THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY

68th Annual Meeting

Ritz-Carlton Lodge, Reynolds Plantation
Greensboro, Georgia

October 18 – 21, 2006

American Association of Neurological Surgeons

Jointly Sponsored by the American Association of Neurological Surgeons

FUTURE MEETINGS

2007

October 31- November 3
Ritz-Carlton
Lake Las Vegas. NV

Mark your calendars now!

GENERAL INFORMATION

HOTEL INFORMATION

The Ritz-Carlton Lodge, Reynolds Plantation
One Lake Oconee Trail
Greensboro, Georgia 30642

Telephone: 706-467-0600
Facsimile: 706-467-7124

MEETING REGISTRATION

Wednesday, Oct. 18th	Meadowlark	12:00 PM – 6:30 PM
Thursday, Oct. 19th	Salon Pre-function	6:30 AM – 1:00 PM
Friday, Oct 20th	Salon Pre-function	6:30 AM – 1:00 PM
Saturday, Oct. 21st	Salon Pre-function	6:30 AM – 1:00 PM

PROGRAM SUMMARY

WEDNESDAY, OCTOBER 18TH

EVENT	TIME	LOCATION
Registration	12:00 PM – 6:30 PM	Meadowlark
Executive Committee Meeting	2:00 PM – 5:00 PM	Directors Room
OPENING RECEPTION		
Cocktail Dinner *CASUAL ATTIRE*	6:00 PM – 8:30 PM	Events Lawn (Salon I Backup)

THURSDAY, OCTOBER 19TH

EVENT	TIME	LOCATION
Registration	6:30 AM – 1:00 PM	Salon Pre-function
Business Breakfast *MEMBERS ONLY*	6:30 AM – 7:30 AM	Salon I

EVENT	TIME	LOCATION
Scientific Session	7:30 AM – 1:00 PM	Salon II & III
Spouse & Guest Breakfast	6:30 AM – 9:30 AM	Reynolds Ballroom
Spouse Program	9:30 AM – 11:30 AM	Reynolds Ballroom
AFTERNOON ACTIVITIES		
Golf	Tee Times from 1:10 PM	Oconee Course
EVENING		
Buffalo Night *CASUAL ATTIRE*	6:30 PM – 9:30 PM	The Landing (Back up Salon I/II)
Reception Dinner	6:30 PM – 7:30 PM 7:30 PM – 9:30 PM	

FRIDAY, OCTOBER 20TH

EVENT	TIME	LOCATION
Registration	6:30 AM – 1:00 PM	Salon Pre-function
Business Breakfast *MEMBERS ONLY*	6:30 AM – 7:30 PM	Salon I
Scientific Session	7:30 AM – 1:00 PM	Salon II & III
Spouse & Guest Breakfast	6:30 AM – 9:30 PM	Reynolds Ballroom
Spouse Program	9:30 AM – 11:30 PM	Reynolds Ballroom
Presidential Address	12:15 PM – 1:00 PM	Salon II & III

AFTERNOON ACTIVITIES

EVENT	TIME	LOCATION
Golf	Tee times from 1:10 PM	National Course
EVENING		
Presidential Reception	6:30 PM – 7:30 PM	Event Lawn (Back Up Salon Pre-function)
Black Tie *(optional)* Dinner & Dance	7:30 PM – 11:00 PM	Salon I & II

SATURDAY, OCTOBER 21ST

EVENT	TIME	LOCATION
Registration	6:30 AM – 1:00 PM	Salon Pre-function
Breakfast for Members, Spouse, & Guest	6:30 AM – 9:30 PM	Reynolds Ballroom
Scientific Session	7:30 AM – 1:00 PM	Salon II & III
Gathering	4:30 PM –6:30 PM	Events Lawn

2006 OFFICERS

PRESIDENT
L. Nelson (Nick) Hopkins, III

PRESIDENT-ELECT
Richard B. Morawetz

VICE PRESIDENT
Robert F. Spetzler

SECRETARY
Ralph G. Dacey, Jr.

TREASURER
James T. Rutka

EXECUTIVE COMMITTEE
L. Nelson Hopkins, III
Richard B. Morawetz
Robert F. Spetzler
Ralph G. Dacey, Jr.
James T. Rutka
Martin B. Camins
Volker K.H. Sonntag
Daniel L. Barrow

HISTORIAN
David G. Piepgras

ACADEMY COMMITTEES 2006

Academy Award Committee:
Charles Branch, Chairman
Fredric Meyer
Robert Rosenwasser

Audit Committee:
Christopher Loftus, Chairman
Paul Nelson
Christopher Ogilvy

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Neil Martin

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L. Nelson Hopkins
Richard Morawetz

Scientific Program Committee:
Stephen Papadopoulos, Chairman
Henry Brem
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Round Robin Editor:
Ralph Dacey, Jr.

Local Arrangements:
Richard Morawetz

AANS Joint Sponsorship Education Representative:
James Markert

WFNS Delegates:
Martin Camins - Senior
Volker Sonntag - Alternate

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2006 Annual Meeting of the

American Academy of Neurological Surgery

SCIENTIFIC PROGRAM
AMERICAN ACADEMY
OF NEUROLOGICAL SURGERY

2006 LEARNING OBJECTIVES

Jointly sponsored by The American Association of Neurological Surgeons October 18 – 21, 2006.

Upon completion of this program, the participants should be able to:

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

Discuss the potential applicability of new technologies to the treatment of complex spinal disease.

Discuss some of the implications of resident work hour restrictions and its impact on neurosurgical training.

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

American Association of Neurological Surgeons

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

The American Association of Neurological Surgeons designates this educational activity for a maximum of 14 category 1 credits towards the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

DISCLOSURE INFORMATION

The American Association of Neurological Surgeons 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.

Name	**Conflict of Interest**	**Company**
Berger, M	University Grants/Research Support Honorarium	NIH VSM Medtech Ltd.
Brem, H	University Grants/Research Support	National Cancer Institute, New Approaches to Brain Tumor Therapy, Accelerated Brain Tumor Cure, NIH National Cooperative Drug Discovery Groups, NIH
Chiocca, EA	University Grants/Research Support Consultant Fee	NIH Oxford Biomedica
Heary, R	University Grants Research Support Consultant Fee Stock or Shareholder	DePuy Spine, Inc. DePuy Spine, Inc. Endius

Name	Conflict of Interest	Company
Levy, E	Industry Grant Support	Boston Scientific Corporation, Cordis
	Honorarium	Boston Scientific Corporation, Cordis
Macdonald, R	University Grants/Research Support	NIH
	Industry Grant Support	Boston Scientific
	Consultant Fee	Actelion Pharmeceuticals
	Stock or Shareholder	Brainsgate
Maciunas, R	Industry Grant Support	Medtronic, Inc., BrainLAB AG
Mayberg M	University Grants/Research Support	NIH/National Institute of Neurological Disorders and Stroke
Riina, H	Industry Grant Support	Boston Scientific
	Stock or Shareholder	Neurovasx
	Honorarium	Boston Scientific, Neurovasx, Guidant
Rutka, J	University Grants/Research Support	National Cancer Institute of Canada, Canadian Institutes of Health Research, Pediatric Brain Tumor Foundation
	Consultant Fee	VSM Technologies
Schwartz, T	University Grants/Research Support	National Institute of Neurologic Disorders and Stroke—R01, R21, K08
	Industry Grant Support	NeuroPace, Inc.
	Stock or Stockholder	Neurologix, Inc.
	Honorarium	Marcel Dekker, Thieme Medical Publishers, Ellman
Spetzler, R	Consultant Fee	Anspach, Zeiss
	Stock or Shareholder	Syndergetics
	Other Financial or Material Support	Allegiance

Name	Conflict of Interest	Company
Timmons, S	University Grants/Research Support	NIH, National Institute of Child Health and Human Development
	Industry Grant Support	Novo Nordisk
	Honorarium	Synthes, Contemporary Forums
Winn, HR	University Grants/Research Support	NIH
	Industry Grant Support	Boston Scientific
	Consultant Fee	Actelion Pharmeceuticals
	Stock or Shareholder	Brainsgate

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

Faculty Name

Awad, IA
Abele, JE
Barbaro, NM
Bederson, JB
Delashaw, JB
Dempsey, RJ
Dirks, P
Drake, J
Foley, KT
Friedman, WA
Gerszten, PC
Hadley, MN
Haines, SJ
Harkey, L
Hopkins, LN
Houkin, K

Kim, D
Lang, FF
Lawton, MT
Lewis, S
Malek, AM
McGregor, J
Morgan, MK
Ogden, AT
Ogilvy, CS
Pamir, MN
Park, TS
Pollock, BE
Robinson, S
Rodts, GE
Sawaya, R
Sheldon, T
Toussaint, G
Tymianski, M
Valadka, AB
Warnick, RE

§§§§§

Speakers and their paper presenters/authors who have refused to disclose whether they have relationships with commercial companies:

Faculty Name

None

§§§§§

Speakers who have disclosed that their presentations will include discussion of an off-label or investigational drug or device:

Faculty Name

None

SCIENTIFIC PROGRAM

American Academy of Neurological Surgery Annual Meeting
Ritz-Carlton Lodge at Reynolds Plantation, Georgia
October 18-22, 2006

THURSDAY, OCTOBER 19th Moderator: Henry Brem

PAPER PRESENTATIONS

7:30 – 7:45 AM **Prevention of Cerebral Vasospasm After Aneurysmal Subarachnoid Hemorrhage with Clazosentan, an Endothelin Receptor Antagonist**

Robert L. Macdonald, M.D., A. Kakarieka, M.D., Stephan A. Mayer, M.D., Alberto Pasqualin, D. Ruefenacht, M.D., Peter Schmiedek, M.D., Neal F. Kassell, M.D., Stephen Weidauer, M.D.

7:45 – 8:00 AM **Coding variants in betaglycan and endoglin (TGF-beta co-receptors) in familial cases of intracranial aneurysm**

Dong Kim, M.D.

8:00 – 8:15 AM **Intracranial Aneurysm Surgery: The Hybrid Approach**

Howard A. Riina, M.D.

8:15 – 8:30 AM **Immune Response in Human Cerebral Cavernous Malformations**

Issam A. Awad. M.D., R. Shenkar, C. Shi, I. Check, H.L. Lipton, A. Rowle

8:30 – 8:45 AM

Outcome of Oculomotor Nerve Palsy from Posterior Communicating Artery Aneurysms: Comparison of Clipping and Coiling

Peng Roc Chen, M.D., Sepideh Amin-Hanjani, M.D., Felipe C. Albuquerque, M.D., Cameron McDougall, M.D., Joseph M. Zabramski, M.D., ***Robert F. Spetzler, M.D.***

8:45 – 9:00 AM

Rationale, Technique and Early Results of Staged-Volume Radiosurgery for Patients with Large Cerebral Arteriovenous Malformations

Bruce E. Pollock. M.D., Robert W. Kline, Paul D. Brown, Michael J. Link, Scott L. Stafford

9:00 – 9:15 AM

Tissue Engineering to Treat Intracranial Aneurysms and Use of a Dynamic Ultra-High Resolution Flat-Panel Volumetric CT to Study the Results

Alim P. Mitha, Brian L.Hoh, Rjiv Gupta, ***Christopher S. Ogilvy, M.D.***

9:15 – 9:30 AM

Preliminary Experience with the Wingspan Stent for the Treatment of Intracranial Atheromatous Disease

David Fiorella, M.D., Ph.D., ***Elad I. Levy, M.D***., Aquilla Turk, M.D., Felipe C. Albuquerque, M.D., David Nieman, M.D., Beverly Aagaard-Kienitz, M.D., Henry Woo, M.D., Peter A. Rasmussen, M.D., L. Nelson Hopkins, M.D., Thomas J. Masaryk, M.D., Ricardo A. Hanel, M.D., Ph.D., Cameron G. McDougall, M.D.

9:30 – 9:45 AM	**A Single-Centre, Prospective, Analysis of the Natural History of Hemorrhage from Brain AVM's With or Without Aneurysms** Leodante DaCosta M.D., Karel G. TerBrugge, M.D., Robert A. Willinsky, M.D., Christopher Wallace, M.D., MSc, ***Michael Tymianski, M.D., PhD.***
9:45 – 10:00 AM	**Mechanisms of Acute Brain Injury After Subarachnoid Hemorrhage** ***Joshua B. Bederson, M.D.***, Fatima A. Sehba, Ph.D.
10:00 – 10:30 AM	Beverage Break
10:30 – 10:45 AM	**Utility of Stackable Carbon-Fiber Cages for Thoracolumbar Reconstructions after Corpectomy** ***Robert F. Heary, M.D.***, Sanjeev Kumar, M.D.
10:45 – 11:00 AM	**Comparison of O-Arm vs. C-Arm Fluoroscopy for Intraoperative Spinal Imaging** ***Kevin T. Foley, M.D.***, Paul Park, M.D., Harvey Smith, M.D., Alex Vaccaro, M.D., Milo Solomito, Ph.D., Stephen Papadopoulos, M.D., Hansen Yuan, M.D.
11:00 – 11:15 AM	**Occipitocervical Stabilization Using the Inverted Y Occipital Plate** ***Louis Harkey, M.D.***
11:15 – 11:30 AM	**Blood Loss During Thoracolumbar Stabilization and Fusion Procedures** Thomas A. Moore III, M.D., Charles L. Wolff III, M.D., Bassam A. Hadi, M.D., ***Mark N. Hadley, M.D., F.A.C.S.***

11:30 - 11:45 AM	**Advances in Computer-Assisted Image-Guided Surgery of the Spine: Electromagnetic Technology** ***Gerald E. Rodts, Jr., M.D.***
11:45 - 12:00 PM	**Motor Outcome Analysis Following Surgical Repair Of Birth Brachial Plexus Injury Using Motor Score Composite** William W. Ashley, Jr., M.D., Ph.D., M.B.A., ***Tae Sung Park, M.D.***
12:00 - 12:15 PM	**Rapid Prototyping: A New Approach to Stereotactic Surgical Guidance** ***William A. Friedman, M.D.***, Frank J. Bova
12:15 - 12:30 PM	**Transsphenoidal Surgery Utilizing Intraoperative Magnetic Resonance Imaging: A 320 Case Series** Matthew A. Hunt, M.D., Gregory J. Anderson, Ph.D., ***Johnny B. Delashaw, Jr., M.D***.

