THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY

71st Annual Meeting

November 4-7, 2009

American Association of Neurological Surgeons

Jointly Sponsored by the
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of Neurological Surgeons

FUTURE MEETINGS

2010

November 3-6, 2010
The Inn at Spanish Bay
Pebble Beach Resorts, Monterey, CA

2011

October 19-22, 2011
The Fairmont Scottsdale Princess
Scottsdale, AZ

Mark your calendars now!

GENERAL INFORMATION

HOTEL INFORMATION

The Breakers, Palm Beach
One South County Road
Palm Beach, FL 33480

Telephone: (561) 655-6611

REGISTRATION DESK LOCATION AND HOURS:

Wednesday, November 4	South Loggia West	12:00 PM – 6:30 PM
Thursday, November 5	South Loggia West	6:00 AM – 12:00 PM
Friday, November 6	South Loggia West	6:00 AM – 12:00 PM
Saturday, November 7	South Loggia West	6:00 AM – 12:00 PM

PROGRAM SUMMARY

WEDNESDAY, NOVEMBER 4

EVENTS	TIME	LOCATION
Continental Breakfast	8:00 AM-9:00 AM	Magnolia Foyer
Registration	12:00 PM-6:30 PM	South Loggia West
ABNS Meeting	1:00 PM-2:00 PM	Gulfstream 5
Academy Executive Comm. Mtg	3:00 PM-5:00 PM	Gulfstream 3
Reception/Dinner (Rain back-up)	6:30 – 9:30 PM	Ocean Lawn The Circle Room

THURSDAY, NOVEMBER 5

EVENTS	TIME	LOCATION
Registration	6:00 AM-12:00 PM	South Ballroom Foyer
Continental Breakfast (Members)	6:00 AM-8:00 AM	Ponce de Leon 3
Continental Breakfast (Spouse/Guest)	6:30 AM-10:30 AM	Gulfstream 3,4
General Session	7:00 AM-1:00 PM	Ponce de Leon 4
Activities (Mr. Ponce, historian)	9:30 AM-10:30 AM	Meet in the Lobby
Activities	1:00 PM-5:30 PM	Ocean Course
South Beach Reception backup – Magnolia Room)	6:00 PM-7:00 PM	Ocean Terrace (Rain
Dinner backup – Mediterranean Ballroom)	7:00 PM-10:00 PM	Ocean Terrace (Rain

FRIDAY, NOVEMBER 6

Registration	6:00 AM-12:00 PM	South Loggia West
Breakfast (Members)	6:00 AM-8:00 AM	Ponce de Leon 5
Breakfast (Spouse and Guest)	6:00 AM-10:30 AM	Gulfstream 3,4
General Session	7:00 AM-1:00 PM	Ponce de Leon 4
Meg Whitman,	9:00 AM-10:30 AM	Magnolia Room
Activities	1:00 PM-5:30 PM	Ocean Course
Reception	6:00 PM-7:00 PM	Magnolia Room
Black Tie Optional Dinner	7:00 PM-10:00 PM	The Circle

SATURDAY, NOVEMBER 7

Registration	6:00 AM-12:00 PM	South Loggia West
Breakfast	6:00 AM-10:30 AM	Ponce de Leon 6
General Session	7:00 AM-1:00 PM	Ponce de Leon 4

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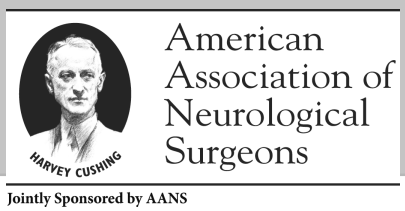
American Academy of Neurological Surgery



Learning Objectives
Upon Completion of this CME activity, participants should be able to:

- Evaluate current methodologies, risk factors, and outcomes for management of cavernous malformations.
- Discuss new approaches and indications for glioma surgery
- Critique the value of surgical and non-surgical treatment options in neurosurgery
- Evaluate the relevance of research methodologies, presented findings, and potential usefulness in the clinical practice of neurosurgery

Accreditation/Continuing Medical Education
This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the AANS and American Academy of Neurological Surgery. The AANS is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

The AANS designates this educational activity for a maximum of 14 *AMA PRA Category 1 Credit*s™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

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Name	**Conflict of Interest**	**Company**
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	Honorarium	Medtronic
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	Consultant Fee	Millenium Pharmaceuticals
Chiocca, E	University Grants/Research Support	NIH
Connolly, S	University Grants/Research Support	NIH
Fehlings, M	Industry Grant Support	Medtronic, Synthes, Depuy
	Consultant Fee	Depuy, Stryker Biotech
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	Consultant Fee	Medtronic, Depuy, Stryker
	Other Support	Fellowship-Depuy, Medtronic
Hoh, B	Industry Grant Support	Micrus Endovascular, Codman Neurovascular
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Levi, A	University Grants/Research Support	NIH/NINDS
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	Other Support	Devices from Boston Scientific, fees for carotid stent training from Abbott, Vascular and ev3
Malek, A	Industry Grant Support	Codman Neurovascular, Microvention
Markert, J	University Grants/Research Support	NIH, Oncolytics
McDermott, M	University Grants/Research Support	NIH
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§§§§§

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

Faculty Name

Abosch, A	Iskandar, B	Rutka, J
Acquaviva, J	Kanaan, I	Ryan, R
Awad, I	Kim, D	Sawaya, R
Bailes, J	Kondiziolka, D	Seol, H
Barrow, D	Lang, F	Shinojima, N
Baskaya, M	Lawton, M	Shi, C
Bendok, B	Lesniak, M	Simard, M
Boulos, A	Liu, Y	Sloan, A
Brotchi, J	Madsen, J	Spetzler, R
Dempsey, R	Medow, J	Watridge, C
Edgar, R	Nanda, A	Zipfel, G
Hodge, C	Oyesiku, N	

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
PRELIMINARY SCIENTIFIC PROGRAM AGENDA

THURSDAY		
Time	**Presentation**	**Presenter**
0730-0825	**GLIOMA SYMPOSIUM**	Mitchel S. Berger, MD Michael R. Chicoine, MD Henry Brem, MD
0825-0837	Results Following Gamma Knife Radiosurgical Anterior Capsulotomies for Obsessive Compulsive Disorder	Douglas Kondziolka, MD
0839-0851	A Supplementary Grading System for Brain Arteriovenous Malformations and Application to 300 Surgical Patients	Michael T. Lawton, MD
0853-0905	Neuroprotective Effects of Mesenchymal Stem Cells Derived from Human Embyryonic Stem Cells in Transient Focal Cerebral Ischemia in Rats	Mustafa Baskaya, MD
0907-0919	Double Hits and the Pathogenesis of Cerebral Cavernous Malformations	Issam Awad, MD
0921-0933	In Vivo Confocal Microscopy of Brain Tumors: A Tool for Real Time Neuropathology	Robert Spetzler, MD
0935-0947	An Assessment of Current Targets for Deep Brain Stimulation Surgery Using Susceptibility-weighted Imaging and 7-Telsa MRI	Aviva Abosch, MD, PhD
0949-1001	Tuberculum Sellae Meningiomas: Surgical Approaches and Modern Day Results	Michael W. McDermott, MD
1003-1023	BREAK	
1023-1035	3-D Endoscopic Pituitary Surgery-A Disruptive Innovation Precipitates the Turning Point	Nelson M. Oyesiku, MD, PhD
1037-1049	Invasion and Migration Autopsy and Clinical Analysis of Modern Day Contact Sport Athletes	Julian Bailes, MD
1051-1103	Folic Acid Supplementation Enhances CNS Regeneration *In Vitro*	Bermans J. Iskander, MD
1105-1117	Platelet derived Growth Factor-BB Mediates the Tropisms of Bone Marrow Mesenchymal Stem Cells for Human Gliomas	Frederick F. Lang, MD
1119-1131	Infection Rate Following Minimally Invasive Spine Surgery	Richard G. Fessler, MD, PhD
1133-1145	Relative Angle of Deflection Correlates with Aneurysmal Rupture Status in Posterior Communicating Artery Aneurysms	Charles J. Prestigiacomo, MD
1147-1159	Magnetic Vortices as Mediators of Cellular Mechano-Transduction and Cell Death in Malignant Glioma	Maciej S. Lesniak, MD
1201-1213	Stent-Assisted Coiling of Paraclinoid Aneurysms Does Not Increase Risk of Treatment	Christopher S. Ogilvy, MD

1215-1227	Endogenous Protective Mechanisms Against Cerebral Vasospasm	Gregory Zipfel, MD
FRIDAY		
Time	**Presentation**	**Presenter**
0730-0742	A Multi-center Prospective Controlled Study Assessing the Efficacy of Surgery in Patients with Differing Severity of Cervical Spondylotic Myelopathy: Results of the AOSpine North America CSM Study in 300 Patients	Michael G. Fehlings, MD, PhD
0744-0756	Novel Genes for Moyamoya Disease	Gary K. Steinberg, MD, PhD
0758-0810	Chemokine Secreting Dendritic Cells for Immunotherapy of Malignant Glioma	Andrew Sloan, MD
0812-0824	Microsurgical Resection of Anterior Skull Base Meningiomas	Anil Nanda, MD
0826-0838	Prospective Analysis of Outcomes After CT Perfusion-Guided Stroke Intervention	Elad I. Levy, MD
0840-0852	Noninvasive Detection of Malignant Glioma Molecular Subtypes Using Magnetic Resonance Perfusion-Weighted Imaging	Donald M. O'Rourke, MD
0854-0906	The Demise of the Posterior Cervical Discectomy	Clarence B. Watridge, MD
0908-0920	Aneurysm Inflow-Angle as a Discriminant for Rupture in Sidewall Cerebral Aneurysms: Morphometric and Computational Fluid Dynamic Analysis	Adel M. Malek, MD, PhD
0922-0934	Glibenclamide is Superior to Decompressive Craniectomy in a Rat Model of Malignant Cerebral Edema	J. Marc Simard, MD, PhD
0936-0948	Gamma Knife Radiosurgery Treatment Outcomes for Trigeminal Neuralgia: The University of California San Francisco (UCSF) Experience	Nicholas Barbaro, MD
0950-1002	The Technique and Clinical Outcomes Using Modest Intravascular Hypothermia after Acute Cervical Spinal Cord Injury	Allan D. Levi, MD, PhD
1004-1024	BREAK	
1024-1038	Cavernous Malformation Radiosurgery: Seeking Détente	L. Dade Lunsford, MD
1040-1058	ACADEMY AWARD WINNER EGFRvIII Antibody Conjugated Iron Oxide Nanoparticles for Targeted Imaging and Therapy of Glioblastoma	Costas Hadjipanayis, MD
1100-1130	Medicine and the Media	Sanjay Gupta, MD
1130-1200	Growing the 21st Century Force	Admiral Vern Clark
1200-1300	Training Neurosurgeons for the 21st Century: Achieving Realignment in the Educational Process	Ralph Dacey, MD
SATURDAY		
Time	**Presentation**	**Presenter**

