoenix, Arizona (Senior)		
JAMES GREENWOOD, JR.	1952.....	1992
Houston, Texas (Senior)		
WESLEY GUSTAFSON	1942.....	1975
Jensen Beach, Florida (Senior)		
WALLACE HAMBY	1941.....	1999
Pompano Beach, Florida (Senior)		
HANNIBAL HAMLIN	1949.....	1982
Providence, Rhode Island (Senior)		
JOHN HANBERY	1959.....	1996
Palo Alto, California (Senior)		

MAJOR GEN. GEORGE HAYES ... 1962	2002
Washington, D. C. (Senior)	
E. BRUCE HENDRICK 1968.....	2001
Toronto, Ontario, CANADA (Senior)	
JESS HERRMANN 1938.....	1994
Oklahoma City, Oklahoma (Senior)	
HENRY HEYL 1951.....	1975
Hanover, New Hampshire (Senior)	
JULIAN HOFF1975.....	2007
Ann Arbor, MI (Senior)	
HAROLD HOFFMAN1982.....	2004
Toronto Ontario, Canada (Senior)	
WILLIAM HUNT 1970.....	1999
Columbus, Ohio (Senior)	
OLAN HYNDMAN 1942.....	1966
Iowa City, Iowa (Senior)	
KENNETH JAMIESON 1970.....	1976
Brisbane, AUSTRALIA (Corresponding)	
SIR GEOFFREY JEFFERSON 1951.....	1961
Manchester, ENGLAND (Honorary)	
HANS-PETER JENSEN 1980.....	2000
Kiel, GERMANY (Senior Corresponding)	

RICHARD JOHNSON	1974.....	1997
Manchester, ENGLAND (Senior Corresponding)		
WILLIAM KEITH	Founder.....	1987
Toronto, Ontario, CANADA (Senior)		
KATSUTOSHI KITAMURA	1970.....	2005
Japan (Senior Corresponding)		
ROBERT KNIGHTON	1966.....	2004
Cherry Valley, California (Senior)		
RICHARD KRAMER	1978.....	2001
Durham, North Carolina (Inactive)		
HUGO KRAYENBUHL	1974.....	1985
Zurich, SWITZERLAND (Honorary)		
KRISTIAN KRISTIENSEN	1967.....	1993
Oslo, Norway (Senior Corresponding)		
THEODORE KURZE	1967.....	2002
Newport Beach, California (Senior)		
THOMAS LANGFITT	1971.....	2005
Philadelphia, Pennsylvania (Senior)		
WALPOLE LEWIN	1973.....	1980
Cambridge, ENGLAND (Corresponding)		
VALENTINE LOGUE	1974.....	2000
London, ENGLAND (Honorary)		

WILLIAM LOUGHEED	1962.....	2004
Toronto, Ontario, Canada (Senior)		
HERBERT LOURIE	1965.....	1987
Syracuse, New York (Senior)		
WILLEM LUYENDIJK	1973.....	1995
Oegstgeest, NETHERLANDS (Senior Corresponding)		
ERNEST MACK	1956.....	2000
Reno, Nevada (Senior)		
M. STEPHEN MAHALEY	1972.....	1992
Birmingham, Alabama (Active)		
LEONARD MALIS	1973.....	2005
Hollis Hills, New York (Senior)		
GEORGE MALTBY	1942.....	1988
Scarsborough, Maine (Senior)		
FRANK MARGUTH	1978.....	1991
Munich, GERMANY (Senior Corresponding)		
DONALD MATSON	1950.....	1969
Boston, Massachusetts (Active)		
FRANK MAYFIELD	Founder.....	1991
Cincinnati, Ohio (Senior)		
AUGUSTUS McCRAVEY	1944.....	1990
Chattanooga, Tennessee (Senior)		

KENNETH McKENZIE	1960.....	1964
Toronto, Ontario, CANADA (Honorary)		
J. MICHAEL MCWHORTER	1989.....	2004
Winston-Salem, North Carolina (Senior)		
WILLIAM MEACHAM	1952.....	1999
Nashville, Tennessee (Senior)		
JAMES MEREDITH	1946.....	1962
Richmond, Virginia (Active)		
J. DOUGLAS MILLER	1988.....	1995
Edinburgh, SCOTLAND (Corresponding)		
W. JASON MIXTER	1951	1968
Woods Hole, Massachusetts (Honorary)		
EDMUND MORRISSEY	1941	1986
San Francisco, California (Senior)		
FRANCIS MURPHEY	Founder	1994
Naples, Florida (Senior)		
GOSTA NORLEN	1973.....	1985
Goteborg, SWEDEN (Honorary)		
FRANK NULSEN	1956.....	1994
Naples, Florida (Senior)		
SIXTO OBRADOR	1973.....	1978
Madrid, SPAIN (Honorary)		