FRIDAY, OCTOBER 20th Moderator: Stephen Papadopoulos

7:30 – 7:35 AM	**Introduction of John Able** ***L. Nelson Hopkins, M.D.***
7:35 – 8:00 AM	**The Art of Innovation** ***John E. Abele*** Founder Chairman, Boston Scientific
8:00 – 8:05 AM	**Introduction of Todd Sheldon** ***Stephen Papadopoulos, M.D.***
8:05 – 8:30 AM	**Surgeons working with Industry: What's**

the law?

Todd Sheldon, Esq.
Vice President and Senior Legal Counsel,
Spinal and Biologics Business,
Medtronic, Inc.

8:30 – 8:45 AM Audience Questions

PAPER PRESENTATIONS

8:45 – 9:00 AM **A Critical Role of Monocyte Chemoattractant Protein-1 (MCP-1) in Neuroblast Migration Following Focal Ischemia**

Robert J. Dempsey, M.D., Yiping Yan, Kurt A. Sailor, Bradley T. Lang, Seung-Won Park, Raghu Vemuganti

9:00 – 9:15 AM **Expression of Hypoxia-Inducible Factor-1 and Vascular Endothelial Growth Factor in Response to Venous Hypertension**

Michael T. Lawton, M.D., Rose Du, M.D., Ph.D., Yiqian Zhu, M.D., Guo-Yuan Yang, M.D. Ph.D., William L. Young, M.D.

9:15 – 9:30 AM **Frequency Dependent Hemodymanic Responses to Direct Bipolar Cortical Stimulation**

Challon Perry, Minah Suh, Hongtao Ma, Mingrui Zhao, ***Theodore H. Schwartz, M.D.***

9:30 – 9:45 AM **How does the Participation of the Training Registrar in Surgery for Small Intracranial Aneurysms Impact on Patient Outcome?**

M. K. Morgan, M.D., N. N. Assaad, A. S. Davidson, W. Oldmeadow

9:45 – 10:00 AM	**Alpha II spectrin breakdown products (SBDP): a prototypical biomarker of vasospasm induced cerebral ischemia** ***Stephen Lewis, M.D.***, Greg Verlat, Lynn Miralia, Regins Wolper, Jada Aikman, Jose Pineda, Linda Papa, Kevin Wang, Ron Hayes
10:00 – 10:15 AM	Beverage Break
10:15 – 10:30 AM	**Piece Meal Resection is Associated with a Higher Rate of Leptomeningeal Disease (LMD) in Patients with Posterior Fossa Metastases (PFM)** ***Raymond Sawaya, M.D.***, Dima Suki, Ph.D., Hiba Abouassi, M.D., Akash J., Patel, B.S., Weiming Shi, M.D., Morris D. Groves, M.D., J.D.
10:30 – 10:45 AM	**Extracranial Radiosurgery: An Evolving Treatment Modality for Neurosurgical Applications** ***Peter C. Gerszten, M.D.***
10:45 – 11:00 AM	**Prolonged Survival in Patients with Recurrent Glioblastoma Multiforme Treated by Resection with Implantation of Permanent I-125 Seeds and BCNU Wafers** ***Ronald E. Warnick, M.D.***, Borimir J. Darakchiev, M.D., Robert E. Albright, M.D., John C. Breneman, M.D.
11:00 – 11:15 AM	**Academy Award Honorable Mention** **Galectin-1, a Gene Preferentially Expressed at the Tumor Margin, Promotes Glioblastoma Invasion** ***Toussaint III, Gerard***, Nilson, AE, Goble, JM, Ballman, KV, James, CD, Uhm, JH

11:15 – 11:30 AM **Academy Award Winner**
A2B5 White Matter Progenitors as Glioma Cells of Origin

Ogden, Alfred T, Lochhead, RA, Fusco, D, Lopez, K, Ellis, JA, Kang, J,Waziri,AE, Assanah, M, Canoll, P, Bruce, Jeffrey

11:30 – 11:45 AM **PRESIDENTIAL INTRODUCTION:**
Robert F. Spetzler, M.D.

11:45 – 12:30 PM ***PRESIDENTIAL ADDRESS:***

"Trouble"
L. Nelson Hopkins, M.D.

SATURDAY, OCTOBER 21st Moderator: Robert Rosenwasser

PAPER PRESENTATIONS

7:30 – 7:45 AM **Upregulation of Mitoxantrone Resistance Protein (MXR/ABCG2) in Patients with Medically Refractory Temporal Lobe Epilepsy**

Kitti Kaiboriboon, M.D., Brian K. Alldredge, Pharm. D. , ***Nicholas M. Barbaro, M.D.***, Andrew W. Bollen M.D., Everett J. Austin, M.D., Stephen L. Nutik, M.D., Daniel H. Lowenstein, M.D., Deanna L. Kroetz, Ph. D.

7:45 – 8:00 AM **Identification of Novel Drugs that Target Neural Stem Cells by High Through-Put Screening**

Phedias Diamandis, Michael Tyers, ***Peter Dirks, M.D.***

8:00 – 8:15 AM **Directed Therapy with Polymers for Brain Tumors**

Henry Brem, M.D.

8:15 – 8:30 AM **Insular Gliomas: Surgical Technique, Morbidity Assessment and Outcome Analysis**

Mitchel S. Berger, M.D., Daniel Hirt, B.S., Kathleen R. Lamborn, Ph.D., G. Evren Keles, M.D.

8:30 – 8:45 AM **Human Bone Marrow-Derived Mesenchymal Stem Cells As Delivery Vehicles for Glioma Therapy**

Frederick F.Lang, M.D., Akira Nakamizo, M.D., Frank Marini, Ph.D., Michael Andreeff, M.D., Ph.D.

8:45 – 9:00 AM **Blockade of Glioma Invasion by Inhibitors of GSK-3**

E. Antonio Chiocca, M.D., Ph.D., M. Oskar Nowicki, Ph.D., Jennifer L. Cutter, Ph.D., Sean Lawler, Ph.D.

9:00 – 9:15 AM **Repeat Blood Brain Barrier Disruption in Patients with Recurrent Primary Central Nervous System Lymphoma**

John McGregor, M.D., Eric Bourekas, M.D.

9:15 – 9:30 AM **High-Resolution Patient-Based Computational Fluid Dynamic Analysis of Intracranial Stenosis**

Adel M. Malek, M.D., Ph.D., Clemens M. Schirmer, M.D.

9:30 – 9:45 AM	**DBS for Tourette Syndrome: From Clinical Case to Clinical Trial** ***Robert J. Maciunas, M.D., M.P.H.*, *F.A.C.S.***, Brian N. Maddux, M.D., Ph.D., David E. Riley, M.D., Christina M. Whitney, R.N.C.S., D.N.Sc, Mike R. Schoenberg, Ph.D., Paula J. Ogrocki, Ph.D., Jeffrey M. Albert, Ph.D., Deborah J. Gould, M.D.
9:45 – 10:00 AM	**Temporal Lobe Surgery for Intractable Epilepsy in Children: An Analysis of Outcomes in 126 Children** ***James T. Rutka, M.D.,*** Mony Benifla, M.D., Carter Snead III, M.D., Hiroshi, Otsubo, M.D.
10:00 – 10:15 AM	Beverage Break
10:15 – 10:30 AM	**Multimodality Management of Skull-Base Chordomas: Experience of a Single Institution** ***M. Necmettin Pamir, M.D.***
10:30 – 10:45 AM	**Blood-Brain-Barrier Disruption is Mediated by Flow Arrest and Circulating Leukocytes** Lijana Bengez, Mohammed Hossain, Vince Fazio, Damir Janigro, ***Marc Mayberg, M.D.***
10:45 – 11:00 AM	**Erythropoietin Improves Outcome in Adult Rats After Perinatal Brain Injury** ***Shenandoah Robinson, M.D.***
11:00 – 11:15 AM	**Cerebral Revascularization for Moyamoya Disease** ***Kiyohiro Houkin, M.D.***
11:15 – 11:30 AM	**Has Regulation Improved the CSF Shunt?**

Stephen J. Haines, M.D. **,**Jeffrey P. Blount, M.D.

11:30 – 11:45 AM **Predicting Post Operative Hydrocephalus in Pediatric Post Fossa Tumor Patients**

J. Drake, J. Riva-Cambrin, M. Lamberti-Pasculli, M. Sargent, D. Armstrong, R. Moineddin, D. Cochrane

11:45 – 12:00 PM **Regional Neurotrauma Care in Memphis, TN**

Shelly D. Timmons, M.D., Ph.D., F.A.C.S.

12:00 – 12:15 PM **Magnesium Sulfate for Neuroprotection after Traumatic Brain Injury**

Nancy Temkin, Ph.D., Gail D. Anderson, Ph.D., ***Richard Winn, M.D.,*** Richard Ellenbogen, M.D., james Schuster, M.D., Timothy Lucas, M.D., David W. Newell, M.D., pamela Nelson Mansfield, R. N., B.S.N., Joan E. Machamer, M.A., Jason Barber, M.S. , Sureyya S. Dikemen, Ph.D.

12:15 – 12:30 PM **The Delivery of Emergency Neurosurgical Care**

Alex B. Valadka, M.D., F.A.C.S.

THURSDAY, OCTOBER 19th

7:30 – 7:45 AM **Prevention of Cerebral Vasospasm After Aneurysmal Subarachnoid Hemorrhage with Clazosentan, an Endothelin Receptor Antagonist**

Robert L. Macdonald, M.D., A. Kakarieka, M.D., Stephan A. Mayer, M.D., Alberto Pasqualin, D. Ruefenacht, M.D., Peter Schmiedek, M.D., Neal F. Kassell, M.D., Stephen Weidauer, M.D.

The purpose of this study was to assess the efficacy and safety of 1, 5, and 15 mg/hour intravenous clazosentan, an endothelin receptor antagonist, in preventing vasospasm after aneurysmal subarachnoid hemorrhage (SAH). This phase 2b, international, multicenter, double-blind, placebo-controlled dose-finding study randomized male and female patients aged 18-70 years with SAH and confirmed ruptured saccular aneurysm to placebo or 1 of 3 drug doses beginning within 56 hours of SAH. Treatment was stopped on day 14. Angiographic vasospasm, classified as moderate (>34% arterial narrowing) or severe (>67% narrowing), was measured quantitatively by comparison of digital subtraction angiography at baseline and on day 9±2. Vasospasm-related morbidity and clinical outcome were assessed by comparison of computed tomography done at baseline, 24 to 48 hours after aneurysm treatment and at 6 weeks and clinical assessment at 12 weeks after the SAH, respectively. Infarct size and volumes of intracranial hemorrhages were all measured quantitatively by central radiology review. Safety and tolerability assessments were also conducted. 413 patients were randomized, of which 71% were female and 29% were male. Mean age was 51 years. Coiling or clipping was performed in 54% and 46% of subjects, respectively. The occurrence and severity of vasospasm will be reported per treatment group. Secondary end points that will be reported for each treatment group are cerebral infarcts as determined by follow-up computed tomography scan, vasospasm-related morbidity, and clinical outcome. The incidence of adverse events will also be presented.

THURSDAY, OCTOBER 19

7:45 – 8:00 AM **Coding variants in betaglycan and endoglin (TGF-beta co-receptors) in familial cases of intracranial aneurysm**

Dong Kim, M.D.

Background

Familial aggregation of intracranial aneurysms (IA) strongly suggests a genetic contribution to pathogenesis. However, genetic risk factors have yet to be defined. For families affected by aortic aneurysms, specific gene variants have been defined, many affecting the receptors to TGF-beta (TGFBR 1 and 2). In recent work, we found that aortic and intracranial aneurysms may share a common genetic basis in some families. We hypothesized, therefore, that mutations in TGF-beta receptors may also play a role in IA pathogenesis.

Methods

To identify genetic variants in TGF-beta and its receptors, genomic DNA was prepared from 44 unrelated probands with familial IA syndromes. Direct sequencing of all exons and exon-intron boundaries were performed for 6 genes: TGF-B1, TGFBR1, TGFBR2, ACVR1, betaglycan and endoglin. Positive findings were confirmed by restriction digestion analyses. Novel variants were analyzed for their association with IA by comparing allele frequencies in IA and control populations.

Results

A total of 83 variants including single nucleotide substitutions, deletions and insertions were identified, of which 57 were polymorphisms known to be prevalent in the general population. Of the 26 novel variants, we focused on six that were non-synonymous. Four of these variants were found in population control samples (90-245 control samples tested). Two variants, affecting 3 probands were negative in all control samples. These variants (W112R in betaglycan and A60E in endoglin) produce amino acid substitutions in highly conserved, functionally significant residues.

Conclusions

In this study, we found that 3 of 45 probands (from families affected by IA) had functionally significant coding variants involving betaglycan and endoglin, both co-receptors to TGF-beta. These data suggest that TGF-beta signaling may play a pathogenetic role in a subset of IA families.

8:00 – 8:15 AM **Intracranial Aneurysm Surgery: The Hybrid Approach**

Howard A. Riina, M.D.

Objective: Increasingly, young cerebrovascular surgeons are emerging from neurosurgical training with skill in both open microneurosurgery and endovascular surgery. A consecutive series of patients with intracranial aneurysms in one hybrid surgeon's practice was retrospectively reviewed to demonstrate the ability to achieve good outcomes in both disciplines.
Methods: The primary author (HAR) treated 241 patients (293 aneurysms) between 2001 and 2005 under the supervision of a senior interventional neuroradiologist and cerebrovascular surgeon. Of these, 109 patients (138 aneurysms) were treated with open microneurosurgical techniques and 121 patients (155 aneurysms) were treated minimally invasively using endovascular techniques.
Results: Complete obliteration was attained in 96.2% of clipped aneurysms and 63.9% and 62.7% of coiled aneurysms immediately and after at least 6 month follow-up, respectively. At latest evaluation, 83 (91.2%) endovascular patients and 81 (88%) microneurosurgical patients had good clinical outcomes (GOS 4 or 5; mean follow up 23 months; combines ruptured and unruptured cohorts). Procedure-related mortality included 1 patient in each group.
Conclusions: Residents pursuing careers in cerebrovascular neurosurgery are currently emerging from residency training facile in both microneurosurgery and endovascular surgery. There has been concern, since each discipline is highly complex, that hybrid cerebrovascular surgeons could not practice each technique effectively and achieve good outcomes. With proper supervision by senior faculty, it is possible for hybrid surgeons to obtain the necessary experience and achieve excellent outcomes through proper patient selection for each technique and by adherence to the high technical standards of both microneurosurgery and endovascular surgery.