0730-0825	**CAVERNOUS MALFORMATION SYMPOSIUM**	Daniel L. Barrow, MD Christopher Wallace, MD Murat Gunel, MD
0825-0837	Role of VEGF in Pediatric Hydrocephalus	Joseph R. Madsen, MD
0839-0851	The Role of CXCR4 (Chemokine (C-X-C Motif) Receptor 4) in Human Intracranial Aneurysms	Bernard Bendok, MD
0853-0905	Challenges in Diagnosis and Management of Cushing's Disease	Iman Kanaan, MD
0907-0919	My Experience in 166 Intramedullary Spineal Cord Ependymomas	Jaques Brotchi, MD, PhD
0921-0933	Inhibition of mTOR Minimizes Reactive Astrogliosis Following Brain Injury	Guy M. McKhann II, MD
0935-0947	Microballoon Angioplasty is Safe and Effective Treatment in Small Distal Cerebral Vessels	Alan Boulos, MD
0949-1001	The Use of Positional Magnetic Resonance Imaging to Assess Patients with Low Back Pain	Ehud Mendel, MD
1003-1023	BREAK	
1023-1035	Experimental Therapies Targeted to Micro RNA Expression in Glioma Stem Cells	E.A. Chiocca, MD
1037-1040	Developing a New Generation of Academic Neurosurgeons	Robert J. Dempsey, MD
1042-1049	Multiple Craniotomies in the Management of Multifocal-Multicentric High Grade Gliomas	Raymond Sawaya, MD
1051-1103	Observer Bias in Bio-Surgical Trials of Novel Parkinson's Disease Therapies	Ron L. Alterman, MD
1105-1117	The Guanine Nucleotide Exchange Factor SWAP70 Modulates Glioma	James T. Rutka, MD, PhD
1119-1131	Transcutaneous Inductive Intracranial Pressure Monitor: An Implantable Wireless Device for Diagnosing Shunt Malfunction	Josh Medow, MD
1133-1145	Statins Decrease Excitotoxic Cell Death and Resultant Cortical Dysfunction	Charles Hodge, MD
1147-1159	Length of Stay and Total Hospital Costs of Clipping Versus Coiling for Ruptured and Unruptured Cerebral Aneurysms in the Nationwide Inpatient Sample Database: 2002-2006	Brian Hoh, MD
1201-1213	Understanding and Eliminating Oncogenic EGFR Signaling in Malignant Gliomas	Al Charest, MSc, PhD
1215	Reserved	

THURSDAY, NOVEMBER 5

8:25 – 7:37 AM RESULTS FOLLOWING GAMMA KNIFE

RADIOSURGICAL ANTERIOR

CAPSULOTOMIES FOR OBSESSIVE

COMPULSIVE DISORDER

Douglas Kondziolka, M.D. Robert Hudack, M.D., John C. Flickinger, M.D.

INTRODUCTION: **Obsessive Compulsive Disorder (OCD) is one of the most disabling behavioral disorders. Intrusive, anxiety-provoking thoughts and ritualized behaviors are manifest in many different forms. These can include self-mutilation, negative social behaviors, inability to work or to function in society, and need for hospitalizations. The purpose of this study was to evaluate the results following bilateral radiosurgical anterior capsulotomy for severe medically-refractory OCD.**

METHODS: **We performed gamma knife anterior capsulotomy (GKAC) on three patients with severe, medically intractable OCD. According to our protocol, all patients were evaluated by at least two psychiatrists who recommended surgery. The patient had to request the procedure, and have severe OCD according to the Yale-Brown Obsessive Compulsive Scale (YBOCS). Patient ages were 37, 55, and 40, and pre-radiosurgery YBOCS scores were 34/40, 39/40, and 39/40. Bilateral lesions were created with two 4mm isocenters to create an oval volume in the ventral internal capsule at the putaminal midpoint. A maximum dose of 140 or 150 Gy was used.**

RESULTS: There was no morbidity after the procedure and all returned immediately to baseline function. All patients noted significant functional improvements, and reduction in OCD behavior. Follow-up was at 55, 30, and 28 months. The first patient reduced her YBOCS score from 32 to 24. One patient with compulsive skin picking and an open wound had later healing of their chronic wound and a reduction in the YBOCS score from 39 to 4/40. At 28 months, the third patient is living and working independently and her YBOCS score is 18.

CONCLUSIONS: **Within a strict protocol, gamma knife radiosurgery provided improvement of OCD behavior with no adverse effects. We believe this technique should be evaluated further in patients with severe and disabling OCD and compared to outcomes using deep brain stimulation.**

THURSDAY, NOVEMBER 5

8:39 – 8:51 AM **A SUPPLEMENTARY GRADING SYSTEM FOR BRAIN ARTERIOVENOUS MALFORMATIONS AND APPLICATION TO 300 SURGICAL PATIENTS**

Michael T. Lawton, MD, Helen Kim, PhD, Charles E. McCulloch, PhD, Bahar Mikhak, MS, MPH, William L. Young, MD

OBJECTIVE: Patient age, hemorrhagic presentation, nidal diffuseness, and deep perforating artery supply are important factors when selecting patients with brain arteriovenous malformations for surgery. We hypothesized that these factors outside of the Spetzler-Martin grading system could be combined into a simple, supplementary grading system that would accurately predict neurological outcome and refine patient selection.
METHODS: A consecutive, single-surgeon series of 300 patients with AVMs treated microsurgically was analyzed in terms of change between preoperative and final postoperative Modified Rankin Scale scores. Three different multivariable logistic models (full, Spetzler-Martin, and supplemental models) were constructed to test the association of combined predictor variables with the change in MRS score. A simplified supplemental grading system was developed from the data which combined age, hemorrhagic presentation, and diffuseness in a manner analogous to the Spetzler-Martin grading system, with points assigned according to each variable and added together for a supplemental AVM grade.
RESULTS: Predictive accuracy was highest for the full multivariable model (receiver operating characteristic curve area, 0.77), followed by the supplemental model (0.73), and least for the Spetzler-Martin model (0.66). Predictive accuracy of the simplified supplemental grade was significantly better than that of the Spetzler-Martin grade (P=0.042), with ROC curve areas of 0.73 and 0.65, respectively. The predictive accuracy of the supplemental grade was only slightly less than a full point score with all 7 weighted variables (P=0.364), with areas under the ROC curve of 0.73 and 0.75, respectively.
CONCLUSIONS: This new AVM grading system supplements rather than replaces the well established Spetzler-Martin grading system, and is a better predictor of neurological outcomes after AVM surgery. The supplemental grading scale has high predictive accuracy on its own and stratifies surgical risk more evenly. Supplemental grades can confirm risk predicted by the Spetzler-Martin grade, or in cases of mismatched grades, may alter management decisions. The supplemental grading system is easily applicable at the bedside, where it is intended to improve preoperative risk prediction and patient selection for surgery.

THURSDAY, NOVEMBER 5

8:53 – 9:05 AM **NEUROPROTECTIVE EFFECTS OF MESENCHYMAL STEM CELLS DERIVED FROM HUMAN EMBRYONIC STEM CELLS IN TRANSIENT FOCAL CEREBRAL ISCHEMIA IN RATS**

Yi-Ping Liu, Ph.D., Hakan Seckin, M.D., Ph.D., Yusuf Izci, M.D., Zhong Wei Du, Ph.D., Yi-Ping Yan, Ph.D., ***Mustafa K. Baskaya, M.D.***

INTRODUCTION: Stroke is a leading cause of disability and death in adults worldwide. Recent studies of stem cell therapy in animal models of stroke have shown benefit using mesenchymal stem cells (MSCs) derived from bone marrow or umbilical cord blood. MSCs administrated intravenously migrate to the infarction region, although long-term cell survival and differentiation are limited. Thus, the decrease in lesion volume and the improvement in the functional outcome following MSC transplantation are presumed to be due to the paracrine action of MSC-associated growth factors. MSC therapy faces several issues: the sources of MSCs are limited and their extraction requires invasive procedures; the *ex vivo* expansion of MSCs, although significant, is nonetheless finite and obtaining homogeneous preparations of MSCs from different donors is difficult. On the other hand, MSCs differentiated from human embryonic stem cells (hESC-MSCs or eMSCs) represent an essentially unlimited source of MSCs and eliminate the need for potentially harmful invasive procedures. Although eMSCs show phenotypes similar to MSCs harvested from bone marrow, such as expressing similar cell markers and differentiating into adipocytes and osteoblasts, their neuroprotective effects after focal cerebral ischemia have been untested.

HYPOTHESIS/AIMS: The aims of the present study were to: 1) Differentiate GFP over-expressing hESCs into eMSCs. This would allow larger source for stem cells without any invasive extraction procedure. 2) Use an easy and less invasive administration route for stem cell transplantation such as intravenous injection. 3) Test the migration, differentiation and survival of eMSCs in rat model of transient focal cerebral ischemia. 4) Test whether the transplanted eMSCs could provide neuroprotective effects.

METHODOLOGY: eMSCs were derived from green fluorescence protein (GFP) over-expressing human embryonic stem cells (hESCs). The eMSCs expressed CD29, CD44, CD73, CD105, CD166 and nestin, but not CD34, CD45, CDI06 SSEA-4 or Oct3/4. Twenty million eMSCs in 1 ml of PBS were injected into the femoral veins of spontaneously hypertensive rats (SHR) after transient middle cerebral artery occlusion. The infarction volume was measured and the migration and differentiation of the eMSC in the ischemic brain were analyzed by immunohistochemistry. Neurological behavior effects were assessed by rotarod and adhesive removal tests.

RESULTS: The transplanted eMSCs migrated to the infarction region and differentiated into neurons, which were positive for β-tubulin III, MAP2, HuC and neurofilament, and into vascular endothelial cells, which were positive for vWF. Differentiation into GFAP-positive astrocytes was not observed. The transplanted cells survived in the infarction region for at least 4 weeks. Somatosensory response in the adhesive removal test improved significantly in the first week after cell transplantation, and rotarod motor function significantly improved starting from the second week. The infarction volume in eMSC group was significantly smaller than that in PBS group at four weeks after infusion.