GUY ODOM	1946.....	2001
Durham, North Carolina (Senior)		
PIETRO PAOLETTI	1989.....	1991
Milan, ITALY (Corresponding)		
WILDER PENFIELD	1960.....	1976
Montreal, Quebec, CANADA (Honorary)		
HELMUT PENZHOLZ	1978.....	1985
Heidelberg, WEST GERMANY (Corresponding)		
BERNARD PERTUISET	1986.....	2000
Paris, FRANCE (Honorary)		
HANS-WERNER PIA	1978.....	1986
Giessen, WEST GERMANY (Corresponding)		
J. LAWRENCE POOL	1940.....	2004
Canaan, CT (Senior)		
ROBERT PUDENZ	1943.....	1998
South Pasadena, California (Senior)		
JOHN RAAF	Founder.....	2000
Portland, Oregon (Senior)		
B. RAMAMURTHI	1973.....	2003
Tharamani, Chennai, INDIA (Senior Corresponding)		
AIDAN RANEY	1946.....	2002
Los Angeles, California (Senior)		

RUPERT RANEY	1939.....	1959
Los Angeles, California		
(Active)		
JOSEPH RANSOHOFF	1965.....	2001
Tampa, Florida		
(Senior)		
THEODORE RASMUSSEN	1947.....	2002
Montreal, Quebec, CANADA		
(Senior)		
BRONSON RAY	1992.....	1993
New York, New York		
(Honorary)		
DAVID REEVES	1939.....	1970
Santa Barbara, California		
(Active)		
DAVID REYNOLDS	1964.....	1978
Tampa, Florida		
(Active)		
R. C. L. ROBERTSON	1946.....	1985
Houston, Texas		
(Senior)		
STEWART ROWE	1938.....	1984
Pittsburgh, Pennsylvania		
(Senior)		
RICHARD SCHNEIDER	1970.....	1986
Ann Arbor, Michigan		
(Senior)		
KURT-FRIEDRICH SCHURMANN ..	1978.....	2005
Germany		
(Senior Corresponding)		
HENRY SCHWARTZ	1942.....	1998
St. Louis, Missouri		
(Senior)		

WILLIAM SCOVILLE	1944.....	1984
Hartford, Connecticut (Senior)		
R. EUSTACE SEMMES	1955.....	1982
Memphis, Tennessee (Honorary)		
C. HUNTER SHELDEN	1941.....	2003
Pasadena, California (Senior)		
ROBERT SMITH	1989.....	2003
Jackson, Mississippi (Senior)		
SAMUEL SNODGRASS	1939.....	1975
Galveston, Texas (Senior)		
GLEN SPURLING	1942.....	1968
La Jolla, California (Honorary)		
C. WILLIAM STEWART	1948.....	1948
Montreal, Quebec, CANADA (Corresponding)		
KENICHIRO SUGITA	1988.....	1994
Nagoya, Japan (Senior Corresponding)		
THORALF SUNDT, JR.	1971.....	1992
Rochester, Minnesota (Active)		
HENDRIK SVIEN	1957.....	1972
Rochester, Minnesota (Active)		
HOMER SWANSON	1949.....	1987
Atlanta, Georgia (Senior)		

WILLIAM SWEET	1950.....	2001
Brookline, Massachusetts (Senior)		
ALFRED UIHLEIN	1950.....	1990
Rochester, Minnesota (Senior)		
JOHN VAN GILDER	1980.....	2007
Iowa City, Iowa (Senior)		
A. EARL WALKER	1938.....	1995
Albuquerque, New Mexico (Senior)		
ARTHUR WARD, JR.	1953.....	1997
Seattle, Washington (Senior)		
THOMAS WEAVER, JR.	1943.....	1985
Dayton, Ohio (Senior)		
W. KEASLEY WELCH	1957.....	1996
Waban, Massachusetts (Senior)		
BENJAMIN WHITCOMB	1947.....	1998
Surrey, Maine (Senior)		
BARNES WOODHALL	1941.....	1985
Durham, North Carolina (Senior)		
FRANK WRENN	1973.....	1990
Greenville, South Carolina (Senior)		