8:15 - 8:30 AM **Immune Response in Human Cerebral Cavernous Malformations**

Issam A. Awad. M.D., R. Shenkar, C. Shi, I. Check, H.L. Lipton, A. Rowley

Cerebral cavernous malformations (CCMs) affect more than 1 million Americans, predisposing them to a lifetime risk of hemorrhagic stroke and epilepsy. A potential role of the immune response in this disease has not been postulated previously, but would be compelling, given the unique antigenic milieu of CCM lesions with sequestered thrombi and leaky blood-brain barrier, and the numerous examples of immune modulation of angiogenesis in other disease states. It could explain, in part, why some CCM lesions remain biologically dormant, while others proliferate with serious clinical consequences.

We present data from differential gene expression of 5 CCM lesions in comparison to control vascular tissue from 5 arteriovenous malformations and 5 superficial temporal arteries, revealing several immunoglobulin and other related genes markedly upregulated within human CCM lesions. Other results are presented from five CCM lesions, revealing infiltration of antibody producing B lymphocytes and plasma cells within the lesions, including novel 3-dimensional confocal immunomicroscopy illustrating clumps of inflammatory cells. And we present recent data demonstrating 5-fold enrichment of gamma globulin to albumin ratio in human lesion as comparted to serum from the same patient. We describe ongoing research aiming to characterize cellular and humoral components of the immune response in CCMs and initiating investigation into its clonality, by isoelectric focusing of the predominant immunoglobulin isotypes isolated from the lesion, in comparison to the patient's serum, and by the distribustion of lengths of complementary-determining regions 3 (CDR3) of the immunoglobulin heavy chain genes in mRNA isolated from lesions and from pooled plasma cells and B cells laser captured from CCMs, in comparison to peripheral lymphocytes from the blood of the same patients.

Immune response could play a role in or represent a potential marker of CCM lesion proliferation and hemorrhage. The ongoing studies will generate preliminary data for future research aimed at comparing the immune response in quiescent versus clinically aggressive CCM lesions. An oligoclonal immune response shown in this research would stimulate future experiments to identify autoimmune or extrinsic antigenic triggers involved in CCM disease.

THURSDAY, OCTOBER 19

8:30 – 8:45 AM **Outcome of Oculomotor Nerve Palsy from Posterior Communicating Artery Aneurysms: Comparison of Clipping and Coiling**

Peng Roc Chen, M.D., Sepideh Amin-Hanjani, M.D., Felipe C. Albuquerque, M.D., Cameron McDougall, M.D., Joseph M. Zabramski, M.D., ***Robert F. Spetzler, M.D.***

BACKGROUND: Recovery of posterior communicating artery (PCoA) aneurysm-induced oculomotor nerve palsy (ONP) following aneurysm coiling has been reported. However, the coil mass could potentially compromise recovery of the nerve. We therefore compared the outcome of coiling and clipping for this indication.
METHODS: We retrospectively compared the outcome of ONP in 13 patients: 6 underwent endovascular coiling and 7 underwent surgical clipping.
RESULTS: ONP recovered completely in 6 of the 7 surgical patients with ONP compared to 2 of the 6 patients in the endovascular group. Of patients with more than 1-year follow up, all 6 surgical patients recovered completely compared to 2 of 4 endovascular patients ($p = .05$). In addition, preoperative complete or partial ONP was also associated with degree of resolution by survival analysis ($p = .03$). All patients with partial ONP in the surgical group and 2 of 3 patients in the endovascular group recovered without residual deficits while 3 of 4 patients with complete ONP in the clipping group and none in the coiling group recovered completely. Regardless of the treatment modality, time to complete resolution of ONP was 6 months in both groups.
CONCLUSION*:* Clipping PCoA aneurysms was associated with a higher probability of complete recovery of ONP than coiling. Degree of preoperative ONP also affected recovery. If patients can tolerate surgery, it should be considered the treatment of choice.

THURSDAY, OCTOBER 19

8:45 – 9:00 AM **Rationale, Technique and Early Results of Staged-Volume Radiosurgery for Patients with Large Cerebral Arteriovenous Malformations**

Bruce E. Pollock. M.D., Robert W. Kline, Paul D. Brown, Michael J. Link, Scott L. Stafford

Introduction: Radiosurgery is generally not considered an option for patients with large arteriovenous malformations (AVM) due to the risk of radiation-related complications. To limit the radiation exposure to the surrounding normal brain, we performed staged-volume radiosurgery for 21 patients with large AVMs.
Methods: Patients underwent a median of 2 procedures (range, 2-4) performed from 3 to 6 months apart. The median patient age was 36 (range, 10-60). The modified Spetzler-Martin Grades were IIIA (n=5), IIIB (n=1), IV (n=11), and V (n=4). The median radiosurgery-based AVM score was 2.72 (range, 1.64-6.40). The median follow-up after the last procedure was 43 months (range, 11-74).
Results: Obliteration was noted in 6 of 18 patients (33%) with imaging performed three or more years after completion of staged-volume radiosurgery. Explainable causes for non-obliteration (5 patients, 28%) included inadequate AVM coverage ("marginal/geographic miss", n=4) and re-canalization of previously embolized AVM component (n=1). No patient bled between procedures; four patients (19%) sustained eight hemorrhages after staged-volume radiosurgery. Three patients suffered neurologic deficits from the bleeds, and one patient died. The annual hemorrhage rate was 6.3% for the first four years after radiosurgery. No patient developed a radiation-related complication. One patient underwent partial AVM resection 40 months after radiosurgery; 5 patients (28%) underwent repeat radiosurgery at a median of 46 months after radiosurgery.
Conclusions: Staged-volume radiosurgery of large AVMs results is associated with a low risk of radiation-related complications. More follow-up is needed to determine whether this technique provides protection against future bleeding for this difficult patient group.

THURSDAY, OCTOBER 19

9:00 – 9:15 AM **Tissue Engineering to Treat Intracranial Aneurysms and Use of a Dynamic Ultra-High Resolution Flat-Panel Volumetric CT to Study the Results**

Alim P. Mitha, Brian L.Hoh, Rjiv Gupta,
Christopher S. Ogilvy, M.D.

Introduction: Tissue engineering uses the combination of cells, materials, and biochemical factors to improve or replace tissue function. For saccular aneurysms, we have investigated the possibility of using Endothelial Progenitor Cells (EPCs) seeded in a fibrin glue matrix to endovascularly treat aneurysms. We have also utilized new techniques of radiographic visualization of the lesions and their treatments with dynamic ultra-high resolution flat-panel volumetric CT scanning.

Methods: EPCs were isolated from the mononuclear fraction of circulating blood of New Zealand White rabbits and cultured in endothelial basal medium containing 5% FBS and 0.1% VEGF at 37 deg C with 5% CO2. The cells were seeded in fibrin glue at a density of 5 million cells /mL. The constructs were studied in vitro for cell growth and migration. In the same rabbits where cells were harvested, aneurysms were constructed in the neck using a modified elastase model. The aneurysms were then clipped surgically, coiled endovascularly or treated with the tissue engineered construct. The lesions were imaged using flat-panel volumetric CT (fpVCT) and conventional multi-detector CT (MDCT) using intravenous contrast.

Results: It was possible to isolate and grow endothelial cells from EPCs in culture and they continued to grow when seeded in the fibrin glue matrix. Endothelial cell confirmation was perfomed using CD31 and Acetylated Low Density Lipoprotein (DiI-Ac-LDL). In a separate set of experiments, it was possible to inject an endothelial cell-seeded matrix into the aneurysm model and achieve endothelial growth at the neck. FpVCT proved to be an excellent technique in showing details of the experimental aneurysms and their surgical and endovascular treatments.

Conclusions: It appears possible to use tissue engineering as a method to treat aneurysms in an animal model; however, the durability of this method has not yet been proven. Further work is planned using different matrix constructs. FpVCT imaging appears to be superior to previous CTA techniques in the evaluation of aneurysm morphology and completeness of treatment.

THURSDAY, OCTOBER 19

9:15 - 9:30 AM **Preliminary Experience with the Wingspan Stent for the Treatment of Intracranial Atheromatous Disease**

David Fiorella, M.D., Ph.D., ***Elad I. Levy, M.D***., Aquilla Turk, M.D., Felipe C. Albuquerque, M.D., David Nieman, M.D., Beverly Aagaard-Kienitz, M.D., Henry Woo, M.D., Peter A. Rasmussen, M.D., L. Nelson Hopkins, M.D., Thomas J. Masaryk, M.D., Ricardo A. Hanel, M.D., Ph.D., Cameron G. McDougall, M.D.

Introduction: The Wingspan (Boston Scientific) is the first self-expanding stent system designed specifically for the treatment of intracranial atheromatous disease. We report the immediate and periprocedural results of our initial experience with the device in a series of 48 patients.

Methods: All patients undergoing angioplasty and stenting with the Gateway balloon–Wingspan stent system were prospectively tracked in our collaborative four-institution endovascular database. Patient data, lesion characteristics, procedural details, and clinical and imaging follow up were recorded.

Results: During a 5-month period, treatment with the stent system was attempted in 48 patients (average age 62.9 years; 16 women) with 51 intracranial atheromatous lesions, of which 36 were more than 70% stenotic. Fifty of 51 lesions were successfully treated (98% technical success). In one case, the stent could not be delivered across the lesion; and the patient was treated solely with angioplasty. Lesions treated involved the internal carotid (n=22; 8 petrous, 6 cavernous, 6 supraclinoid segment, 2 terminus), vertebral (n=9; V4 segment), basilar (n=11), and middle cerebral (n=9) arteries. Average pretreatment stenosis was 74.8%, improving to 46.1% after balloon angioplasty and to 29.8% after stent placement. Of the 51 lesions treated, there was one periprocedural neurological complication with a pontine stroke and subsequent death (2%); otherwise, there were no permanent neurological complications. One additional patient experienced a transient neurological deficit after the procedure with a visual field deficit that completely resolved within 36 hours. None of

the 43 patients for whom 30-day clinical follow-up is available have experienced a major adverse neurological event.

Conclusions: Angioplasty and stenting for symptomatic or flow-restrictive intracranial atheromatous disease can be performed with the Gateway balloon-Wingspan stent system with a high rate of technical success and low periprocedural morbidity. Our initial experience indicates that this procedure represents a viable treatment option for this patient population.

THURSDAY, OCTOBER 19

9:30 - 9:45 AM **A Single-Centre, Prospective, Analysis of the Natural History of Hemorrhage from Brain AVM's With or Without Aneurysms**

Leodante DaCosta M.D., Karel G. TerBrugge, M.D., Robert A. Willinsky, M.D., Christopher Wallace, M.D., MSc, ***Michael Tymianski, M.D., PhD.***

Introduction: Brain Arteriovenous malformations (bAVMs) are heterogeneous vascular malformations in which a serious consequence to patients is a brain hemorrhage. However, factors predictive of hemorrhage are poorly understood, and may include not only the presence of an AVM nidus, but also other features that may be associated with an altered likelihood of future hemorrhage.

Methods: 678 bAVM patients were prospectively followed up (f/u) for a total of 1931.7 patient-years (mean f/u = 2.85yrs, max 17.43yrs). f/u terminated at the time of latest visit, or at the time of AVM cure. Multiple logistic regression analysis and survival analyses were used to determine the significance of the following factors in contributing to an interval hemorrhage during f/u: Hemorrhagic presentation, Seizures, Associated Aneurysms (prenidal, intranidal or remote), AVM size, Deep AVM location and deep venous drainage.

Results: The hemorrhage rates during F/U were: For the entire cohort (n= 678), 4.61%/yr; Initial hemorrhagic presentation (n=258), 7.48%/yr; Initial seizure presentation (n=260), 4.16%/yr; Patients not harboring aneurysms (n=556), 3.99%/yr; Patients with Associated aneurysms (n=122), 6.93%/yr; AVM size <3cm (n=418), 4.55%/yr; AVM size >3cm (n=260), 4.66%/yr; Deeply situated AVM (n=85), 5.2%/yr; Deep venous drainage (n=365), 5.42%/yr. Multiple logistic regression analysis revealed the following to be significant independent predictors of hemorrhage in f/u: Hemorrhagic presentation (Odds Ratio = 2.1), Associated aneurysms (Odds Ratio = 1.95) and deep venous drainage (Odds Ratio = 1.80). Seizures, AVM size and deep location were not associated with a significantly increased future hemorrhage risk.

Discussion: Our results indicate that patients who have presented with an initial hemorrhage, or who have associated aneurysms, or whose AVMs exhibit deep venous drainage, have an approximately twofold higher likelihood of suffering additional bleeds in follow up as compared with other AVM patients. Awareness of these risk factors may be helpful in guiding the choice of treatment of patients with bAVMs.

THURSDAY, OCTOBER 19

9:45 - 10:00 AM **Mechanisms of Acute Brain Injury After Subarachnoid Hemorrhage**

Joshua B. Bederson, M.D., Fatima A. Sehba, Ph.D.

The major cause of death and disability after subarachnoid hemorrhage is acute cerebral ischemia occurring at or shortly after the initial bleed, but the mechanisms of this injury are incompletely defined and few treatments exist. This presentation outlines our investigations of these mechanisms with a focus on alterations of the Nitric Oxide/Nitric Oxide Synthase (NO/NOS) pathway.

Using the rat endovascular filament model of SAH developed in this laboratory we have demonstrated acute (within minutes) constriction of large and small cerebral vessels, acute intraluminal platelet aggregation, degradation of basal lamina, and blood brain barrier dysfunction and perfusion deficits immediately after SAH. We have also shown that 1) cerebral NO levels decrease within 10 minutes after SAH and recover 3 hours later, 2) administration of an NO donor 10 minutes after SAH attenuates acute vasoconstriction and CBF reductions and limits degradation of basal lamina and blood brain barrier dysfunction and 3) NOS-3 (endothelial NOS) decreases in large and small cerebral vessels 10-60 minutes after SAH. Acute loss of vascular NOS-3 has not been demonstrated in other forms of cerebral ischemia and may contribute to SAH-induced ischemia by promoting vasoconstriction, intraluminal platelet aggregation, and direct microvessel injury.