CONCLUSIONS: Human embryonic stem cell-derived MSCs administered intravenously into rats differentiated into neuronal and endothelial cells reduced the infarction volume and improved behavioral functional outcome significantly in transient focal cerebral ischemia. Thus, eMSCs represent an accessible and beneficial source of cells for stem cell transplant studies.

THURSDAY, NOVEMBER 5

9:07 – 9:19 AM **DOUBLE HITS AND THE PATHOGENESIS OF CEREBRAL CAVERNOUS MALFORMATIONS**

Issam Awad, MD, MSc, FACS; Robert Shenkar, PhD; Douglas Marchuk, PhD; Rebecca Stockton, PhD and Mark H. Ginsberg, MD, PhD

INTRODUCTION/HYPOTHESIS: Multifocal cerebral cavernous malformations (CCMs) occur in the setting of genetic predisposition, with heterozygous null mutations of *Ccm2 (*Malcavernin, OSM*), Ccm3* (PCDC10), or *Krit1* (CCM1), a Rap-1 effector that stabilizes endothelial cell-cell junctions. Physical interaction of CCM proteins is required for endothelial cell-cell junctional localization, and deficit of either protein destabilizes barrier function by sustaining activity of RhoA and its effector, Rho kinase (ROCK). CCM lesions also occur in the setting of pre-existing developmental venous anomalies (solitary lesions), and after brain irradiation. We hypothesize that CCM lesion genesis results from "double hits" related to cerebrovascular hyperpermeability.

METHODOLOGY: Excised sporadic and familial human CCM lesions are analyzed for inflammatory response, for phosphorylation of myosin heavy chain (pMHC), as an index of ROCK activation, and for somatic biallelic mutations in *Ccm* genes. Heterozygous *Ccm* +/- murine models are examined for background cerebrovascular hyperpermeability and for the same features in CCM lesions.

RESULTS: A robust oligoclonal antibody mediated immune response is present in human CCM lesions including sporadic and familial cases, genotyped for *Ccm* mutations. ROCK was hyperactive in sporadic and familial human CCM endothelium as judged by increased pMHC expression. Somatic biallelic mutations for *Ccm* genes were demonstrated in endothelial cells of familial cases with known heterozygous germ line mutations in the same genes. Protein-haploinsufficient *Krit1 +/-* or *Ccm2 +/-* mice exhibited increased endothelial permeability *in vitro* and vascular leak *in vivo*, reversible by fasudil, a ROCK inhibitor. Primordial and more mature CCM lesions develop in these animals only in the setting of genetic null mutations in *P53 -/- or MSH -/-* genes which predispose to somatic mutations. Vascular leak and inflammatory response were documented in the murine CCMs, more prominent in mature lesions.

CONCLUSIONS: Human CCM lesions occur in the setting of double hits including somatic mutations within lesions themselves (familial lesions) affecting vascular permeability and a sustained oligoclonal antibody mediated immune response (familial and sporadic lesions). Animal models demonstrate a background of cerebrovascular hyperpermeability in the setting of heterozygous genetic predisposition to CCM disease, which is nullified by fasudil. ROCK signaling pathway is dysregulated in human CCM endothelium (familial and sporadic lesions), indicating that fasudil could ameliorate both CCM disease and vascular leak. Developmental venous anomalies and brain irradiation also likely cause regional cerebrovascular hyperpermeability. We hence formulate a uniform theory for the genesis of CCM lesions in familial and sporadic settings, including double hits of background cerebrovascular hyperprermeability and a sustained antibody mediated immune response. This motivates novel therapeutic strategies for this disease, aimed at modifying one or both hits.

THURSDAY, NOVEMBER 5

9:21 – 9:33 AM **IN VIVO CONFOCAL MICROSCOPY OF BRAIN TUMORS: A TOOL FOR REAL TIME NEUROPATHOLOGY**

Robert W. Ryan, M.D., Tejas Senkar, MDCM., Jennifer Eschbacher, M.D., Stephen W. Coons, M.D., Kris A. Smith, M.D., Peter Nakaji, M.D., **Robert F. Spetzler, M.D.**, Mark C. Preul, M.D.

INTRODUCTION: The histopathological diagnosis of brain tumors plays a critical role in determining treatment strategy. This often begins with a preliminary diagnosis reached by examination of tissue samples provided intra-operatively, and can influence the resection goals of the surgeon. Some shortcomings with the current methodology include sampling errors, the time required for a full examination and an inability to demarcate tumor boundaries, which can often be infiltrative and ill-defined on histology and under the operating microscope as well. Confocal microscopy provides the ability to visualize cellular features by facilitating deeper and clearer observations of intact tissue, but is usually restricted to a laboratory benchtop. We sought to assess the feasibility of using a novel, miniaturized, in vivo confocal microscopy apparatus for the study of brain tumors, including cellular features and tumor boundaries, and comparing the results with conventional histopathology, in both humans and an animal model.

METHODOLOGY: The GL261 mouse glioblastoma model allowed for direct comparison of in vivo confocal microscopy images and histopathological sections cut from the same region of tumor tissue, normal brain and the tumor infiltrative margin. Two types of fluorescent dye, acriflavine and fluorescein, were used. Human patients undergoing tumor resection and able to provide informed consent were enrolled in the study, and after administration of intravenous fluorescein, in vivo confocal microscopy images were recorded from areas of tumor and compared with conventional histology of tissue resected from the same location.

RESULTS: In the mouse model, images of striking cellular detail superbly correlated with corresponding histological images of normal brain and tumor were obtained. The technology convincingly delineated infiltrative tumor boundaries, while generating unprecedented dynamic views of tumor cell motility, structure, and blood supply. In the human patients, a strong correlation was again noted between the confocal images of the tumor and corresponding histological slides, and brain and tumor vasculature were displayed in unique detail.

CONCLUSIONS: In vivo confocal microscopy offers the potential to identify brain tumor cells in situ during an operation, as well as delineating tumor margins. This may provide a major advantage in achieving safe maximal tumor resections and obtaining high yield diagnostic biopsies.

THURSDAY, NOVEMBER 5

9:35 – 9:47 AM **AN ASSESSMENT OF CURRENT TARGETS**

FOR DEEP BRAIN STIMULATION

SURGERY USING SUSCEPTIBILITY-WEIGHTED IMAGING AND 7-TELSA MRI

Aviva Abosch, M.D., Ph.D., Noam Harel, Ph.D.

OBJECTIVE: Deep brain stimulation (DBS) surgery is used for treating movement disorders, including Parkinson's disease and essential tremor. Successful DBS surgery is critically dependent on precise placement of DBS electrodes into target structures. Frequently, DBS surgery relies on indirect targeting: normalized atlas-derived diagrams are superimposed on patient brain MRI scans, followed by microelectrode recording and macrostimulation to refine ultimate electrode position. Microelectrode recording carries a risk of hemorrhage and requires active patient participation during surgery. Using high-field MRI, we sought to enhance direct targeting for DBS surgery, with the ultimate goal of improving the accuracy of anatomic target selection, thereby improving clinical efficacy of stimulation.

TECHNIQUE DEVELOPMENT: Using a 7-Tesla MRI scanner combined with an array of acquisition schemes employing multiple image contrasts, we obtained high-resolution images of human deep nuclei in healthy subjects. We then imaged a stereotactic head frame and fiducial localizer box in the 7T scanner to check for image distortion.

RESULTS: Superior image resolution and contrast obtained at 7T in vivo, using susceptibility-weighted imaging, dramatically improved anatomical delineation of DBS targets and allowed for identification of internal architecture within these targets. A patient-specific, 3-D model of each target area was generated based on the acquired images.

CONCLUSION: Technical developments in MRI at 7T have yielded improved anatomical resolution of deep brain structures, thereby holding the promise of improving direct targeting for DBS surgery. Future study is ongoing to validate this technique in improving accuracy of direct targeting in DBS surgery.

THURSDAY, NOVEMBER 5

9:49 – 10:01 AM **TUBERCULUM SELLAE MENINGIOMAS: SURGICAL APPROACHES AND MODERN DAY RESULTS**

Michael W. McDermott, M.D., Nader Sanai, M.D., Catherine Christine M.D., Kenny Chung, B.Sc., Charles B. Wilson, M.D., Sandeep Kunwar, M.D., Mitchel S. Berger, M.D.

INTRODUCTION: A variety of surgical approaches have been described for the surgical removal of tuberculum sellae meningiomas. Options for the surgical approach include transcranial and transnasal routes with a number of variations related to skin incisions and the use of the microscope versus the endoscope. We reviewed our current experience with surgery for removal of these tumors since 1992 to serve as a baseline for comparisons to newer evolving techniques.
METHODS: The department database was reviewed for cases of tuberculum meningioma operated between September 1992 and December 2008. Clinical charts and operative records were reviewed to determine the population demographics, symptoms prior to surgery, imaging features, surgical approach, Simpson extent of resection, visual outcome and complication profiles.
RESULTS: There were 69 cases, 16 males and 53 females. Median age at time of surgery was 54 yrs. (40-63 yrs.). The median time from diagnosis to surgery was 4 months (1-12.5 mo.). Pre-operative visual symptoms were present in 87% of patients. None had prior history of seizures. The median maximum tumor dimension was 2.35 cm (1.8 – 2.9 cm.) and the median tumor volumes were 7.68 cc. (2.25-18 cc.). A bifrontal extended frontal craniotomy was used in 55% of cases, a unilateral approach in 38%, a transsphenoidal approach in 4%, a bifrontal approach 3% in and a fully endoscopic approach in 0%. Pathology revealed WHO Grade 1 or 2 in 71% and 29%, respectively. Simpson extent of resection was classified as 1,2,3,4 or 5 in 47%, 28%, 2%, 21% and 2%, respectively. Median post-op clinical and radiographic follow-ups were 20 and 19 months. There was no operative 30 day mortality. Overall morbidity was 11%. Three patients had loss of vision post-operatively, 2 unilaterally and 1 bilaterally. CSF leak occurred in 2 patients post-operatively (3%).
CONCLUSIONS: Transcranial approaches for tuberculum sellae meningiomas can yield good visual and surgical results with an acceptable complication profile. Even the more extensive skull base approaches seem to be well tolerated in the approach to larger tumors in this region and the CSF leak rate is low. An update for this series and discussion of the surgical approaches will be presented.