Micovascular structure and permeability are altered immediately after SAH in brain, contributing to the acute ischemic injury seen in this condition. This condition appears to be amenable to pharmacological treatment strategies, which will be reviewed.

THURSDAY, OCTOBER 19

10:30 – 10:45 AM **Utility of Stackable Carbon-Fiber Cages for Thoracolumbar Reconstructions after Corpectomy**

Robert F. Heary, M.D., Sanjeev Kumar, M.D.

Introduction: Stackable carbon-fiber cages offer the following advantages for reconstructing the thoracolumbar spine: similar modulus of elasticity to bone (unlike titanium), immediate stability, modularity, and radiolucency.

Materials and methods: Over five years, 37 consecutive patients underwent thoracolumbar reconstruction/fusion, after corpectomy, using stackable carbon-fiber cages. There were 25 males (68%) and 12 females (32%) with mean age 39 years (range 14-79). Surgical indications were trauma-27, degenerative-5, and infectious-5. Tumors were excluded. Eleven patients (30%) were neurologically intact and 26(70%) had deficits. Levels were thoracic-12(32%), thoracolumbar-19(51%), and lumbar-6(16%). All patients had instrumented anterior stabilization constructs and 10 patients (27%) had posterior supplemental instrumentation. Radiographs and clinical evaluations were performed at regular intervals postoperatively. Autologous bone filled all cages. All patients had postoperative CT scans. The width of decompression and sagittal plane balance were assessed. Fusions required intact instrumentation, bridging trabecular bone, and no motion on flexion-extension films.

Results: The mean follow-up was 26 months. One patient died (unrelated to surgery) and one was lost to follow-up. The mean preoperative Cobb angle of 16º corrected to 6º. The mean decompression width was 20 mm. No neurological deteriorations occurred and solid fusions were proven in 31 patients (84%). Four patients (11%) had early signs of fusion. No instrumentation related complications and no late construct failures occurred.

Conclusion: Stackable carbon fiber cages offer modularity, radiolucency, immediate stability, and a favorable modulus of elasticity. This is the first large series to demonstrate the long-term outcomes using this technique. These results demonstrate an excellent option when reconstruction is needed, following corpectomy, in the thoracolumbar spine.

THURSDAY, OCTOBER 19

10:45 – 11:00 AM **Comparison of O-Arm vs. C-Arm Fluoroscopy for Intraoperative Spinal Imaging**

Kevin T. Foley, M.D., Paul Park, M.D., Harvey Smith, M.D., Alex Vaccaro, M.D., Milo Solomito, Ph.D., Stephen Papadopoulos, M.D., Hansen Yuan, M.D.

Introduction

O-arm fluoroscopy is a new, robotically-controlled intraoperative imaging technology, capable of both 2- and 3-dimensional spinal imaging. As this technology utilizes flat-plate detectors and can routinely position the x-ray source opposite the surgeon, it should be capable of reducing radiation exposure to the operator. The purpose of this study was to compare C-arm and O-arm fluoroscopy in terms of radiation exposure and image acquisition time.

Methods

Percutaneous screw-rod constructs were placed at L1-2, L3-4, and L5-S1 in two fresh cadavers. Imaging was alternated between C-arm and O-arm fluoroscopy. Outcome measures included image acquisition time and radiation exposure to the eye, thyroid, torso, and index finger. Post-procedure 3-dimensional images were obtained to assess screw placement accuracy.

Results

Image acquisition times for the C-arm were 1268 seconds at L1-2, 1030 seconds at L3-4, and 832 seconds at L5-S1. Image acquisition times for the O-arm were 372 seconds at L1-2, 500 seconds at L3-4, and 447 seconds at L5-S1. Mean radiation exposure to the eye was 14 mrem (C-arm) and 5 mrem (O-arm). Mean thyroid exposure was 19 mrem (C-arm) and 10 mrem (O-arm). Mean torso exposure was 34 mrem (C-arm) and 17 mrem (O-arm). Mean finger exposure was 15 mrem (C-arm) and 1.7 mrem (O-arm). There were no significant differences between the C-arm and O-arm in screw placement accuracy.

Conclusions

Although both techniques resulted in accurate screw placement, image acquisition with the O-arm was consistently faster than with the C-

arm. This was likely due to the robotic controls integrated into the O-arm imaging unit. Additionally, radiation exposure was less with the O-arm unit than with the C-arm. The O-arm allows the x-ray source to be easily positioned opposite the surgeon (thus reducing Compton scatter) and has a lower dose per "unit on time."

THURSDAY, OCTOBER 19

11:00 - 11:15 AM **Occipitocervical Stabilization Using the Inverted Y Occipital Plate**

Louis Harkey, M.D.

Introduction: Stabilization of the occipitocervical junction can be a challenging surgical task. The majority of surgical implants used in this region are not specifically designed for this indication. Since 2001, the Inverted Y occipital plate (Depuy Spine) has been commercially available for use in the United States. It is specifically indicated for fixation to the midline keel of the occipital bone. As yet, no clinical series has been published establishing the efficacy of this device.

Methods: All cases of occipitocervical stabilization using the inverted Y occipital plate (Depuy Spine) at the University of Mississippi were retrospectively reviewed for indication, technique, clinical outcome and longterm stability.

Results: 20 patients underwent occipitocervical stabilization over the 5 year period that the inverted Y plate has been available. Average follow up for the series was 21.5 months. There has been one case of screw loosening but all patients have stable unions. One patient expired three days after surgery and several complications were observed.

Conclusion: The inverted Y occipital stabilization plate is an effective, easily applied implant for stabilization of the occipitocervical junction.

THURSDAY, OCTOBER 19

11:15 - 11:30 AM **Blood Loss During Thoracolumbar Stabilization and Fusion Procedures**

Thomas A. Moore III, M.D., Charles L. Wolff III, M.D., Bassam A. Hadi, M.D., ***Mark N. Hadley, M.D., F.A.C.S.***

The use of autologous blood transfusion, blood salvage techniques and relaxation of the traditional "safe limits" of hemodilution have led to a decrease in the rate of homologous blood transfusion in surgery patients. Thoracolumbar internal fixation and fusion procedures are often associated with significant blood loss. The purpose of this study was to identify patients, procedures, and etiologies associated with significant blood loss in order to predict peri-operative resuscitation needs.

METHODS

Between January 1995 and January 2001, 290 patients underwent thoracolumbar stabilization procedures and were prospectively assessed. Etiology was degenerative disease (201), trauma (82), osteomyelitis (22), tumor (14), renal osteodystrophy (4), and inflammatory disease (2). Hematocrits were obtained preoperatively, after positioning and hydration, in the recovery room, and on the first two postoperative days. Intraoperatively patients were transfused for hematocrit less than 25 with ongoing blood loss. Patients with post-operative hematocrits of 22 or less were transfused. Patients with post-operative hematocrits between 23 and 30 were transfused only if symptomatic.

RESULTS

Increased rates of transfusion were identified for patients with tumor (86%), trauma (67%), osteomyelitis (68%) and renal osteodystrophy (50%). Anterolateral operations required transfusion most often (75%), particularly when combined with a dorsal procedure (86%). Dorsal only stabilization/fusion incorporating four or more levels had a transfusion rate of 60%. Patients requiring dorsal only fixation/fusion for degenerative disease involving three levels or less had an 18% transfusion rate. One hundred ninety-two patients had a low hematocrit of 30 or less; 61 had a hematocrit of 25 or lower. Forty percent of patients received blood transfusions in the perioperative

period. Complications included CVA (1), Myocardial Infarction (1), Cardiac Ischemia (1), and Death (1). Only one patient required a post-discharge transfusion within one month following surgery.

CONCLUSIONS

1. Isovolemic hemodilution is a safe alternative to blood transfusion.
2. Intraoperative blood salvage is encouraged for trauma and degenerative disease involving four or more levels.

THURSDAY, OCTOBER 19

11:30 - 11:45 AM **Advances in Computer-Assisted Image-Guided Surgery of the Spine: Electromagnetic Technology**

Gerald E. Rodts, Jr., M.D.

Three-dimensional computer-assisted systems have enabled surgeons to place spinal instrumentation with greater accuracy and confidence. Three dimensional systems provide axial, sagittal and coronal views as well as a 3-D model of the spine. Two-dimensional, fluoro-based systems have reduced surgeon exposure to radiation and all virtual navigation of the patient's anatomy. Until recently, most systems relied upon infra-red, line-of-site technology. Instruments, surgeons' hands, drapes, Mayo stands, etc. often interfere with proper transmission of the signal. At our institution, we have experience using a fluoro-based, 2-D guidance system that employs electromagnetic technology. The line-of-site limitations are eliminated and spinal or bodily reference points can be established deeper in body cavities without the need to be "seen" by a receiver. This system is analogous to technology used for Global Positioning Systems (GPS). The essential details of how the technology works, inherent limitations and pitfalls, and our overall clinical experience in lumbar fusion surgery will be
presented.

THURSDAY, OCTOBER 19

11:45 - 12:00 PM **Motor Outcome Analysis Following Surgical Repair Of Birth Brachial Plexus Injury Using Motor Score Composite**

William W. Ashley, Jr., M.D., Ph.D., M.B.A., ***Tae Sung Park, M.D.***

Due to the complex and variable nature of brachial plexus injury, outcomes analysis can be cumbersome and imprecise. Many scales have been devised, but no single scale is used uniformly. Moreover, despite several studies that report brachial plexus surgical data, no highly predictive clinical model has been defined. With this in mind, we conducted a retrospective analysis of 114 consecutive brachial plexus surgeries performed over the past 14 years at SLCH. 63 were included in this study.

We defined the motor score composite (MSC) and used this novel metric to perform a detailed analysis of our surgical outcomes. The mean MSC was 0.50 pre-op, 0.71 at one year post-op and 0.80 at two years post-op. 89% of the patients made a good or excellent recovery by two years post-op. Age at surgery, time to visit, location, and severity were predictive of outcome. Using MSC data, we developed a prognostic model that enabled accurate prediction of surgical outcomes using pre-operative variables. The MSC model predicts surgical outcomes in our patients with 88% accuracy

The MSC is an efficient metric for the reporting of brachial plexus outcome data. It provides information about extent and severity of injury in a single proportion and facilitates complex data analysis. We confirmed that surgery is beneficial in a select group of patients with most improvement occurring in the first two years after surgery. Our data also suggests that age at surgery, time to visit, location, and severity are key predictive variables. Finally, we used the MSC model to accurately predict surgical outcome. This could have wide applicability for the prediction of post-operative recovery and could facilitate surgical decision-making and improve family counseling.

THURSDAY, OCTOBER 19

12:00 - 12:15 PM **Rapid Prototyping: A New Approach to Stereotactic Surgical Guidance**

William A. Friedman, M.D., Frank J. Bova

Introduction: Frame based stereotactic systems have been used for years in neurosurgery to perform functional procedures and brain lesion biopsies. More recently, frameless systems have become popular as a means to guide cranial and spine surgery. In general, they are believed to make such procedures more minimally invasive, effective, and safe, by increasing the certainty of localization of normal and abnormal imaged anatomical structures. Such systems, however, often require special preoperative scans, infrared cameras, and intraoperative computer systems. This equipment can pose problems regarding the placement of other equipment during surgery (i.e. microscope, fluoroscope). Their use requires special training and additional personnel and can add substantial time to the procedure. These issues may curtail the use of such systems where stereotactic guidance would almost certainly be of value (i.e. ventricular shunt placement, pedicle screw placement). These drawbacks of current image guidance systems led us to consider a mechanical alternative.
Method and Materials: Recently, a new generation of 3-dimensional printers (rapid prototyping machine) has been developed. These systems are capable of fabricating OR compatible objects within an hour of design. The goal of this project was to develop from a diagnostic image dataset and to then plan a surgical procedure. Utilizing the 3D patient specific model, the software is then used to design a patient-specific stereotactic guide and fabricates this guide using rapid prototyping technology. Basically, an inexpensive, one-time use, disposable stereotactic guide can be manufactured from any preoperative MRI or CT scan. The Device is applied to the patient's head or spine during surgery. It can be used to guide skin incision, bone removal, catheter or biopsy probe insertion, pedicle screw placement, etc.
Results: The necessary computer interface has been developed and tested. Rapid prototyping stereotactic guides have been developed in two initial areas: pedicle screw placement and ventricular catheter placement. Under IRB approved protocols, this new technology is now being tested in the operating room.

Conclusions: A new approach to stereotactic guidance, requiring no special equipment in the operating room, has been developed at the University of Florida. A detailed description of this technology as well as preliminary results of surgical testing will be presented at the Academy meeting.

THURSDAY, OCTOBER 19

12:15 - 12:30 PM **Transsphenoidal Surgery Utilizing Intraoperative Magnetic Resonance Imaging: A 320 Case Series**

Matthew A. Hunt, M.D., Gregory J. Anderson, Ph.D., ***Johnny B. Delashaw, Jr., M.D***.

Introduction: Intraoperative magnetic resonance imaging (IMRI) technology allows both neuronavigation and confirmation of tumor resection. Transsphenoidal surgery utilizing IMRI has previously been reported as useful in confirming decompression of the optic chiasm and completeness of tumor resection. At our institution IMRI has been used for all patients undergoing transsphenoidal surgery over the last four years and we describe a case series.

Methods: A retrospective chart review of patients who underwent transsphenoidal surgery (320 surgeries) utilizing IMRI was undertaken. Data collected on the utility of IMRI included: abort frequency, rescan frequency, reexploration frequency, demographics (age, sex, diagnosis) and operative time.

Results: Intraoperative MRI was successfully used in 290 of 320 cases (10% abort frequency). Diagnosis affected success, with Cushing's disease patients having a higher abort frequency (13%). Installation and use of a second generation IMRI unit significantly improved the proportion of patients successfully imaged. Over the case series, IMRI use involved a significant learning curve. Patients with hormonally active tumors were less likely to undergo repeat imaging than patients with non-functional pituitary tumors or Rathke's cleft cysts. In patients who did undergo repeat imaging, IMRI provided information that affected operative decision making in 22% of patients. Operative time decreased following the installation and use of the second generation IMRI unit.

Conclusions: Intraoperative MRI can be routinely and successfully utilized for transsphenoidal surgery, regardless of the patient's diagnosis. Intraoperative MRI provides valuable information that may modify the surgeon's decision making process with the ultimate goal of improved patient outcome.

FRIDAY, OCTOBER 20th

8:45 – 9:00 AM **A Critical Role of Monocyte Chemoattractant Protein-1 (MCP-1) in Neuroblast Migration Following Focal Ischemia**

Robert J. Dempsey, M.D., Yiping Yan, Kurt A. Sailor, Bradley T. Lang, Seung-Won Park, Raghu Vemuganti

Neurogenesis following cerebral ischemia has brought hope that this endogenous mechanism of cell replacement might be enhanced to repair the damaged brain. After focal ischemia, the neuroblasts formed in the subventricular zone (SVZ) migrate towards the ischemic area, but the mechanisms underlying this process are not understood. We hypothesize that chemokines such as MCP-1 formed in excess attracts the neuroblasts to migrate towards the ischemic region. We presently report that doublecortin (DCX) positive neuroblasts migrated from the ipsilateral SVZ towards the penumbra of the ischemic striatum and cortex between days 2 and 6 of reperfusion following transient middle cerebral artery occlusion in adult rats. The DCX positive neuroblasts also migrated from the posterior peri-ventricle (PPV) to the ischemic cortex. Immunostaining showed that the expression of MCP-1 is localized in the activated microglia and astrocytes in the ischemic area between days 1 and 3 of reperfusion. Cultured neural progenitor cells when grafted into the non-ischemic ipsilateral cortex migrated towards the infarcted region. Furthermore, infusion of MCP-1 into the striatum induced neural progenitor migration to the site of MCP-1 infusion. The migrating neural progenitors expressed the MCP-1 receptor CCR2. In knockout mice that lacked either MCP-1 or CCR2, there was a significantly decrease in the number of migrating neuroblasts from the ipsilateral SVZ to the ischemic striatum. These results show that MCP-1 is one of the factors that attract the migration of newly formed neurons from the neurogenic region to the damaged region following focal ischemia. The project was supported by NIH RO1 grants NS045143, NS044173 and NS049448.

Keywords: neural progenitors, migration, chemokines, adult neurogenesis; stroke**;** subventricular zone.

FRIDAY, OCTOBER 20th

9:00 – 9:15 AM **Expression of Hypoxia-Inducible Factor-1 and Vascular Endothelial Growth Factor in Response to Venous Hypertension**

Michael T. Lawton, M.D., Rose Du, M.D., Ph.D., Yiqian Zhu, M.D., Guo-Yuan Yang, M.D. Ph.D., William L. Young, M.D.

OBJECTIVE: Experimentally, a fistula created surgically between the carotid artery and jugular vein, together with occlusion of venous sinuses, generate venous hypertension, which can induce dural arteriovenous fistula (DAVF) formation intracranially in rats. Our aim was to study the effect of non-ischemic venous hypertension on the elaboration of the angiogenic signal, hypoxia-inducible factor-1 (HIF-1) and its downstream signal, vascular endothelial growth factor (VEGF).

METHODS: Sixty rats were exposed to venous hypertension for times ranging from 4 hours to 3 weeks. Western blot analysis, transbinding assays, enzyme-linked immunosorbant assays (ELISA), and immunohistochemistry quantified HIF-1 and VEGF expression in brain. Forty-eight control rats underwent similar surgical procedures without creating venous hypertension. Cerebral blood flow (CBF) was measured at baseline, after surgery, and before sacrifice.

RESULTS: Venous hypertension did not impair CBF. Relative to controls, HIF-1 expression increased five-fold in response to venous hypertension ($p<0.005$), with peak expression one day later, localized to endothelial cells in venules next to the sagittal sinus. VEGF expression also increased three-fold in response to venous hypertension ($p<0.05$), with peak expression 7 days later, localized to parasagittal astrocytes. HIF-1 and VEGF were minimally expressed in rats normal venous pressures.

CONCLUSIONS: In this model, venous hypertension stimulates angiogenesis by a mechanism other than ischemia. HIF-1 expression may result from dilation of parasagittal veins and endothelial deformation. HIF-1 and VEGF appear to be molecular agents that convert venous hypertension into intracellular signals and angiogenesis activity.

FRIDAY, OCTOBER 20th

9:15 – 9:30 AM **Frequency Dependent Hemodymanic Responses to Direct Bipolar Cortical Stimulation**

Challon Perry, Minah Suh, Hongtao Ma, Mingrui Zhao, ***Theodore H. Schwartz, M.D.***

Cortical stimulation is playing an increasing role in clinical neuroscience in the treatment of epilepsy, stroke and pain. The neuromodulatory effects of cortical stimulation include both excitation and inhibition. As a result, cortical stimulation also influences cerebral hemodynamics, including perfusion and oxygenation, which may vary depending on the stimulation parameters.

Using bipolar tungsten electrodes, the rat sensory neocortex was stimulated at variable frequencies in a pseudo-random fashion at 5 Hz, 10 Hz, 50 Hz, 100 Hz, and 250 Hz at a constant charge (CC). The amplitude remained constant at 1 mA. CC was maintained by delivering stimulation at 100 spikes at each frequency and varying the duration of stimulation. Simultaneous field potential (f.p.) recording and ORIS was performed at 570 nm and 610 nm to quantitatively measure total hemoglobin (Hbt) and deoxygenated hemoglobin (Hbr).

A large increase in Hbr, consistent with a dip in oxygenation, was recorded at all stimulation frequencies but the maximum occurred at 10 Hz (2.12±0.20%, n=3). There was a significant correlation between the duration of stimulation and the duration of the increase in Hbr ($r=.86, p<0.05$). The largest increase in Hbt occurred at 50 Hz (4.04±1.08 %, n=3) with no significant relationship with duration of stimulation. The duration of the increase in Hbt was generally longer than the duration of stimulation by 80 seconds, regardless of frequency. These results demonstrate that the duration of stimulation is more important than the frequency in determining the duration of the dip in oxygen. In order to achieve a maximal increase in blood flow with minimal ischemia, one should use short duration, high frequency stimulation.

FRIDAY, OCTOBER 20th

9:30 - 9:45 AM **How does the Participation of the Training Registrar in Surgery for Small Intracranial Aneurysms Impact on Patient Outcome?**

M. K. Morgan, M.D., N. N. Assaad, A. S. Davidson, W. Oldmeadow

From a prospectively collected data-base aneurysms no greater than 10 mm, of the internal carotid artery (beyond the paraclinoid segment) and middle cerebral artery were selected for analysis between 1991 and 2006. The presence or absence of an advanced training registrar was noted. Advanced training registrars were present for every case in the first three years. Comparison were made:
1 In quintiles to establish the stability and incidence of adverse outcomes over time.
2 Between the first quintile and the remainder. This was to establish whether a learning curve existed. The first three years were selected as this was in a period that no surgery was performed in the absence of a registrar.
3 Between cases where an advanced training registrar was present or absent. This was to establish whether trainees increased the incidence of permanent adverse outcomes.
Results: 355 cases (196 with registrar and 159 without a registrar) were evaluated. The overall incidence of permanent adverse outcomes was seen to occur in 11 (3.1% of total) cases, all due to branch artery occlusion. The incidence of permanent adverse outcome in the first three years was 10.7% and 2.4% thereafter (p=0.015). There was no difference in incidence of adverse outcome demonstrated comparing surgery with and without the presence of an advanced training registrar.
This study supports the existence of a learning curve for the senior author in these surgeries. However, no increased incidence in adverse outcome was demonstrated with the participation of the advanced training registrar. The resolution of this paradox lies in the synergy between the neurosurgical supervisor and trainee. This study suggests that patients can be informed that the team approach to their surgery is at least as good as having the experienced surgeon performing all aspects of the surgery.

FRIDAY, OCTOBER 20th

9:45 - 10:00 AM **Alpha II spectrin breakdown products (SBDP): a prototypical biomarker of vasospasm induced cerebral ischemia**

Stephen Lewis, M.D., Greg Verlat, Lynn Miralia, Regins Wolper, Jada Aikman, Jose Pineda, Linda Papa, Kevin Wang, Ron Hayes

A strategy to identify biological markers of cellular injury in cerebrospinal fluid or serum may provide real time information, warning of impending ischemia from cerebral vasospasm and thus provide an earlier window of opportunity for therapeutic intervention.

This study aimed to assess the diagnostic and prognostic utility of alpha-II spectrin breakdown product (ASBDP) biomarkers in patients with aneurysmal subarachnoid hemorrhage (ASAH) by examining the relationship between levels of ASBDP's, injury severity (WFNS score) and onset of cerebral vasospasm.

Patients suffering ASAH requiring ventriculostomy were included in this study. CSF samples were collected every 6 hours until day 12 post hemorrhage. ASBP's were identified using SDS-PAGE technique and immunohybridization. Demographics, clinical grade, Fisher grade, onset of vasospasm, and extended Glasgow outcome scores were prospectively collected. Vasospasm was defined as a change in clinical status and was confirmed radiologically.

20 patients with ASAH, 5 males and 15 females were included in the study. Mean age was 54 years. Cerebral vasospasm developed in 13 of 20 patients. A significant 3 to 5 fold increase in densitometric levels was observed in initial CSF samples from poor grade patients (WFNS grade 4 to 6) compared to good grade patients (WFNS grade 1 to 3) ($p<0.001$) . ASBDP levels were shown increase significantly above baseline at least 12 hours prior to the clinical diagnosis of vasospasm ($p<0.05$).

ASBDP represent a non subjective putative biomarker capable of early detection of ASAH-induced vasospasm. This may provide an earlier opportunity for therapeutic intervention and improved clinical outcome.

FRIDAY, OCTOBER 20th

10:15 - 10:30 AM **Piece Meal Resection is Associated with a Higher Rate of Leptomeningeal Disease (LMD) in Patients with Posterior Fossa Metastases (PFM)**

Raymond Sawaya, M.D., Dima Suki, Ph.D., Hiba Abouassi, M.D., Akash J., Patel, B.S., Weiming Shi, M.D., Morris D. Groves, M.D., J.D.

STUDY DESIGN: Retrospective chart review

INTRODUCTION: Resection of PFM has been cited as a risk factor for developing LMD. We test the hypothesis that patients with PFM treated surgically are at a higher risk of LMD compared to those treated with stereotactic radiosurgery (SRS).

METHODS: Patients with PFM treated with resection or SRS between 1990 and 2005 were identified. Patients were excluded if they received prior resection or SRS for a PFM; had a prior history of LMD; or were not followed at the center after the index procedure. 379 eligible patients are included in this report. The primary outcome was the incidence of LMD, diagnosed by cytologic cerebrospinal fluid analysis or neuroimaging.

RESULTS: 260 patients underwent resection; 119 received SRS. Median age was 56 years; 51% were males; 93% had a Karnosfsky score over 70. Common primary cancer sites were lung (35%), breast (19%), melanoma (14%), and renal (12%). 33 patients (9%) developed LMD, 10% following resection and 6% following SRS [Rate ratio (RR) for resection = 2.06; 95% confidence interval (CI), 0.89-4.75; p=0.09]. Stratification of surgical methodology by en bloc (EB) (N=123) versus piece meal (PM) (N=137) showed a significantly higher risk of the latter approach, both compared to EB [RR (PM) = 3.40; 95% CI, 1.43-8.12; p=0.006] and SRS [RR (PM) = 3.37; 95% CI, 1.41-8.04; p=0.006], with no significant difference between EB and SRS [RR (EB) =0.98; p=0.98; simple incidence estimates of LMD were 14%, 6%, and 6%, respectively]. In the multivariate Cox model, the ratio and significance for PM were consistent with a strong effect (RR=2.51; 95% CI, 1.24-5.11; p=0.02).

CONCLUSION: Piece meal resection of a PFM is associated with an increased rate of LMD. This difference, although clinically and statistically significant, is not as alarming as previously published and is not applicable when en bloc resection is achieved.

FRIDAY, OCTOBER 20th

10:30 – 10:45 AM **Extracranial Radiosurgery: An Evolving Treatment Modality for Neurosurgical Applications**

Peter C. Gerszten, M.D.

The role of radiosurgery for the treatment of intracranial benign and malignant tumors, vascular malformations, and functional therapies is well established and is an integral specialty within the field of neurological surgery. With the advent of extracranial radiosurgery technologies, radiosurgery for the treatment of extracranial neurosurgical disease processes is rapidly evolving.

Since 2001, our center has explored the role of frameless radiosurgery for the treatment of extracranial neurosurgical diseases including benign tumors, primary tumors, metastatic tumors, and vascular malformations. Site locations have included intramedullary, intradural extramedullary, extradural, spinal and paraspinal, brachial plexus, and peripheral nerve. Over 700 patients have been treated and evaluated in a prospective manner representing the largest single center experience in the world. The primary indications for radiosurgery treatment were pain, as a definitive therapy, radiographic tumor progression, post-surgical treatment for residual disease, progressive neurological deficit, and as a boost for bulky or radioresistant tumors.

Radiosurgery now has become an integral part of the treatment paradigm for patients with extracranial neurosurgical diseases treated at our institution. Radiosurgery is well tolerated, safe, and clinically effective. Extracranial radiosurgery is used as an alternative therapeutic modality for lesions not amenable to open surgical techniques, in medically inoperable patients, for lesions located in previously irradiated sites, or as an adjunct to surgery.

The role of radiosurgery for the treatment of extracranial neurosurgical applications is rapidly expanding with the rapid acquisition of this technology by neurosurgeons. Careful attention towards sound clinical and basic science research, standardized practices, resident teaching, credentialing, and technology assessment will become ever more important to our specialty in the future.

FRIDAY, OCTOBER 20th

10:45 - 11:00 AM **Prolonged Survival in Patients with Recurrent Glioblastoma Multiforme Treated by Resection with Implantation of Permanent I-125 Seeds and BCNU Wafers**

Ronald E. Warnick, M.D., Borimir J. Darakchiev, M.D., Robert E. Albright, M.D., John C. Breneman, M.D.