THURSDAY, NOVEMBER 5

10:23 – 10:35 AM **3-D ENDOSCOPIC PITUITARY SURGERY- A DISRUPTIVE INNOVATION PRECIPITATES THE TIPPING POINT**

Nelson M Oyesiku, MD, PhD, FACS

INTRODUCTION: Endoscopic transsphenoidal pituitary surgery has re-emerged as a viable technical procedure. The advantages proffered include the wider angle of view. That asset has been impacted by the diminished stereopsis with conventional 2-D endoscopes, attenuating an evolutionary advantage in favor of the surgeon operating with native vision or the microscope. Diminished stereopsis can impair the perception of depth, size or shape. These critical judgments may result in significant errors when operating deep in the brain. Stereo (3-D) endoscopes restore this precise optical advantage while maintaining the wider field of view. Previously, their use was limited by the wider bore required and lackluster image resolution. Recently, an innovative 3-dimensional (3-D) stereo endoscope with a "compound eye" technology consisting of a microarray of lenses has emerged that incorporates the best of both worlds.
METHODS: Thirty patients underwent endonasal endoscopic transsphenoidal surgery at Emory University all by a the same team of one neurosurgeon and one ENT surgeon using 4.0-mm, 0- and 30-degree rigid 3-D stereoendoscopes (Visionsense, Ltd., Petach Tikva, Israel). Surgeon estimation of facility, safety, ease of resection, visualization and operating time were documented. A comparison of the 3-D endoscope with the 2-D endoscope and microscope was performed during the procedure. A variety of sella and suprasellar tumors were operated on including functional microadenomas and macroadenomas, non-functional macroadenomas, Rathke's cleft cysts, and craniopharyngiomas.
RESULTS: The 3-D endoscope was used as the only tool in 25 of the 30 cases, 5 other cases included some element of the 2-D endoscope or microscope for comparison. There were no intraoperative misadventures. All patients had gross tumor resections. Operative times, length of stay were not different from the usual experience in pituitary surgery at our institution. Stereopsis was vastly improved compared with 2-D endoscopes.
Conclusion: In this series of 30 patients using the 3-D transsphenoidal endoscope for pituitary surgery, we were convinced that improved stereopsis and the wider field of view afforded was instrumental in improved facility, superb access, excellent outcomes with facilitated operative time. 3-D endoscopes may be the critical disruptive technology that results in the final tipping point in favor of endoscopic pituitary surgery.

THURSDAY, NOVEMBER 5

10:37 – 10:49 AM **AUTOPSY AND CLINICAL ANALYSIS OF MODERN DAY CONTACT SPORT ATHLETES**

Julian E. Bailes, M.D., Bennet Omalu, M.D.

INTRODUCTION: Recent clinical and research data indicate that repetitive mild traumatic brain injury (MTBI) may lead to cumulative and long term brain damage. In those situations, most commonly contact sports but increasingly now recognized in soldiers exposed to blast injuries, a threshold likely exists whereby chronic changes in the brain's metabolism and ultrastructure may lead to subsequent clinical manifestations later in life.
While originally described in boxers, chronic traumatic encephalopathy (CTE) has now been identified in modern day athletes using specific immunohistochemical staining techniques.

METHODS: Using available medical records and athletic career, psychiatric, behavioral, and drug use information obtained from family members, we reconstructed the decedents' past history. With staining methods to document tau protein accumulation, postmortem analysis through brain autopsy has been performed in 17 contemporary athletes: 8 were NFL players, 4 professional wrestlers, 3 high school football players, and one boxer and one mixed martial artist. They were all males ranging in age from 17 to 52 years of age, with suicide the predominant cause of death.

RESULTS: Available data regarding concussion history and substance abuse suggest in many cases that multi-factorial insults may exist. The involved areas also suggest a predilection for the frontal, temporal, and limbic lobes, perhaps explaining the similar behavioral and emotional disturbances producing a near identical psychiatric expression. In every case, abnormal tau protein was distributed in a fashion suggesting diffuse and multiple sites and times of injury. There were no instances of gross brain abnormality, or findings typically seen in acute trauma such as cerebral contusion. . Genetic testing also demonstrated the unexpected predominance of the apoE3 homozygous condition. In addition, a new brainstem predominant form will be described.

CONCLUSIONS: Contact sports such as football, soccer, ice hockey, boxing, the mixed martial arts, and wrestling all share a propensity for repetitive head collisions. While attempts have been made to reduce brain injury through protective helmets and rules changes, there nonetheless exists the possibility for repetitive brain injury and the syndrome of CTE in all of these sports. This unprecedented examination using tau protein staining has shed new light on our understanding of the phenomenon of behavioral neuropsychiatric disturbances with anatomical correlation. The recognition that a multi-factorial risk exposure-based prospect exists for a small but important minority of contact sport athletes will assist in proper management of the concussed athlete. The detection of injury, the stratification of risk, thresholds for additive effects of concussion, and strategies for mitigation of the potential for permanent brain damage will be discussed.

THURSDAY, NOVEMBER 5

10:53 – 11:03 AM **FOLIC ACID SUPPLEMENTATION ENHANCES CNS REGENERATION *IN VITRO***

Bermans J. Iskandar, M.D., Krista Stewart, BS, Brenton Meier, M.D., Elizas Rizk, M.D., Nithya Hariharan, M.D.

INTRODUCTION: We have previously shown that folic acid supplementation significantly enhances regeneration of the injured CNS *in vivo.* To determine whether folate acts at the level of the neurons or glia, we studied the behavior of folic acid in an *in vitro* model of neuronal regeneration.

METHODS: Sprague-Dawley rats were subjected to bilateral C3 dorsal column transections. Half the rats were pretreated with intraperitoneal folic acid (80ug/kg), the dose optimal for spinal regeneration *in vivo.* The lumbar DRGs, which contain the cell bodies corresponding to the injured spinal axons, were removed 3 days postoperatively, dissociated, then placed in a culture medium permissive for growth. Neurons were fixed/plated at time intervals 5-48 hours after culture initiation. Axonal elongation was assessed blindly using ImageJ software.

RESULTS: 1) Spinal cord injury suppressed the ability of DRG neurons to grow long axons in a 72-hour culture. 2) However, when the animals were pretreated with folic acid, these neurons produced a significantly greater percentage of long axons (>300µm) than those in the untreated group (22% compared to 7%, $p<0.05$). In addition, 3) growth of folate-treated neurons occurred 20 hours earlier than the controls. At all time points in culture, folate-treated neurons exhibited longer axons.

DISCUSSION: Folic acid enhances CNS regeneration through a direct effect on the neuron, seemingly reversing the deleterious effect of a spinal cord injury. A concomitant effect of folic acid on the inhibitory glia cannot be ruled out. Nonetheless, the neuronal effect is very significant and might impact not just our understanding of repair processes in the mature CNS, but also the growth/developmental mechanisms that lead to congenital defects such as spina bifida. This could lead to novel treatment and prevention strategies for patients with degenerative as well as developmental CNS problems.

THURSDAY, NOVEMBER 5

11:05 – 11:17 AM **PLATELET DERIVED GROWTH FACTOR-**

BB MEDIATES THE TROPISMS OF BONE

MARROW MESENCHYMAL STEM CELLS FOR HUMAN GLIOMAS

Naoki Shinojima, M.D., Nobuhiro Hata, M.D., ***Frederick F. Lang, M.D.***

OBJECTIVE: Bone marrow mesenchymal stem cells (BM-MSCs) are capable of localizing to gliomas after systemic delivery, and can be used to carry therapeutic agents to brain tumors. Endogenous BM-MSCs may also have an inherent tropism for brain tumors and may contribute to the biology of gliomas in situ. Despite the increasing importance of BM-MSCs in glioma therapy and biology, the mechanism underlying the tropism of BM-MSCs for gliomas remains unclear. *In vitro* studies have suggested that tumor-secreted growth factors, particularly platelet derived growth factor (PDGF-BB), may mediate the tropism between BM-MSCs and gliomas. However, a causal role of PDGF-BB has not been demonstrated heretofore. Consequently, we tested the hypothesis that PDGF-BB mediates the attraction of BM-MSCs for gliomas.

METHODS & RESULTS: U87 or LN229 cells were transfected with plasmids encoding human PDGF-B. Stable transfected clones that secreted large amounts of PDGF-BB (e.g. U87-PDGF-high, mean PDGF 23 $x10^2$ pg/ml) and clones that produced low levels of PDGF (e.g. U87-PDGF-low, mean PDGF 0.26 $xl0^2$ pg/ml) were chosen. *In vitro* Matrigel invasion assays showed that significantly more BM-MSCs migrated toward the U87-PDGF-high clones (86±1 MSCs/10hpf) compared with U87-PDGF-low clones (40±2 hMSCs/10hpf) ($p<0.006$). Similar results were obtained for high and low PDGF-secreting LN229 clones. Treatment of the conditioned media with anti-PDGF-BB-neutralizing antibody abrogated the increase in migration of BM-MSCs toward PDGF-BB-high secreting clones. Likewise, pretreatment of BM-MSCs with inhibitory antibodies against PDGF-receptor-β (PDGF-β), which are located on BM-MSCs, reduced BM-MSC migration. To demonstrate that PDGF-BB mediates the localization of hMSCs *in vivo*, bioluminescence imaging (BLI) of BM-MSCs was undertaken. Intracranial xenografts of U87-PDGF-high (N=9) or U87-PDGF-low (N=9) cells were established in the frontal lobes of nude mice. Seven days after tumor inoculation, BM-MSCs were transduced with an adenovirus vector containing firefly luciferase gene, and injected (10^6 cells) into the carotid artery of tumor-bearing mice. Animals were treated with Luciferin (150 mg/kg, IP) and imaged with the IVIS Imaging System (Xenogen, CA). The average signal intensity of U87-PDGF-high tumors (614±46 photons x 10^3/sec/cm^2) was significantly greater than that of the U87-PDGF-low tumors (87±13 photons $x10^3$/sec/cm^2, $P<0.0002$). To verify this result and to control for PDGF-BB-induced differences in tumor size and vascularity, BM-MSCs were transfected with green fluorescent protein and injected into the carotid arteries of animals harboring 4-day old PDGF-BB-high secreting xenografts (N=3) or 7-day old PDGF-BB-low secreting xenografts (N=4). At these times tumors were similar in size and vessel density. Statistically significant more hMSCs localized to PDGF-BB-high secreting xenografts (24.8+/-6.3 MSCs/mm^2) compared with PDGF-BB-low secreting xenografts (5.0+/-1.8 MSCs/mm^2). Pretreatment of hMSCs with anti-PDGFR-β-inhibitory antibodies significantly decreased the localization of BM-MSCs in this intracranial model.

CONCLUSION: Secretion of PDGF-BB by human gliomas and activation of PDGF-R-β on hMSCs mediates the tropism of BM-MSCs for human gliomas. These finding can be exploited for advancing MSC-based treatment and suggest new targets for altering endogenous BM-MSC homing to gliomas.

THURSDAY, NOVEMBER 5

11:19 – 11:31 AM **INFECTION RATE FOLLOWING MINIMALLY INVASIVE SPINE SURGERY**

Richard G. Fessler, M.D., Ph.D., John O'Toole, M.D., Kurt Eichholz, M.D.

INTRODUCTION: Reported rates of wound infection after traditional open spinal surgery range from 0.9% after routine discectomy to greater than 15% after arthrodesis with instrumentation. In the latter cases, these infections can be troubling to treat and, in general, augment the utilization of health care resources significantly. The techniques used in minimally invasive spine (MIS) surgery confer many benefits, but the incidence of postoperative wound infections when using these approaches has not been specifically studied in the literature.
METHODS: A retrospective review was performed of a single surgeon's prospectively collected database of MIS operations over a 6 year period, and the incidence of postoperative wound infection was calculated. The rates of infection for the different types of procedures performed were also analyzed.
RESULTS: A consecutive series of 884 MIS operations were performed on 857 patients with a mean age of 54.8 years and a male to female ratio of 1.4:1. All patients received a single dose of perioperative prophylactic antibiotics. Minimum follow-up was 12 weeks and the mean was 32.5 weeks. The cohort demonstrated only 2 postoperative infections for a 0.2% infection rate. One of the infections occurred after a lumbar microendoscopic decompression of stenosis (MEDS) in a patient with an undetected preoperative urinary tract infection and postoperative urosepsis. The other infection developed after minimally invasive transforaminal lumbar interbody fusion (MI TLIF) and percutaneous instrumentation. Both patients were successfully treated with antibiotic therapy without re-operation or removal of instrumentation. The procedure-specific infection rates, therefore, were 0.7% for MEDS, 0.8% for MI TLIF, and 0% for all other procedures.
CONCLUSION: This series of MIS operations using tubular retractors demonstrates an incidence of postoperative infection that is demonstrably less than that published in the literature for traditional open spinal surgery. The diminished rate of wound infection is particularly notable for the instrumented cases. It is our hypothesis that this reduced incidence stems from 1) the reduced exposure of deep tissues during MIS surgery, 2) the physical barrier to operative instrument contact with patients' skin provided by tubular retractors, and 3) the markedly reduced amount of postoperative dead space after MIS surgery. Future studies will be useful to determine if MIS techniques are preferable in patients at high risk for postoperative wound infections.

THURSDAY, NOVEMBER 5

11:33 - 11:45 AM **RELATIVE ANGLE OF DEFLECTION CORRELATES WITH ANEURYSMAL RUPTURE STATUS IN POSTERIOR COMMUNICATING ARTERY ANEURYSMS**

Charles J. Prestigiacomo, MD, FACS, John Quinn, MD, Wenzhuan He, MD, Chirag D Gandhi, MD

INTRODUCTION: Mathematical modeling of aneurysms is critical in gaining understanding into the biophysical phenomena that contribute to aneurysm growth and rupture. Prior studies have demonstrated that certain primary and secondary biomorphometric characteristics correlate with aneurysm rupture. Aneurysms of the posterior communicating artery (PCoA) have a high propensity for rupture. This study analyzes the predictive value of these previously described parameters and defines other significant parameters for predicting PCoA aneurysm rupture through analysis of the angular relationships between aneurysm and the related vessels. By performing binary logistic regression analysis, we have derived a relational equation that describes the rupture potential for PCoA aneurysms within this cohort.

METHODS: Patients presenting to our institution with ruptured and unruptured aneurysms were evaluated with CT angiography (CTA) using a GE systems 16-slice CT scanner. Maximum Intensity Projections (MIP) was generated and measurements were made in planes orthogonal to primary blood flow vectors. Aneurysm height and vessel diameters, derived measurements were obtained to include the aneurysm's angle of deflection relative to primary flow vector, the aneurysm's angle relative to the parent and daughter vessels, the angle of the posterior communicating artery origin relative to the internal carotid artery. Investigators involved in determination of all calculations were blinded to the aneurysm's rupture status. Independent t-test was used to assess the differences in mean.

RESULTS: A total of 78 patients with aneurysms of the posterior communicating artery were evaluated: 21 presented as unruptured and 57 presented as ruptured. Of the twelve primary and secondary criteria evaluated, aneurysm height ($p=0.017$), aneurysm neck ($p=0.009$), the deflection of the aneurysm neck from the primary flow vector ($p=0.03$), the angle of the primary vector of the aneurysm relative to the primary blood flow vector ($p=0.005$) and the aspect ratio ($p=0.03$), were statistically correlated to aneurysm rupture status. Preliminary analysis by binary logistic regression analysis suggests a sensitivity of 82% an overall accuracy of 80% in predicting PCoA aneurysm rupture in this cohort.

CONCLUSION: This binary logistic regression model of PCoA aneurysm represents an accurate means of predicting rupture status through measurement of simple biomorphometric characteristics.

THURSDAY, NOVEMBER 5

11:47 - 11:59 AM **MAGNETIC VORTICES AS MEDIATORS OF CELLULAR MECHANO-TRANSDUCTION AND CELL DEATH IN MALIGNANT GLIOMA**

Dong-Hyun Kim, PhD, Elena A. Rozhkova, PhD, Ilya V. Ulasov, PhD, Samuel D. Bader, PhD, Tijana Raj, PhD, Valentyn Novosad, PhD, and ***Maciej S. Lesniak, MD***

We report proof-of-concept of targeted magneto-mechanical cancer cell destruction using disk-shaped magnetic nanoparticles that possess a spin vortex ground state. Gold-coated microdisks with an Fe-Ni magnetic core were fabricated using conventional lithography and thin-film deposition techniques and then biofunctionalized with anti-human-IL13α2R antibody for specifically targeting of human glioblastoma cells. When an alternating magnetic field is applied, the magnetic disks oscillate, which causes a magneto-mechanic stimulus to be transmitted to the cell and subcellular components. Cytotoxicity assays, along with comprehensive cells morphology studies, demonstrate that a magnetic field of <100 Oe amplitude and frequency of a few 10's of Hz applied during 10 min time span was sufficient to achieve nearly complete cancer cell destruction. Terminal dUTP nick-end labeling assay revealed significant nuclear DNA damage. A strong cytotoxic effect can be understood as a combined result of primary alteration of cellular membrane integrity, as well as the transduction and amplification of a magnetic vortex-induced mechanical stimulus into a pronounced cellular signal, namely the initiation of programmed glioma cell death.

THURSDAY, NOVEMBER 5

12:01 – 12:13 PM **STENT -ASSISTED COILING OF PARACLINOID ANEURYSMS DOES NOT INCREASE RISK OF TREATMENT**

Christopher S. Ogilvy, M.D., Xinyu Yang, M.D., Ph.D., Kenneth V. Snyder, M.D., Ph.D., L. Nelson Hopkins, M.D., Adnan H. Siddiqui, M.D., Ph.D., Elad I. Levy, M.D.

OBJECTIVES: Stent assistance for the treatment of wide-based aneurysms is becoming rapidly accepted. The purpose of the present study was to review our experience with .the use of stent-assisted coiling for aneurysms that arise in the paraclinoid location of the internal carotid artery.

METHODS: We retrospectively analyzed cases of paraclinoid aneurysms treated with intracranial stents and/or bare platinum coils between September 2001 and December 2008 from a database that was prospectively gathered. We identified 71 patients treated with stenting and coiling *(one case of stenting alone)* of broad-necked, large or giant, or recanalized paraclinoid aneurysms and also 24 patients treated with coiling alone of narrow-necked paraclinoid aneurysms. Among the 71 patients, stenting and coiling of paraclinoid aneurysms was achieved either as a one-time treatment or as a two-step maneuver with the stent being placed several weeks prior to coiling. In a third group of patients, stent assisted coiling was used as a second maneuver in aneurysms that had recanalized after previous coiling.

RESULTS: In 71 patients treated with stenting and coiling, 88.7% had >95% aneurysm occlusion at completion of the treatment, a result that compared favorably with the 79.2% rate of >95% occlusion for patients treated with coiling alone. At last follow-up recorded, 58 patients treated with stenting and coiling had an 84.5% incidence of >95% obliteration; 83.3% of patients treated with coiling alone had aneurysm obliteration >95% (not significant, $P > 0.05$). In-stent stenosis was not observed in patients treated with stent-assisted coiling. Thrombus occurred during stent deployment in one patient (1.4%) without neurologic sequelae, and stent displacement occurred in one patient (1.4%) with neurologic sequelae. Among patients treated with stent-assisted coiling, overall clinical outcome was good or excellent (modified Rankin scale ≤2) in 52 of 54 (96.2%) patients, compared with 94.4% modified Rankin scale ≤2 in patients treated with coiling alone.

CONCLUSION: We found that stent-assisted coiling of paraclinoid aneurysms did not add significantly to morbidity, and overall effectiveness was at least equal to that of bare coiling of narrow-necked aneurysms.

THURSDAY, NOVEMBER 5

12:15 – 12:27 PM **ENDOGENOUS PROTECTIVE MECHANISMS AGAINST CEREBRAL VASOSPASM**

Gregory Zipfel, MD, FAHA; Eric Milner, BS; Charis Gn; Jeffrey Gidday, PhD; Byung Hee Han, PhD

INTRODUCTION: Vasospasm is the most common cause of secondary neurological injury in patients with aneurysmal subarachnoid hemorrhage (SAH). Yet no significant advances in the pharmacological prophylaxis of vasospasm have occurred since the 1980's. Recent evidence demonstrates that potent endogenous protective mechanisms against vessel injury and dysfunction exist. This is best documented in the setting of cerebral preconditioning (PC) where the brain's inherent resistance to injury can be augmented by prior exposure to a sublethal PC stimulus such as hypoxia. Following PC, several adaptive vascular responses are noted including improved vascular endothelial cell resistance to injury, augmented endothelium-dependent vasodilation after ischemia, and less severe blood flow reduction after ischemia. In this proof-of-concept study, we sought to determine whether endogenous vascular protective mechanisms (induced via hypoxic PC) attenuate vasospasm and improve neurological outcome following SAH; and if so, whether the observed neurovascular protection is mediated via endothelial nitric oxide synthase (eNOS).