OBJECTIVE: Effective treatment options are limited for patients with recurrent glioblastoma multiforme. Survival is usually less than a year. Novel treatment approaches are needed. Localized adjunct treatment with carmustine (BCNU) wafers or permanent, low-activity iodine-125 seeds has been shown to be effective in glioblastoma. This study assessed the efficacy and safety of these therapies in combination following tumor resection.

METHODS: Thirty-four patients with recurrent glioblastoma were treated with maximal tumor resection followed by implantation of BCNU wafers and permanent iodine-125 seeds into the tumor cavity. Patients were followed with clinical evaluations and magnetic resonance imaging scans once every 3 months. Survival and progression-free survival were evaluated.

RESULTS: During follow-up, local disease progression was observed in 27 patients, and 23 of these patients died. Median survival was 72 weeks, and median progression-free survival was 39 weeks. The 12-month survival and progression-free survival rates were 65% and 30%, respectively. Baseline factors associated with prolonged survival included Karnofsky Performance Status $\geq$70 and iodine-125 seed activity $\geq$0.8 mCi per cm^3 of tumor cavity. Brain necrosis developed in 8 patients and was successfully treated with surgery or hyperbaric oxygen therapy.

CONCLUSION: The use of combination adjunct therapy with BCNU wafers and permanent iodine-125 seeds resulted in survival that compares favorably with data from similar studies in recurrent glioblastoma. The incidence of brain necrosis appeared to be higher than that expected with either treatment alone. Brain necrosis was manageable and did not affect survival. This novel approach warrants further investigation in recurrent and newly diagnosed glioblastoma.

FRIDAY, OCTOBER 20th

11:00 - 11:15 AM **Academy Award Honorable Mention**
Galectin-1, a Gene Preferentially Expressed at the Tumor Margin, Promotes Glioblastoma Invasion

Toussaint III, Gerard, Nilson, AE, Goble, JM, Ballman, KV, James, CD, Uhm, JH

High-grade gliomas are recalcitrant to local therapy in part because of their ability to invade the normal brain parenchyma surrounding these tumors. Using a xenograft model that retains the invasive characteristics of patient-derived tumors, we compared gene expression profiles of actively invading cells at the tumor-brain interface to those of cells at the tumor core. Controlling for the possible contamination of samples collected at the invasive edge by normal mouse brain cells allowed us to identify galectin-1 as preferentially expressed at the tumor margin. To delineate the functional significance of galectin-1 overexpression, we generated appropriately transfected clones of the U87MG glioma cell line. Galectin-1 overexpression did not increase the proliferative rate of these cells nor did it alter their attachment to extracellular matrix components. However, the rate of two-dimensional migration and transwell invasion was proportional to the level of galectin-1 expression. In addition, animals hosting orthotopic xenograft tumors from U87MG cells overexpressing galectin-1 had shorter survival times than parental-cell xenografted mice. The survival differences may be explained by changes in the invasive pattern of these tumors. Based on these and other existing data, galectin-1may be an important mediator of the invasive phenotype of high-grade gliomas.

FRIDAY, OCTOBER 20th

11:15 - 11:30 AM **Academy Award Winner**
A2B5 White Matter Progenitors as Glioma Cells of Origin

Ogden, Alfred T, Lochhead, RA, Fusco, D, Lopez, K, Ellis, JA, Kang, J,Waziri,AE, Assanah, M, Canoll, P, Bruce, Jeffrey

Introduction:

Identification of the cell of origin in gliomagenesis has important implications for understanding this therapeutically recalcitrant disease. Many reports have outlined similarities between neural stem cells and glial tumors, but none have presented evidence regarding a candidate cell of origin. The stem cell marker, CD133, has been reported to identify putative glioma stem cells. Recently, we demonstrated that retroviral infection of white matter progenitors to induce high levels of PGDF rapidly produces tumors that display all the histological hallmarks of glioblastoma. These tumors are largely composed of non-transformed A2B5+ white matter progenitors, which are the most abundant and best characterized neural stem cells in the adult human brain.

In this study we investigate the relationship between white matter progenitors and human glial tumors, showing that human glial tumors also contain high percentages of cells expressing A25B with progenitor like properties. Expression of O4, considered to connote commitment of A2B5 progenitors to the oligodendroglial lineage, was prevalent as well. Based on co-expression of glial markers CD133 and IL-13 receptor, the presence of aneuploidy in the A2B5+ population, and the ability to form tumors in vivo, some of these cells are clearly transformed tumor cells. Others, however, lack glioma marker expression and are grossly euploid, suggesting heterogeneity within the A2B5+ population that may be consistent with progenitor recruitment.

Given their abundance, the predilection of human gliomas for white matter, and the prevalence of A2B5 expressing cells with progenitor-like properties in human and experimental gliomas, we propose white matter progenitors as the likely cells of origin for a large percentage of human gliomas.

Conclusion:

A2B5+ white matter progenitors are likely cells of origin for many human gliomas. A2B5 and O4 expression is wide spread in adult gliomas over a range of grades. The ratio of O4/A2b5 expression inversely correlates with tumor grade. Many A2B5+ cells within tumors are transformed based on co-expression of glioma markers, aneuploidy, and the ability to generate tumors in vivo. Other A2B5+ cells isolated from tumors are euploid, lack glioma like marker expression, and could represent recruited progenitors. Unlike A2B5+ cells from PDGF-driven rat glioma model, human A2B5+ tumor cells lack the ability to differentiate into oligodendroglia, raising questions regarding the effects of transformation on differentiation potential.

SATURDAY, OCTOBER 21st

7:30-7:45 AM **Upregulation of Mitoxantrone Resistance Protein (MXR/ABCG2) in Patients with Medically Refractory Temporal Lobe Epilepsy**

Kitti Kaiboriboon, M.D., Brian K. Alldredge, Pharm. D. , ***Nicholas M. Barbaro, M.D.***, Andrew W. Bollen M.D., Everett J. Austin, M.D., Stephen L. Nutik, M.D., Daniel H. Lowenstein, M.D., Deanna L. Kroetz, Ph. D.

RATIONALE: Upregulation of members of the ATP-binding cassette (ABC) transporter protein superfamily has been implicated as a potentially important factor in the development of refractory epilepsy. To date, brain overexpression of P-glycoprotein, multidrug resistance-associated protein 1 (MRP1) and MRP2 has been documented in refractory epilepsy. Recently, another member of the ABC superfamily named mitoxantrone resistance protein (MXR, encoded by *ABCG2*), has been detected in cerebral capillary endothelial cells. However, the role of MXR in refractory epilepsy is unknown. To further elucidate the role of MXR in refractory epilepsy, we examined the distribution and expression of MXR in resection specimens from patients with medically refractory temporal lobe epilepsy (TLE).

METHODS: A total of 62 brain specimens from 18 consecutive patients surgically treated for medically refractory TLE and 4 normal brain specimens were analyzed in these studies. A combination of immunohistochemistry (IHC) and quantitative reverse transcriptase polymerase chain reaction (qRT-PCR) was used to examine the distribution and expression level of MXR protein and *ABCG2* mRNA. Expression at the protein and RNA levels in the lesional tissue was compared with expression in the histologically normal adjacent tissue.

RESULTS: Fourteen patients had mesial temporal sclerosis (MTS) and four patients had focal cortical dysplasia (FCD). IHC was performed in 8 specimens from patients with FCD, 27 specimens from patients with MTS, and 4 specimens from normal brain. In all FCD and the majority of MTS cases, MXR expression was noted in neurons from both lesional and histologically normal adjacent tissues. However, immunoreactivity was more abundant and more intense in

the lesional tissue. In normal brains, strong MXR immunoreactivity was detected in blood vessels but not in neurons or glial cells from the hippocampus or lateral temporal cortex. qRT-PCR was performed in 27 specimens from patients with MTS. There was a 1.6-fold increase in *ABCG2* mRNA in the hippocampus compared to that in the lateral temporal cortex.

CONCLUSION: Our findings suggest that upregulation of MXR/*ABCG2* is widespread in human epileptic brain. This supports a potential role of MXR in the cellular mechanisms underlying drug resistance.

SATURDAY, OCTOBER 21st

7:45-8:00 AM **Identification of Novel Drugs that Target Neural Stem Cells by High Through-Put Screening**

Phedias Diamandis, Michael Tyers, ***Peter Dirks, M.D.***

The identification and characterization of neural stem cells from the mammalian brain has been enormously aided by the development of cell culture techniques. The most widely used culture assay of neural stem cells is the neurosphere assay. This in vitro assay system has been vital to support the importance of effects of extrinsic signals and intrinsic genetic alterations on neural stem cells and progenitors in vivo. In addition to genetic approaches to understanding neural stem activity, high through-put screening (HTS) of small molecules will aid in developing clinical drugs that can be used for in situ or ex vivo neural stem cell manipulation. Here we report the first small molecule high-throughput screen (HTS) of mouse neurosphere cultures. Using a library of 1,280 pharmaceutically active compounds, we identify novel drugs which altered the proliferation of neurosphere cultures as measured by the MTT proliferative assay. From the 1280 compounds present in the library, we identified 234 (18.28%) candidate small molecules that decreased neurosphere proliferation by at least 3 standard deviations when compared to controls. To our surprise, many of the classes of active compounds in the neurosphere assay are presently in clinical use for a variety of nervous system diseases. The classes of drugs effective against neural precursor cells will be discussed. These data support the use of this assay to identify new candidate drugs as well as off target effects of current drugs in clinical use that effect neural stem cell self renewal and proliferation, and have implications for brain repair as well as for developing novel anti-cancer drugs that may be effective on brain tumor stem cells.

SATURDAY, OCTOBER 21st

8:00-8:15 AM **Directed Therapy with Polymers for Brain Tumors**

Henry Brem, M.D.

Polymer delivery systems are being utilized to target multiple pathways in tumorigenesis. Polymer delivery of antiproliferative agents such as the microtubule stabilizer paclitaxel and the topoisomerase I inhibitor camptothecin show promise in preclinical studies. Resistance modifiers can also be delivered optimally. Local delivery of the recently approved oral agent temozolomide alone and in combination with both resistance modifiers and radiation therapy is being explored. Tumor angiogenesis is being targeted with the release of the anti-angiogenic agents: endostatin and minocycline. Local delivery of the anti-glutaminergic agent riluzole protects normal brain and decreases tumor invasion. Vaccines have also been delivered successfully by this mechanism. Novel applications under investigation include delivery of siRNA and enzymes. A logical evolution of local controlled release is the creation of devices that release multiple agents in multiphasic patterns to optimize therapeutic regimens or more accurately recapitulate the physiologic norm. This was addressed with the development of biodegradable poly (L-lactic acid) multi-well passive microchips with poly (D,L-lactic-co-glycolic acid) membranes. Improved clinical outcomes with Gliadel™ demonstrate the utility of controlled local delivery for brain tumors. Development of this approach of therapeutic research for the treatment of brain tumors offers the ability to circumvent physiologic barriers and effectively deliver a multitude of novel agents in a controlled manner.

SATURDAY, OCTOBER 21st

8:15 - 8:30 AM **Insular Gliomas: Surgical Technique, Morbidity Assessment and Outcome Analysis**

Mitchel S. Berger, M.D., Daniel Hirt, B.S., Kathleen R. Lamborn, Ph.D., G. Evren Keles, M.D.

Despite recent advances in intraoperative imaging and contemporary surgical technology, surgery for insular gliomas remains challenging. Although grouped together as "insular" gliomas, tumors located at different anatomical parts of the insula and their various patterns of extension necessitate surgical approaches that are specific to the location and growth pattern of the tumor.

We retrospectively analyzed our series of insular gliomas operated on by the senior author. The aim of this study was to describe the surgical technique for both dominant and non-dominant insular gliomas, define operative morbidity and assess outcome based on the location and extension of insular gliomas. In addition, extent of resection was evaluated quantitatively with the endpoint being time to tumor progression.

A total of 109 patients with insular gliomas met the inclusion criteria which consisted of availability of clinical information and follow-up, as well as pre- and early postoperative magnetic resonance scans, and a detailed description of the surgical procedure including the maps generated during intraoperative cortical and subcortical stimulation mapping. The median age of the patient population was 44, and 58% were female (n=63). 71 patients (65%) had a tumor located in the dominant hemisphere and were operated on with the use of language mapping techniques. The majority of the tumors were low-grade (71%, n=77), and the median follow-up is 5.2 years.

We will report on the extent of resection as it relates to the location of the tumor in the insula based on a new classification scheme for predicting extent of resection, and compare progression-free survival and malignant transformation rates (which will be updated at the time of the meeting) with non-insular low grade gliomas.

SATURDAY, OCTOBER 21st

8:30 – 8:45 AM **Human Bone Marrow-Derived Mesenchymal Stem Cells As Delivery Vehicles for Glioma Therapy**

Frederick F.Lang, M.D., Akira Nakamizo, M.D., Frank Marini, Ph.D., Michael Andreeff, M.D., Ph.D.

The poor survival of patients with malignant gliomas relates partly to the inability to deliver therapeutic agents to the tumor. To improve delivery, we assessed the tropism of human bone marrow-derived mesenchymal stem cells (hMSCs) for brain tumors. hMSCs were isolated from bone marrow, fluorescent-labeled and injected into the carotid artery of mice bearing human glioma intracranial xenografts. hMSCs were seen exclusively within the tumors after intravascular administration. Studies using sex-mismatched hMSCs (male, XY genotype) confirmed the presence of Y+-hMSCs within female xenografts (U87, XO) after intracarotid injection. Bioluminescence imaging using a luciferase construct to visualize hMSCs documented localization of functional hMSCs in real time within tumors *in vivo*. Similar results were found when mouse MSCs (C57/B6) were injected intravascularly into mice bearing syngeneic gliomas (AST11.9). To assess the potential of hMSCs to track human gliomas, we injected hMSCs directly into the hemisphere opposite an established human glioma, and demonstrated that hMSCs were capable of migrating into the xenograft *in vivo*. Likewise, *in vitro* matrigel invasion assays suggested that tumor-derived growth factors (PDGF, EGF, or SDF-1-alphạ but not FGF or VEGF) may underly the tropism of hMSCs for gliomas. Most importantly, hMSCs engineered to release IFN-beta significantly increased the survival of animals harboring intracranial glioma xenografts. Other studies demonstrated the potential of hMSCs to deliver to tumors Delta-24-RGD, a tumor selective, oncolytic adenovirus. These studies demonstrate that hMSCs have a tropism for human gliomas that may be mediated by tumor-derived growth factors, and that can be exploited to therapeutic advantage.