METHODS: First, adult male C57BL/6 mice were subjected to hypoxic PC followed 24 hr later by SAH (Hypoxia: SAH), normoxia followed 24 hr later by SAH (Normoxia: SAH), or normoxia followed 24 hr later by sham operation (Normoxia: Sham). Vasospasm and neurological deficits were then assessed. Second, adult male C57BL/6 mice underwent sham operation, SAH, or hypoxic PC and sacrificed 24 hr later for immunohistochemical analysis of arterial eNOS. Third, adult male eNOS knockout mice and control C57BL/6 mice underwent hypoxic PC followed 24 hr later by SAH. Vasospasm and neurological deficits were then assessed. In all experiments, hypoxic PC was induced via exposure to 8% O_2 for 4 hr, SAH was induced via endovascular perforation, neurological deficits were assessed by rotarod test and sensorimotor neuroscore, and vasospasm was assessed via post-mortem middle cerebral artery (MCA) diameter measurements.

RESULTS: SAH causes marked vasospasm (average MCA diameter = 70 ± 5 μm in Normoxia: SAH mice vs. 104 ± 5 μm in Normoxia: Sham mice; $p<0.05$) and significant neurological deficits. Strikingly, hypoxic PC completely prevents vasospasm (average MCA diameter = 102 ± 4 in Hypoxia: SAH mice vs. 70 ± 5 μm in Normoxia: SAH mice; $p<0.001$) and markedly improves rotarod performance and neuroscore following SAH ($p<0.01$ and $p<0.05$, respectively). These data indicate that endogenous protection against vasospasm exists and can be induced via PC stimuli. We next examined the effect of hypoxic PC on eNOS expression. As expected, SAH reduces eNOS expression in cerebral arteries. Interestingly, hypoxic PC markedly increases vascular eNOS expression, raising the possibility that hypoxic PC attenuates SAH-induced vasospasm via its effect on eNOS expression. To directly examine this possibility, we examined the vasospasm protection afforded by hypoxic PC in eNOS knockout vs. control C57BL/6 mice. As noted previously, hypoxic PC almost completely prevents SAH-induced vasospasm in C57BL/6 mice. In eNOS knockout mice, however, hypoxic PC provides substantially less vasospasm protection (average MCA diameter = 97 ± 4 in C57BL/6 mice vs. 80 ± 5 μm in eNOS knockout mice; $p=0.0153$). Neurological deficits in eNOS vs. control mice were not significantly different.

CONCLUSIONS: Our results indicate that hypoxic PC induces powerful endogenous protection against SAH-induced vasospasm, and that this protection is mediated, at least in part, via eNOS. Augmentation of endogenous vascular protective pathways via PC stimuli may be a new strategy towards preventing or reducing SAH-induced vasospasm. Future studies are needed to investigate clinically relevant PC strategies and to further elucidate the underlying molecular mechanisms.

FRIDAY, NOVEMBER 6

7:30 – 7:42 AM

A MULTI-CENTER PROSPECTIVE CONTROLLED STUDY ASSESSING THE EFFICACY OF SURGERY IN PATIENTS WITH DIFFERING SEVERITY OF CERVICAL SPONDYLOTIC MYELOPATHY: RESULTS OF THE AOSPINE NORTH AMERICA CSM STUDY IN 300 SUBJECTS

Michael G. Fehlings MD PhD FRCSC FACS, University of Toronto; Branko Kopjar MD PhD; Tim Yoon MD PhD; Paul Arnold MD; Eric Massicotte MD MSc; Alexander Vaccaro MD PhD; Jens Chapman MD; Darrel Brodke MD; Christopher Shaffrey MD; Eric Woodard MD; Michael Janssen DO

INTRODUCTION: Cervical Spondylotic Myelopathy (CSM) is a degenerative disease resulting in compression of the cervical spinal cord or nerve roots, leading to neurological dysfunction. Standard practice reserves surgical intervention for severe CSM, but treatment for mild or moderate disease remains controversial. Given this controversy, we performed a multi-center prospective, non-randomized controlled study to assess the efficacy of operative decompression in patients with varying severity of CSM.

METHODS: A total of 300 subjects with clinically and radiographically confirmed CSM were enrolled at 13 sites in the USA and Canada. Subjects were treated by an anterior or posterior decompressive/reconstructive approach. Outcomes assessments included the modified Japanese Orthopedic Association (mJOA) scale, Neck Disability Index (NDI), Nurick score, Short Form 36 (SF-36v2) and a review of adverse events. Subjects were classified as having mild (mJOA $\geq$ 15), moderate (mJOA 12-14) or severe CSM (mJOA < 12) based on baseline mJOA scores. Fourteen subjects withdrew from the study prior to the 12 month follow-up. Twelve month follow-up data were available for 234 (82.4%) out of 286 subjects. Data for 17.6% subjects with missing 12 month follow-up were derived using multiple imputation techniques.

RESULTS: The mJOA, NDI, Nurick, and SF-36v2MCS scores improved significantly in all severity groups. Patients with severe CSM, however, did not show significant improvement in SF-36v2PCS scores. The extent of relative improvement in NDI, Nurick, SF-36v2PCS/MCS was similar across all severity groups. The extent of improvement in the mJOA scores was associated with the severity of myelopathic impairment. Nine patients (3%) experienced neurological complications, the most common of which were delayed, transient C5 palsies. Only one patient (0.3%) experienced a decline in spinal cord function perioperatively.

CONCLUSION: Surgical treatment for CSM is effective in improving neurological impairment, function, and quality of life for patients with all levels of disease severity. While all groups showed improvement with surgical intervention, patients with moderate and severe CSM retained substantial residual impairment, emphasizing a need for earlier diagnosis and intervention for this common treatable cause of neurological dysfunction**.**

FRIDAY, NOVEMBER 6

7:44 – 7:56 PM **NOVEL GENES FOR MOYAMOYA DISEASE**

Gary Steinberg, MD, PhD, Teresa Bell-Stephens, RN, Monika Varga, BS, Maria Coburn, BS, Hariyadarshi Pannu, PhD, Steven Scherer, Limin Gong, Ellen Regalado, MS, Zhao Ren, Carlos Villamizar, Teresa Kuncewicz, Nili Avidan, Jay Johnson, Dongchuan Guo, Dianna Milewicz, MD, PhD

Moyamoya disease (MMD) is a progressive occlusive angiopathy involving the Circle of Willis arteries. While the etiology of MMD is unclear, approximately 10% of cases are familial and prior linkage studies have identified candidate chromosomal regions on 3p24.2-p26, 8q23 and 17q25. However, until recently, single genes have not been identified that contribute to MMD.

From 1991-2009 we treated 396 MMD patients at Stanford University Medical Center. We extracted genomic DNA from peripheral blood of 183 MMD patients and compared it to DNA from 188 controls. We found 1 MMD patient (0.5%) with a unique mutation of *ACTA2* that regulates expression of the specific isoform of α-actin, a major component of the contractile apparatus in vascular smooth muscle within arteries. *ACTA2* mutations lead to increased smooth muscle cell and myofibroblast proliferation *in vivo* and *in vitro*. Seven additional unrelated MMD patients (4%) had another unique single gene mutation "*MMD2*" that alters arterial wall components. Analysis of DNA from family members demonstrated that both novel MMD gene mutations increase the risk of early onset coronary artery disease, early non-MMD stroke and thoracic aortic aneurysms and dissections, in addition to MMD. Both genes have a high penetrance that increases the risk of vascular disease by 80-90%.

Novel gene mutations in *ACTA2* and "*MMD2*" predispose to developing MMD and a range of other vascular arterial diseases.

FRIDAY, NOVEMBER 6

7:58 – 8:10 AM **CHEMOKINE SECRETING DENDRITIC CELLS FOR IMMUNOTHERAPY OF MALIGNANT GLIOMA**

Andrew E. Sloan, M.D., FACS, Robert Miller, Ph.D., Jill Barnholtz-Sloan, Ph.D., John Lowe, M.D., James J. Mule' Ph.D.

INTRODUCTION/HYPOTHESIS: Despite three decades of intense research, prognosis for patients with malignant gliomas remains poor. Clinical studies suggest that various types of immunotherapy effectively induce a systemic anti-tumor immune response. However, clinical efficacy appears to be restricted to selected patients with minimal tumor burden and rarely results in cure. Resistance to immunotherapy has been attributed to failure of failure of antigen presentation in the brain as well as the presence of an immunosuppressive microenvironment characterized by the presence of regulatory T-lymphocytes (T_{reg}) in the blood and tumor, as well as glioma infiltrating microglia/macrophages (GIM) and NKT cells. Recent studies have demonstrated that secondary lymphoid chemokine (SLC) may mediate co-migration of antigen presenting cells and naïve lymphocytes to stimulate extra-nodal-priming of the immune response. We **hypothesized** that dendritic cells (DC) transduced with SLC would improve anti-tumor immune response, ameliorate the immunosuppressive microenvironment within the brain, and improve survival in a pre-clinical model.

METHODS: C57/BL6 mice bearing 7 day orthotopic, syngeneic GL261 gliomas were vaccinated with subcutaneous (sq), or intra-tumoral (it) PBS, DC, DC transduced with full-length SLC or a non-functional truncated SLC (SLC_T) using adenoviral vector ; and pulsed or unpulsed with tumor lysate. Twelve animals per group were treated and survival was quantitated by Kaplan Meier methodology. SLC secretion was assessed using ELISA and a microchemotaxis assay. The presence of various subpopulations of immune effector cells in the tumor, contralateral brain, blood, spleen, ipsilateral and contralateral lymph nodes was quantitated by multiparameter flow cytometry and analyzed using Wilcoxon test.

RESULTS: DC transduced with full-length SLC secreted median 867 ng SLC /10^6 DC/day. This induced 3.5 - 4-fold increase of $CD3^+$T-lymphocytes by microchemotaxis assay. Vaccination with DC-SCL-Lys (sq) improved survival compared to all other s.q. treatments ($p < 0.01$) with median survival of 58 days and 30% of mice surviving more than 100 days. Intra-tumoral treatment with DC-SLC-Lys resulted in improved survival compared to all other it or sq vaccines with median survival of 71 days and 42% surviving more than 100 days. Animals treated with it DC-SLC-Lys demonstrated increased intra-tumoral T_h, NK, and DC infiltration ($p < 0.01$); and increased T_h, and T_c in ipsilateral lymph nodes ($p < 0.02$); as well as decreased NKT, $Gr1^+$ granulocytes, and GIM infiltrating the tumor ($p <0.01$); and decreased T_{reg} in peripheral blood and ipsilateral lymph node ($p <0.01$).

CONCLUSIONS: Vaccination with dendritic cells secreting secondary lymphoid chemokine significantly improved survival in an orthotopic syngeneic preclinical model of malignant glioma. Intratumoral treatment is more effective than subcutaneous vaccine and preliminary studies suggest that this treatment appears to modify the immunosuppressive milieu within the tumor microenvironment. Future studies will assess the role of various co-stimulatory molecules and cytokines in as well as the presence of immunological memory in this model. Translational implications will be discussed.

FRIDAY, NOVEMBER 6

8:12 – 8:24 AM **MICROSURGICAL RESECTION OF ANTERIOR SKULL BASE MENINGIOMAS**

Anil Nanda, M.D., FACS, Vijayakumar Javalkar, M.D.

OBJECTIVE: The objective of this study was to analyze our experience with microsurgical resection of anterior cranial base meningiomas. Specifically, we evaluated our series of 92 such cases with special emphasis placed on surgical outcomes, complications and tumor recurrence.

MATERIAL AND METHODS: We retrospectively reviewed the medical records of these 92 patients who underwent surgical excision of anterior skull base meningiomas from 1990 to 2009.

RESULTS: From 1990-2009, 92 patients underwent microsurgical resection of an anterior skull base meningiomas. Fifty-five percent (55%) of the tumors were large (>5 cm). Majority of the patients were female (67%). Mean age was 57 years. Majority of the tumors were based along the olfactory groove. The three most common surgical approaches were frontotemporal orbitozygomatic (FTOZ) craniotomy, pterional craniotomy and bifrontal craniotomy. Eighty-three (90%) patients underwent gross total excision. Nine patients had residual tumor. Mean follow-up was 19 months. At the time of discharge, 94.5% of the patients had a Glasgow Outcome Scale rating of 5. Visual improvement was noted in 12 patients out of 42 who presented with visual compromise. Of 3 patients who developed cerebrospinal fluid (CSF) leaks, 2 had rhinorrhea and 1 had leakage through the incision. Two patients developed cranial neuropathies. Postoperative endocrinopathies were seen in 7 patients. Hydrocephalus developed in 6 patients, 5 of whom required a ventriculoperitoneal shunt. Five patients developed tumor recurrence after surgical resection (9%). One patient died due to surgery related cause (1.3%).

CONCLUSION: Anterior cranial base meningiomas can be safely excised with minimal morbidity and mortality by microsurgical technique. Addition of skull base modifications to standard craniotomy provided improved exposure without increasing morbidity.

FRIDAY, NOVEMBER 6

8:26 – 8:38 AM **PROSPECTIVE ANALYSIS OF OUTCOMES AFTER CT PERFUSION-GUIDED STROKE INTERVENTION**

Elad I. Levy MD FACS, Sabareesh K. Natarajan MD MS, Yuval Karmon MD, Hajime Ohta MD PhD, Kenneth V. Snyder MD PhD, Adnan H. Siddiqui MD PhD, and L. Nelson Hopkins MD FACS

AIM: To determine the recanalization rate, symptomatic intracranial hemorrhage (SICH) rate, and outcomes 3 months after discharge of patients undergoing CT perfusion-based selection and endovascular revascularization for acute ischemic stroke at our center from between 2006 and 2008.
METHODS: Patients with acute ischemic stroke who were not candidates for intravenous thrombolysis (IVT) or failed to improve neurologically after IVT with a confirmed accessible vascular occlusion by CT angiography were selected for endovascular pharmacologic and/or mechanical thrombolysis based on CT perfusion imaging. Data collected prospectively included patient demographics, treatment characteristics, immediate and 3-month outcomes, and SICH rates. Differences in outcomes between subgroups based on time-to-thrombolysis, location of occlusion, and type of thrombolysis were analyzed by Chi-Square test using SPSS software.
RESULTS: A total of 193 patients (23 with wake-up strokes and 170 with known stroke symptom onset) with mean presentation National Institutes of Health Stroke Scale (NIHSS) score of 13.9±5.4 (median–14) were included. The average time from stroke symptom onset to initiation of thrombolysis was 345±336 minutes (median–249 minutes, range 34–1126 minutes) (treatment within 0–3 hours, n=60; 3–6 hours, n=80; >8 hours, n=30). The number of patients treated for complete-to-near-complete vessel occlusion (TIMI 0/1) and occlusion locations included anterior circulation–169, posterior circulation–24; tandem extracranial and intracranial–23, extracranial supraaortic vessels–34, petrous internal carotid artery (ICA)–9, ICA-terminus–29, anterior cerebral artery (ACA)–16, and middle cerebral artery (MCA) proximal M1–44, distal M1–59, and M2–67. The number of patients and thrombolytic therapies included mechanical thrombolysis only–94, pharmacologic thrombolysis only–24, and both–75; intravenous thrombolysis–21, intraarterial thrombolysis–98, Merci device–108, intracranial stent–52, extracranial stent–36, intracranial angioplasty–55, extracranial angioplasty–24, Penumbra device–4. Fifty-five patients received intraarterial glycoprotein (GP) IIb/IIIa inhibitors, 38 received intravenous GP IIb/IIIa inhibitors, and all received heparin. SICH occurred in 18 patients (9.3%). At 3 months, total mortality was 28.5% (n=55); 38.9% (n=75) achieved modified Rankin Scale (mRS) score ≤2. The only statistically significant different outcome was the lower recanalization rate (48.3%, P=0.007) observed with patients who had an ICA-terminus occlusion. No other statistically significant differences were found in outcomes of the different subgroups. A summary of the results is provided in Table 1.
CONCLUSIONS: Endovascular treatment of acute ischemic stroke among patients with a mean presentation NIHSS of 13.9±5.4 and who were not candidates for IVT or in whom IVT failed resulted in an immediate recanalization rate of 69.4%, mRS≤2 in 38.9% of patients, and mortality of 28.5% at 3 months. Patients with ICA-terminus occlusion had a significantly lower recanalization rate after endovascular treatment.

Table 1. Summary of Results

	No. of patients	Recanalization rate (%)	SICH (%)	mRS≤2 at 3 months (%)	Mortality at 3 months (%)
Total	193	69.4	9.3	38.9	28.5
Time from stroke symptom onset to thrombolysis					
0-3 hrs	60	61.7	5	40	31.7
3-8 hrs	80	70	7.5	46.3	22.5

>8 hrs	30	70	20	20	40
Wake-up strokes	23	87	13	34.8	26.1
		Location of occlusion			
Extracranial supra-aortic vessels	34	67.6	14.7	35.3	29.4
Tandem extra-cranial intracranial	23	69.6	17.4	26.1	34.8
Anterior circulation	169	68	9.5	39.1	27.8
Posterior circulation	24	79.2	8.3	37.5	33.3
Petrous ICA	9	44.4	11.1	11.1	55.6
ICA-terminus	29	**48.3 (P= 0.007)**	3.5	24.1	34.5
MCA	138	71.7	9.4	40.6	25.4
ACA	16	81.3	6.3	31.3	25
		Type of treatment			
Intracranial stent	52	71.2	11.5	42.3	21.2
Extracranial stent	36	72.2	8.3	47.2	22.2
Intracranial angioplasty	55	63.6	7.2	38.2	32.7
Extracranial angioplasty	24	75	12.5	45.8	20.8
Merci device	108	67.6	10.2	33.3	29.6
		Type of Thrombolysis			
Mechanical only	94	71.2	11.7	41.5	16.6
Pharmacologic only	24	62.5	4.2	41.7	33.3
Both	75	69.3	8	34.7	29.3

FRIDAY, NOVEMBER 6

8:40 – 8:52 AM **NONINVASIVE DETECTION OF MALIGNANT GLIOMA MOLECULAR SUBTYPES USING MAGNETIC RESONANCE PERFUSION-WEIGHTED IMAGING**

Gurpreet S. Kapoor Ph.D, Elias R. Melhem, M.D., ***Donald M. O'Rourke, M.D***.

BACKGROUND: Coexpression of EGFRvIII mutation and PTEN by GBMs is associated with therapeutic response to EGFR kinase inhibitors. Similarly, chemosensitivity of oligodendrogliomas (OL) and prolonged patient survival are predicted by 1p and 19q loss of heterozygosity (LOH). We have utilized MR perfusion-weighted imaging (MR-PWI) to predict and identify MR surrogates of EGFRvIII-expressing GBMs and 1p/19q deleted oligodendrogliomas.

METHODS: 44 patients with oligodendroglial tumors and 35/97 patients with primary GBMs were retrospectively reviewed for preoperative MR-PWI. OLs were stratified as Group 1 (1p or 1p/19q LOH), and Group 2 (19q LOH or intact alleles). Primary GBM tumors were grouped as EGFRvIII (-) (n=72) and EGFRvIII (+) (n=25) tumors using EGFRvIII-specific RT-PCR assay. The relative cerebral blood volumes (rCBV) were calculated in relation to contralateral white matter. Tumor tissues were analyzed for VEGF expression using real PCR to correlate with changes in rCBV.

RESULTS: The Group 1 grade II OLs showed significantly greater rCBV than Group 2 tumors (p=0.013). In grade III neoplasms, the differences between Group 1 and Group 2 were not significant. Grade III neoplasms showed a significantly higher rCBV than grade II neoplasms. Multivariate logistic regression analysis showed significant association of rCBV with 1p19q LOH and the expression of EGFR and VEGF in grade II OLs.

EGFRvIII (+) GBMs showed statistically significant higher maximum rCBV when compared to EGFRvIII (-) tumors (p=0.0017). Expression of VEGF was higher in EGFRvIII (+) GBMs compared to EGFRvIII (-) tumors. Accuracy of rCBV in detecting EGFRvIII mutation was higher (Az=0.82) than that of VEGF (Az=0.59). Multivariate logistic regression analysis revealed that rCBV assessment was a significant independent predictor of EGFRvIII mutation (p=0.014).

CONCLUSIONS: EGFRvIII mutation in GBMs and 1p/19q deletions in OLs are associated with significant elevations in rCBV as determined by MR-PWI. Collectively, our data suggest that MR-PWI may provide non-invasive surrogate biomarkers that define molecular subtypes of distinct malignant gliomas.