SATURDAY, OCTOBER 21st

8:45 – 9:00 AM **Blockade of Glioma Invasion by Inhibitors of GSK-3**

E. Antonio Chiocca, M.D., Ph.D., M. Oskar Nowicki, Ph.D., Jennifer L. Cutter, Ph.D., Sean lawler, Ph.D.

Infiltration of normal brain by invading tumor cells is a major factor in the recurrence and poor prognosis seen in malignant gliomas. Therapeutic strategies to prevent the invasion process or to target invading cells are therefore highly sought after. Here we report that glioma cell invasion in vitro and in vivo can be inhibited by pharmacologic blockade of glycogen synthase kinase-3 (GSK-3). Invasion is blocked in all cell lines we have so far tested using multiple assays such as spheroid invasion, transwell migration and scratch assays. The inhibition was dose-dependent and reversible even after prolonged (96 hour) of drug exposure. Microscopic studies of drug treated cells revealed a change in morphology, the cells no longer sending out protrusions at the leading edge. In vivo animal models show a qualitative decrease of glioma infiltration in brain after pharmacologic blockade of GSK-3. This data suggests that targeting GSK-3 or GSK-3 related pathways may be relevant in the treatment of invasive gliomas.

SATURDAY, OCTOBER 21st

9:00 - 9:15 AM **Repeat Blood Brain Barrier Disruption in Patients with Recurrent Primary Central Nervous System Lymphoma**

John McGregor, M.D., Eric Bourekas, M.D.

Intraarterial chemotherapy (IA) with blood brain barrier disruption (BBBD) without radiation therapy has been demonstrated to be effective in the treatment of primary central nervous system lymphoma (PCNSL) with a five year survival rate of 42% without cognitive loss. Patients with relapsed disease remain a difficult treatment group. Previous reports have described the effectiveness of repeat BBBD with IA carboplatin therapy for treatment of recurrent PCNSL following initial treatment with a Methotexate-based regime. We are currently participating in a multicenter evaluation of a protocol which includes intravenous rituximab given in association with BBBD for recurrent disease. We have treated 3 patients (2 males, 1 female, ages 49, 59 and 63 respectively) diagnosed at 62, 10, 25 months from completion of first BBBD therapy. The 3 patients have undergone at total of 48 disruptions. One patient has had a partial response (PR) and two have had a complete response at 3, 5, and 16 months from onset of recurrent treatment. There have been no observed toxicities or treatment related complications. The ability to enhance the delivery of large molecules such as rituximab across the blood brain barrier may improve treatment of CNS malignancies.

SATURDAY, OCTOBER 21st

9:15 - 9:30 AM **High-Resolution Patient-Based Computational Fluid Dynamic Analysis of Intracranial Stenosis**

Adel M. Malek, M.D., Ph.D., Clemens M. Schirmer, M.D.

INTRODUCTION: Intracranial stenosis is associated with a significant risk of stroke despite anti-platelet and anticoagulant therapy. The hemodynamic conditions in and around intracranial lesions are poorly defined. We examined the flow pattern and wall shear stress (WSS) magnitude and spatial gradients in patient-based intracranial stenosis geometries using computational fluid dynamic (CFD) analysis.

METHODS: High-resolution three-dimensional rotational angiographic images of eight intracranial stenotic lesions were obtained in seven patients. Following reconstruction and generation of a fine resolution computational mesh, CFD analysis was performed under pulsatile conditions assuming non-Newtonian properties of blood. Non-laminar and transitional flow conditions were captured with a k-omega turbulence model.

RESULTS: In the post-stenotic region, time-dependent complex flow patterns with two-fold increased axial velocities and areas of recirculation were observed. The mean Reynolds number in the throat of the stenoses was 219 (range 63-495). Increased helicity was also noted in the post-stenotic region consistent with three-dimensional twisting of this non-laminar transitional flow. Endothelial cells in the throat of the stenosis experience transients of 10-fold increased WSS magnitudes along with direction reversal of WSS in the post-stenotic region. The normal curvature of the vessel was seen to influence the relationship between the flow pattern and WSS, including the formation of heterogenous patterns of WSS and viscosity distal to the stenosis.

CONCLUSIONS: CFD simulations of intracranial stenosis offer valuable insights into the abnormal transition from a laminar flow pattern in a healthy vessel to complex flow patterns with recirculation and turbulent features. The flow pattern depends on the severity and location of the stenosis as well as the geometric constraints such as bends and external confines of the bones at the skull base. These complex patterns of flow and WSS may help quantify the thromboembolic risk of a given intracranial lesion and thus serve as a therapeutic guide.

SATURDAY, OCTOBER 21st

9:30 - 9:45 AM **DBS for Tourette Syndrome: From Clinical Case to Clinical Trial**

Robert J. Maciunas, M.D., M.P.H., F.A.C.S., Brian N. Maddux, M.D., Ph.D., David E. Riley, M.D., Christina M. Whitney, R.N.C.S., D.N.Sc, Mike R. Schoenberg, Ph.D., Paula J. Ogrocki, Ph.D., Jeffrey M. Albert, Ph.D., Deborah J. Gould, M.D.

Background: Medically refractory Tourette syndrome (TS) can be disabling. We performed bilateral thalamic DBS in one case of compassionate use. The results led to institutional interest, media attention, and patient referrals. We chose to conduct a prospective, double-blind crossover trial of DBS in five adults with TS.
Methods: We obtained IRB approval, FDA IDE clearance, and external funding for this trial. Bilateral thalamic electrodes were implanted. Optimized stimulator settings were established. Subjective and objective results were assessed in a double-blind randomized fashion for four weeks, with each week spent in one of four states of activation of unilateral or bilateral stimulation. Results were similarly assessed three months after un-blinded bilateral stimulator activation in an open fashion.
Results: In the randomized phase of the trial, a statistically significant ($p < 0.03$, Friedman's exact test) reduction of score on the modified Rush Video Rating Scale was identified in the bilateral on state. Improvement in motor and sonic tic counts, as well as the Yale Global Tourette Severity Scale and Tourette Syndrome Symptom List scores, was also noted. Benefit was persistent at three months after conclusion of the randomized portion of the trial. Quality of life indices were improved. Three of five patients improved markedly by all primary and secondary outcome measures.
Conclusion: This trial confirmed our initial impressions after one case. Bilateral thalamic DBS appears to reduce tic frequency and severity in some patients with TS who exhaust medical therapy. The impact of the approval process on performing trials of experimental neurosurgery will be discussed.

SATURDAY, OCTOBER 21st

9:45 - 10:00 AM **Temporal Lobe Surgery for Intractable Epilepsy in Children: An Analysis of Outcomes in 126 Children**

James T. Rutka, M.D., Mony Benifla, M.D., Carter Snead III, M.D., Hiroshi, Otsubo, M.D.

Object: Temporal lobectomy is a well established neurosurgical procedure for temporal lobe epilepsy. In this study, we conducted a retrospective review of children with drug-resistant temporal lobe epilepsy to evaluate seizure outcome after temporal lobe surgery.
Methods*:* We reviewed the medical records of 126 children who had surgery for temporal lobe epilepsy at The Hospital for Sick Children between 1983 and 2003. The records were examined for pre-operative and intra-operative factors which could predict patient outcome after surgery.
Results: The mean age at seizure onset was 5.9 years. The mean seizure duration prior to surgery was 5.6 years. All patients had either pre-operative CT or MRI scans, or both. The mean age at the time of surgery was 13.5 years. Sixty-two patients underwent left temporal, and 64 right temporal resections. The histopathology of the temporal resections revealed low grade brain tumors in 65 (52%) children. Mesial temporal sclerosis was found in 16 patients (13%), astrogliosis in 15 (12%), cortical dysplasia in 8 (7%). Post-operative follow-up of at least 2 years was available for 106 patients and ranged up to 13.0 years. Seventy-four percent of patients had an Engel class I and II outcome. Patients with temporal lobe lesions had better outcomes in comparison to those without lesions ($p < 0.05$). Patients without a history of secondary generalization of seizures also had a better outcome when compared to those with secondary generalization. Twelve patients had a second temporal lobe procedure for intractable recurrent seizures. After a second procedure, 7 patients returned to a seizure-free state.
Conclusions*:* Temporal lobe resections for epilepsy in children are effective and safe procedures with a favorable impact on seizure control. Repeat temporal resections for recurrent seizures may also be effective in restoring a seizure-free outcome to children.

SATURDAY, OCTOBER 21st

10:15 - 10:30 AM **Multimodality Management of Skull-Base Chordomas: Experience of a Single Institution**

M. Necmettin Pamir, M.D.

Between 1986 and April 2006, 41 chordomas and 5 chondrosarcomas received multimodality treatment in our department. Of these 46 patients 28 patients were male and 18 were female and the patients' age ranged between 5 and 82 (average of 39 years). A total of 98 procedures (69 tumor excision surgeries, 19 gamma-knife radiosurgeries and other operations to treat complications) were performed. The mean follow-up period is 76 months. Analyses of radiological features relatieve to histopathology were performed for 42 patients. Immunehistochemical comparison of chordomas were studied. Genomic instabilities were investigated by FISH.

Nine patients died during follow-up. Two of the deaths were due to surgical complications, six resulted from tumor recurrence and one was unrelated to neoplasia. Residual rate with conventional approaches were 30% and with skull-base approaches were 14.2%. Surgical morbidity were 19.4%. During the 32 months mean follow-up time of the gamma-knife radiosurgery, local tumor control was observed.

Conclusions:

- Chordomas and chondrosarcomas have completely different survival rates because of this they should be considered differently
- Completeness of resection is determined by the approach. Skull base approaches enable more complete resections
- None of the MRI or CT features appear to be useful for differentiating chordomas from chondrosarcomas preoperatively.
- Gamma-knife radiosurgery may provide local tumor growth.
- Recurrence in chordomas may correlate with expression of the growth factors TGF-α and β-FGF
- Cumulative genetic instability at the 1q25 locus were detected

SATURDAY, OCTOBER 21st

10:30 – 10:45 AM **Blood-Brain-Barrier Disruption is Mediated by Flow Arrest and Circulating Leukocytes**

Lijana Bengez, Mohammed Hossain, Vince Fazio, Damir Janigro, ***Marc Mayberg, M.D.***

Cerebral ischemia causes disruption of the blood-brain barrier (BBB), mediated in part by inflammatory processes involving cytokines and alterations in the expression and activity of membrane metalloproteinases (MMP-2 and MMP-9) and their inhibitors (TIMP-1/TIMP-2). We used a dynamic *in vitro* BBB model to analyze the individual contributions of flow, cytokine levels and circulating white blood cells (WBC) on BBB and MMP/TIMP expression under various conditions of oxygenation and glucose levels. With WBC-free perfusate, both normoxic/normoglycemic or hypoxic/hypoglycemic flow cessation and reperfusion had only a minor, transient effect on cytokine release and BBB-permeability. After addition of WBC to the luminal perfusate, normoxic/normoglycemic flow cessation/ reperfusion caused biphasic BBB opening associated with increases in TNF-alpha and IL-6 , increased levels (ELISA) and activity (zymography) of MMP-9 and MMP-2, and reduction of TIMP-1/TIMP-2. Pretreatment of WBC with a cyclooxygenase inhibitor (ibuprofen), inhibited cytokine release and prevented BBB disruption. Antibody to IL-6 or TNF-alpha, or infusion of TIMP-2 prevented BBB disruption, and eliminated increases in MMP expression and activity. To clarify whether post-ischemic MMP-9 is derived from brain vs. circulating WBC, chimeric mice were generated by irradiation and bone marrow transplantation in combinations of wild-type (*wt*) and MMP-9 knockout (*ko*) mice . Gel zymography of the ischemic hemisphere at 24h after 90 minute MCA occlusion/reperfusion demonstrated MMP-9 activity only in animals with *wt* marrow; MMP-9 was barely detectable in mice with *ko* marrow. These data indicate that circulating blood leukocytes and loss of flow are major factors in metalloprotease-mediated BBB disruption, and that bone marrow-derived WBC are the critical source of MMP-9 in the ischemic brain.

Supported By: AHA SDG 0230015N, NIH NS046513, NS043284

SATURDAY, OCTOBER 21st

10:45 - 11:00 AM **Erythropoietin Improves Outcome in Adult Rats After Perinatal Brain Injury**

Shenandoah Robinson, M.D.

Children who are born preterm are susceptible to cerebral palsy, epilepsy, and cognitive delay. Current interventions have not reduced long-term neurologic morbidity. Erythropoietin (EPO), a growth factor required for neurogenesis, is neuroprotective in adults. We investigated whether EPO improves outcomes after perinatal brain injury using a rat model of systemic prenatal hypoxia-ischemia. This systemic prenatal insult reliably induces damage that mimics placental insufficiency in humans at about 25 weeks gestation.

The uterine arteries were transiently occluded in rats on embryonic day 18, and pups were born at term. Sham controls had surgery without arterial occlusion. Newborn pups were administered 2000U/kg EPO or saline intraperitoneally daily for 5 days. Motor skills were assessed in pups and adults. Seizure threshold was tested in adults using an escalating dose paradigm with pentylenetetrazol. Histological, cellular, and biochemical techniques were used to define the function of EPO and its receptor on neural cells after the insult.

Postnatal day 9, EPO-treated insult pups had fewer apoptotic cells, reactive microglia ($p<0.001$), and more GABAergic neurons ($p=0.01$), compared to saline-treated insult pups ($n=3$). At 2 weeks, EPO-treated insult rat pups had significant improvement in motor skills, compared to saline-treated insult pups ($p=0.05, n=13$-15). Adult EPO-treated rats showed elevation of the seizure threshold back towards baseline, compared to saline-treated insult rats. Similarly, after EPO treatment GABAergic neuronal cell number was also improved in adults in multiple cortical regions.

Conclusions: These results suggest that postnatal EPO improves motor skills and seizure threshold in rats that have suffered a prenatal systemic insult.

SATURDAY, OCTOBER 21st

11:00 - 11:15 AM **Cerebral Revascularization for Moyamoya Disease**

Kiyohiro Houkin, M.D.

Background

It is well known that surgical revascularization is effective for moyamoya disease. It is supposed to be safe surgery. However, the peri-operative complication and its morbidity has not well analyzed. The authors report complication and morbidity rate of surgical treatment for moyamoya disease.

Patients and Method

One hundred and thirty-two patients including 52 adults patients and 80 pediatric patients are included in this study. These are consecutive cases experienced in Hokkaido University and Sapporo Medical University from 1992 to 2004. For these 132 patients, 218 surgeries have been performed using combined surgical technique of direct bypass and indirect surgery of encephalo-duro-arterio-myo-synangiosis (EDAMS).

Results

Among these 218 surgeries, 8 cases of peri-operative complications including two cases of intracerebral hemorrhage, one case of serious seizure, two cases of cerebra infarction and two cases of infection are seen. Permanent neurological deficit has seen in two cases of intracerebral hemorrhage and one case of cerebral infarction. Thereofore, the surgical morbidity was 2.3% for patients number and 1.4% for surgeries.

Conclusion

The complication and morbidity rate of combined surgery of direct and indirect bypass is supposed to be 1-4% for patients with moyamoya disease. The surgical treatment for moyamoya disease is quite safe option. However, this complication rate has to be considered in case of surgery in particular minimum symptomatic patients and adult hemorrhagic patients.

SATURDAY, OCTOBER 21st

11:15 - 11:30 AM **Has Regulation Improved the CSF Shunt?**

Stephen J. Haines, M.D., Jeffrey P. Blount, M.D.

BACKGROUND: CSF shunting for hydrocephalus was well established when the FDA was given substantial regulatory authority for medical devices in 1976. Prior to that time the identification of shunt complications was entirely dependent on investigations and publications from the neurosurgical community. The imposition of regulation after 1976 provided an opportunity for a formal regulatory process to have an impact on the safety and effectiveness of these devices.

OBJECTIVE: To investigate the role of regulation in improving the safety and effectiveness of CSF shunts.

METHODS: As part of an Institute of Medicine study of pediatric medical devices, we reviewed 4027 adverse event reports in the FDA MDR and MAUDE databases. The literature on complications of CSF shunting from 1950 to the present was reviewed and classified. The FDA reports were classified using the same classification scheme. FDA health device alerts and action items have been searched for CSF shunt related warnings and recalls. Data mining techniques were applied to the FDA AE reports to determine if significant clusters of complications not identified in the published literature could be detected.

RESULTS: In 1976 the CSF shunt was determined to a class II device, not requiring burdensome "pre-market approval" (PMA) studies prior to marketing and sale. No shunt device has been required to undergo a formal PMA process by the FDA. Five health device alerts have been issued, all related to manufacturing difficulties with specific shunt parts. The FDA has never recalled a CSF shunt. The published literature has focused far more attention on shunt infection than is evident in the FDA device failure reports. Otherwise the complications reported are similar. Data mining identified a cluster shunt fractures from two companies which were also identified in three published reports.

CONCLUSIONS: If the CSF shunt were introduced today, it would undoubtedly be a class III device requiring intense pre-market scrutiny and post-market surveillance. There is little evidence that the passive monitoring system imposed by the FDA in 1976 has played an important role in improving the safety or efficacy of CSF shunts: no important complication of CSF shunts has been identified by the FDA that was not identified in the medical literature. There is no evidence that the adverse event reporting system has led to important improvements in shunt function or safety. However, the enforcement of compliance with technical standards for bio-compatibility manufacturing quality, sterility, etc. has been associated with a noticeable absence of complications related to these fundamental aspects of manufacturing.

SATURDAY, OCTOBER 21st

11:30 - 11:45 AM **Predicting Post Operative Hydrocephalus in Pediatric Post Fossa Tumor Patients**

J. Drake, J. Riva-Cambrin, M. Lamberti-Pasculli, M. Sargent, D. Armstrong, R. Moineddin, D. Cochrane

Introduction: In pediatric patients with post fossa tumours (PFT), much controversy surrounds the risks of developing post operative hydrocephalus (POH), typically occurring in 30%. Reports of improved outcomes with pre tumor surgery endoscopic third ventriculostomy (ETV), broadly applied, has further confused things. We developed a pre-operative prediction score based on our own institution's patients, to identify those at highest risk. The score was then validated on a separate PFT cohort at another institution.

Methods: Following a literature search and having sought expert opinion, potential important patient pre operative variables were identified. Data from 343 patients (1988-2003) from HSC Toronto was analyzed retrospectively. Mulitivariate logistic regression was used to identify risk factors and construct a prediction model. This was simplified into a prediction score representing preoperative risk of hydrocephalus. Data from 111 patients (1990-2003) from BCCH Vancouver (1988-2003) was then used to validate the prediction model.

Results Significant risk factors and their prediction score points () were: age<2 (3), papilledema (1), hydrocephalus severity (2), cerebral metastatic spread (3), and presumed tumor pathology (1): total range (0-10). Scores > 5 were considered 'High Risk'. When applied to the Vancouver cohort, the prediction score was validated using chi-square statistics (p-value of 0.0486) and misclassified only 20.7% (23/111) of patients' risk.

Discussion: This prediction score resolves many of the issues concerning risk of POH in PFT patients. We believe it will allow improved patient counseling, and surgical planning. It additionally identifies patients at high risk, who selectively, might be exposed to the risks of pre operative ETV to improve outcome.

SATURDAY, OCTOBER 21st

11:45 - 12:00 PM **Regional Neurotrauma Care in Memphis, TN**

Shelly D. Timmons, M.D., Ph.D., F.A.C.S.

Regionalization of neurotrauma care in the U.S. has recently become a popular goal amongst neurosurgeons. Consideration of this concept has been driven by several issues, some of which are at crisis levels in certain areas: neurosurgical work force issues (including emergency neurosurgery coverage), declining overall reimbursement for medical care, increasing liability costs and exposure, and advances in the field of neurotrauma critical care.

This paper assesses regional neurotrauma care in Memphis, TN and the surrounding area, describing the role of the demographics, geography, and economics of the region, the context of the history of the various institutions providing trauma care, and the mechanisms by which neurotrauma care is provided. Those areas in which the regional neurotrauma care system serves as a "best practice" as well as those factors which threaten the delivery of high-quality healthcare in this arena are highlighted. Case mix and critical care statistics are presented, along with outcomes data. The interdependent role of practitioners in other disciplines and the role of graduate medical education in the delivery of trauma care are also discussed.

The system of regionalized neurotrauma care that has evolved in Memphis, TN to care for injured patients in the Mid-South region has resulted in a well-organized center with a large referral base and overall good outcomes. However, the singular nature of the center, the financial challenges in the region, and the early stages of disaster preparedness (specifically with regard to surge capacity for mass casualties) place this system at a moderate degree of risk.

SATURDAY, OCTOBER 21st

12:00 - 12:15 PM **Magnesium Sulfate for Neuroprotection after Traumatic Brain Injury**

Nancy Temkin, Ph.D., Gail D. Anderson, Ph.D., ***Richard Winn, M.D.,*** Richard Ellenbogen, M.D., james Schuster, M.D., Timothy Lucas, M.D., David W. Newell, M.D., Pamela Nelson Mansfield, R. N., B.S.N., Joan E. Machamer, M.A., Jason Barber, M.S. , Sureyya S. Dikemen, Ph.D.

BACKGROUND

Traumatic brain injuries (TBI) represent an important and costly health problem. Previous studies of putative neuroprotective therapies have failed to improve outcome. Supplemental magnesium positively affects many of the processes involved in secondary injury after TBI and consistently improves outcome in animal models.

METHODS

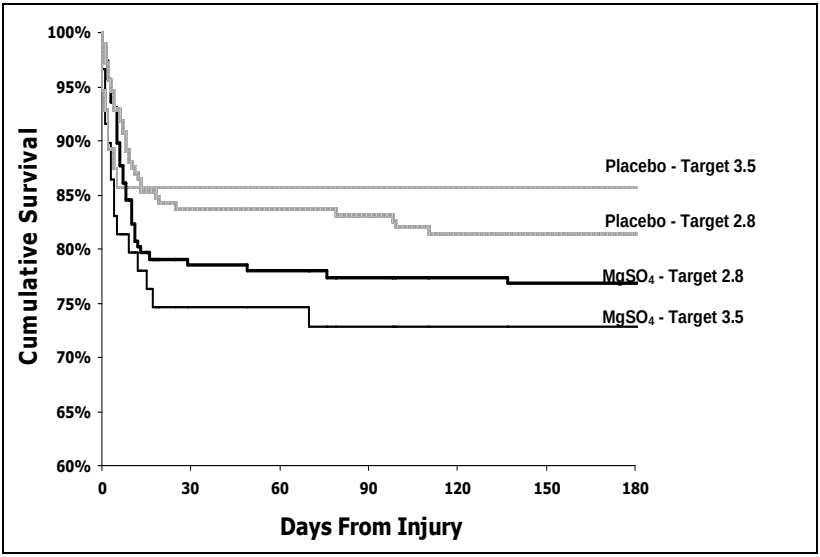
In a double-blind trial, 499 people age 14 or over with moderate or severe TBI were randomized to one of two doses of magnesium or placebo beginning within eight hours of injury and continuing for 5 days. Magnesium doses were targeted to achieve serum magnesium concentrations of 1.4 mmol/L or 1.75 mmol/L. The primary outcome was a composite of mortality, seizures, functional measures, and neuropsychological tests evaluated up to six months after injury. The composite outcome allowed a study with 400 cases to have power equal to a 900 case study using only the dichotomized Glasgow Outcome Scale.

RESULTS

At either dose, magnesium showed no significant positive effect on the composite or individual measures. Moreover, those randomized to magnesium at the lower dose did significantly worse on the composite ($p=.02$). There was a trend toward higher mortality with each dose of magnesium. No subgroups were identified in which magnesium had a significantly positive effect.

CONCLUSIONS

Contrary to the positive results of supplemental magnesium in experimental models of TBI, infusions of magnesium given within 8 hours to patients with moderate or severe TBI and continued for five days were not neuroprotective in this trial and may well have a negative impact in the treatment of significant head injury.



SATURDAY, OCTOBER 21st

12:15 - 12:30 PM **The Delivery of Emergency Neurosurgical Care**

Alex B. Valadka, M.D., F.A.C.S.

The specialty of trauma surgery is dying. Trauma surgeons have long lamented the progressive decline in interest in careers in trauma. To save the specialty, the American Association for the Surgery of Trauma (AAST) hopes to transform trauma surgery into "acute care surgery." These new specialists would treat both trauma and nontrauma emergencies. Supposed selling points of this new specialty will include controlled lifestyle via defined work shifts, guaranteed salary, and more operative cases than a pure trauma surgeon sees.

The AAST proposal calls for training in ICP monitor insertion and possibly in performance of ventriculostomies and craniotomies. Although the leaders of this AAST proposal are academicians, they cite a perceived void in community neurosurgical coverage as a major reason why general surgeons must perform neurosurgical procedures. However, AANS survey data indicate that most neurosurgeons do participate in emergency coverage. Although some published reports describe good outcomes after non-neurosurgeons perform neurosurgical procedures, other published series, as well as anecdotal data, document substandard results.

Solutions to the problems of emergency coverage must focus on patient welfare, not on protecting the future of one's specialty. Interspecialty cooperation must replace competition. Logic dictates that emergency neurosurgical patients will have better outcomes if they are cared for by neurosurgeons. Regionalization of care may help ensure optimal utilization of the neurosurgical resources in an area. Other parts of the solution will include tort reform, appropriate compensation, and preservation of recent changes to EMTALA that allow neurosurgeons to remain active in the emergency care system.

SPECIAL GUESTS

GUESTS	SPONSORS
Jim Bean Lexington, KY	Don Quest
Josh Bederson New York, NY	Kalmon Post
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Peter Dirks Toronto, Canada	Jim Drake
Wink Fisher Birmingham, AL	Mark Hadley
Peter Gerszten Pittsburgh, PA	Bruce Pollock
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Robert M. Friedlander……..……..1999
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Peng Chen……..…2001
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Gelareh Zadeh. .....….2003
Eric C. Leuthardt……...2004
Manish Aghi……..2005
Alfred T. Ogden…….2006

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, Cincinnato, 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
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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
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The Lincoln Hotel Post Oak, Houston, Texas ..... October 27-30, 1985
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Hyatt Regency, San Antonio, Texas ..... October 7-10, 1987
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The Cloister, Sea Island, Georgia ..... November 3-6, 1994
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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, Georgia……...October 18-21, 2006

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Nicholas Zervas..... 1991
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Donald O. Quest……...2002
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Volker K.H. Sonntag……2004
Martin B. Camins……2005

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Arthur R Elvidge 1946
F Keith Bradford 1949
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J Lawrence Pool 1952
Rupert B Raney 1953
David L Reeves 1954
Stuart N Rowe 1955
Jess D Hermann.....1956
George S Baker 1957
Samuel R Snodgrass.....1958
C Hunter Shelden 1959
Edmund Morrissey 1960
Donald F Coburn 1961-62
Eben Alexander, Jr 1963
George L Maltby 1964
Robert Pudenz 1965
Francis A Echlin.....1966
Benjamin Whitcomb 1967
Homer S Swanson 1968
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Edward W Davis 1971
John R Green.....1972
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Stewart Dunsker 1991
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Martin B Camins…….……2002
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L. N. Hopkins……...2005

PAST SECRETARY-TREASURERS

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

PAST SECRETARIES

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

PAST TREASURERS

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

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DECEASED MEMBERS

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

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)

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
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JOSEPH EVANS ..... Founder 1985
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JOHN FRENCH 1951 1989
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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
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JAMES GREENWOOD, JR. ..... 1952.....1992
Houston, Texas
(Senior)

WESLEY GUSTAFSON ..... 1942.....1975
Jensen Beach, Florida
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WALLACE HAMBY..... 1941.....1999
Pompano Beach, Florida
(Senior)

HANNIBAL HAMLIN ..... 1949.....1982
Providence, Rhode Island
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JOHN HANBERY..... 1959.....1996
Palo Alto, California
(Senior)

MAJOR GEN. GEORGE HAYES…1962 2002
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