FRIDAY, NOVEMBER 6

8:54 – 9:06 AM **THE DEMISE OF THE POSTERIOR CERVICAL DISCECTOMY**

Clarence B. Watridge, M.D., FACS

INTRODUCTION: Since Semmes and Murphey's 1943 discovery and report that posterolateral cervical disc herniations cause neck, shoulder and arm pain (cervical radiculopathy), numerous patients have had successful relief of pain and neurologic deficits by surgical intervention. Many innovations have occurred in the pursuit of optimizing the surgical pursuit of this condition. The explosion of anterior approaches to the cervical spine has resulted in a diminished interest and expertise in the workforce for performance of the posterior cervical discectomy, a very successful procedure. Its future utilization is in jeopardy.

METHODOLOGY: A review of Medicare trend and expense data from 1992 to 2007 was performed to determine the trend and expense associated with cervical disc surgical procedures in that database. In addition the personal series of 129 consecutive single level cervical disc procedures performed by the author is reported. These procedures included all anterior and posterior single level cervical disc surgeries performed from 2005 through May 2009. Clinical results, complications and sample financial data are presented.

RESULTS: The Medicare database trend and expense data demonstrates a dramatic increase in the number of anterior surgical procedures performed. In 1992, 5,060 single level anterior cervical decompressions (63075) were reported whereas in 2007 16,953 procedures were submitted for claims. Addition of additional level surgeries (63076) raises the 1997 number by 2,219 (7,279 total) but the 2007 number for additional level surgery is 12,148 (29,101 total). 1997 charges were $6,428,263 while 2007 charges were $23,360,536. These costs do not include the charges for arthrodesis and instrumentation. In 1997 1,449 posterior cervical discectomies (63020) were claimed at a charge of $1,920,099 and in 2007 the posterior cervical discectomy was reported 1,261 times for charges of $1,270,009.

Analysis of the author's 129 single level disc procedures shows 64 were operated by a posterior cervical discectomy while 65 were operated anteriorly with allograft and cervical plating. The results were favorable with the complaints of neck pain and stiffness or no benefit occurring in a minority of anteriorly operated patients and a new neurologic deficit in one patient. There were no deaths, no infections, and no new spinal cord injuries. The physician and surgical facility charges averaged $11,500 for the posterior procedures and $30,000 for the anteriorly operated patients. Physician charges and reimbursement were 3 ½ times greater for the anterior procedures.

CONCLUSIONS: The posterior cervical discectomy may prove to be historical soon for several reasons.
* The physician is financially rewarded to perform the anterior procedure as opposed to a posterior approach. * Considering the few exposures of residents to the posterior procedure, competence and confidence in this procedure are absent in the younger surgeons. * Few orthopedic surgeons choose to perform a cervical disc procedure posteriorly.
The costs to society for the anterior procedure is triple the posterior surgery. Without recognition of this trend and intervention by neurosurgical educational societies, a very good surgical procedure will be lost to society. opportunity for therapeutic intervention and improved clinical outcome.

FRIDAY, NOVEMBER 6

9:08 – 9:20 AM

ANEURYSM INFLOW-ANGLE AS A DISCRIMINANT FOR RUPTURE IN SIDEWALL CEREBRAL ANEURYSMS: MORPHOMETRIC AND COMPUTATIONAL FLUID DYNAMIC ANALYSIS

Merih I. Baharoglu, M.Sc., Clemens M. Schirmer, M.D., Daniel A. Hoit M.D., M.P.H., Bu-Lang Gao M.D., and ***Adel M. Malek, M.D., Ph.D.***

BACKGROUND AND OBJECTIVE: The ability to discriminate between ruptured and unruptured cerebral aneurysms on a morphological basis may help in clinical risk stratification. The objective was to evaluate the importance of inflow-angle (IA), the angle that separates parent vessel and aneurysm dome axes.

METHODS: IA, maximal dimension (D_{max}), height-to-width ratio (H/W), and dome-to-neck aspect ratio (AR) were evaluated in sidewall-type (SW) aneurysms with respect to rupture status in a cohort of 116 aneurysms in 102 patients using high-resolution catheter-based cerebral 3-dimensional angiography. Computational fluid dynamic (CFD) analysis was performed in an idealized model with variational analysis of the effect of IA on intra-aneurysmal hemodynamics.

RESULTS: Univariate analysis identified IA as significantly more obtuse in the ruptured subset (124.9° ±26.5 vs. 105.8°±18.5, p = 0.0001); similarly D_{max}, H/W and AR were significantly greater in the ruptured subset; multivariate logistic regression identified only IA (p = 0.0121) and H/W (p = 0.0005), but not D_{max} or AR, as independent discriminants of rupture status. CFD analysis showed increasing IA leads to deeper migration of the flow recirculation zone into the aneurysm, with higher peak flow velocities and a greater transmission of kinetic energy into the distal portion of the dome. Increasing IA also resulted in higher inflow velocity, and wall shear stress magnitude and spatial gradients in both the inflow-zone and dome of the aneurysm.

CONCLUSION: Inflow-angle is a significant discriminant of rupture status in SW aneurysms, possibly via higher energy transmission to the dome; its relative importance as a predictor of rupture risk will require validation in a prospective trial.

Morphometric Feature	Ruptured	Unruptured	p-value	Area Under ROC Curve
IA, Inflow	124.9 ±	105.8 ±	**0.0001**	0.71
D_{max} (mm)	9.1 ± 3.7	6.2 ± 3.1	**0.0002**	0.75
H/W	1.60 ±	1.07 ±	**<0.0001**	0.76
AR	1.88 ±	1.36 ±	**0.0007**	0.73

Table 1: Univariate Analysis with Receiver Operating Characteristic Evaluation.

Morphometric Feature	χ^2	Prob > χ^2
Whole Model	34.44	**<0.0001**
IA, Inflow Angle	6.29	**0.0121**
D_{max}	3.27	0.0707
H/W	12.25	**0.0005**
AR	0.34	0.5605

Table 2: Multivariate Logistic Regression Analysis.

FRIDAY, NOVEMBER 6

9:22 – 9:34 AM **GLIBENCLAMIDE IS SUPERIOR TO DECOMPRESSIVE CRANIECTOMY IN A RAT MODEL OF MALIGNANT CEREBRAL EDEMA**

J. Marc Simard, M.D., Ph.D., Natalia Tsymbalyuk, M.D., Oresst Tsymabalyuk, M.D., Svetlana Ivanova, Ph.D., Volodymyr Gerzanick, M.D., PhD.

Decompressive craniectomy (DC) is currently used as the end-stage "salvage" treatment to prevent death in humans with severe ischemic brain swelling. Although death is the most dreaded endpoint in stroke, preclinical studies have seldom focused on treatments to reduce mortality. We developed a model of malignant cerebral edema (MCE) in male Wistar rats in which MCE is produced by 6-h ischemia (documented by LDF to be >75% reduction in rCBF) due to filament occlusion of the middle cerebral artery, followed by reperfusion with filament withdrawal. In untreated controls, this insult yields massive brain swelling, resulting in 24-hr mortality of >90%. Imrnunolabeling brain tissues following ischemia/reperfusion showed extensive upregulation of sulfonylurea receptor I (SURl), the regulatory subunit of the SURl-regulated NCCa-ATP channel that has been implicated in pathophysiological manifestations of CNS ischemia and trauma. In our model of MCE, treatment with a non-hypoglycemogenic dose of the SURI blocker, glibenclamide (10 Jlg/kg i.p. loading dose at 6 h, followed by continuous infusion of 200 nglh subcutaneously) reduced brain swelling by half, compared to vehicle-treated controls. We then compared the effect glibenclamide versus DC. both at 6 h, on mortality and neurological function using two independent examinations (an 8 point scale and a 21 point scale). Both glibenclamide and DC eliminated mortality. However, neurological function was significantly better in rats treated with glibenclamide compared to those treated with DC, with the difference in neurological function being apparent for the entire 2 weeks of serial neurological examinations. Even though both groups had large lesions due to the protracted period of severe ischemia, glibenclamide was much more effective in preserving deep white matter, which we hypothesize is the reason for their better neurological function. We conclude that pharmacological prevention of swelling by glibenclamide is superior to surgical decompression by craniectomy in terms of both tissue preservation and long-term neurological function.

FRIDAY, NOVEMBER 6

9:36 – 9:48 AM

GAMMA KNIFE RADIOSURGERY TREATMENT OUTCOMES FOR TRIGEMINAL NEURALGIA: THE UNIVERSITY OF CALIFORNIA SAN FRANCISCO (UCSF) EXPERIENCE

Nicholas M. Barbaro, MD, FACS, Thomas. T. Bui, MD, Tene. A. Cage, BS, Rodney. A. Gabriel, BS, Mariann M. Ward, RN, NP, Vivian Weinberg, Kim Huang, MD, Jean Nakamura, MD, Patricia Sneed, MD, William Wara, MD

INTRODUCTION: Gamma Knife radiosurgery has been used to treat trigeminal neuralgia (TN) pain since 1997 at our institution as part of a multi-procedural treatment algorithm that includes radiosurgery, microvascular decompression and radiofrequency lesion. We established a prospective database wherein all patients were followed at regular intervals for degree of pain relief, medication use and sensory complications. Herein we report our 12-year experience using Gamma Knife radiosurgery for TN.

METHODS: The records were reviewed for 354 consecutively accrued patients evaluated for TN from August 1997 to February 2009. We focused on 202 patients who were treated with Gamma Knife (GK) radiosurgery. We excluded patients with <6 mos follow-up (n=25), patients with co-morbid multiple sclerosis (n=17), patients with a prior surgical procedure for TN (n=4), and patients whose GK was a repeat procedure (n=5). This is a retrospective review of long-term pain outcome based on data collected in a prospective database of TN patients. A baseline questionnaire was supplemented by a complete history and neurological examination detailing pain distribution, frequency and intensity as well as sensory findings. Follow-up was at 6-weeks, 6-months and yearly. Neurologic evaluation was done with specific attention to trigeminal sensory function. Neuralgic medications were evaluated at each visit noting changes in number of medications taken and medication doses. Patients quantified the degree of pain reduction, if any, as $\leq$20%; >20%; >50%; >70%; >90%, and complete (100%). We defined significant pain relief as >50% and almost complete pain relief >90% pain reduction compared to baseline.

RESULTS: The total number of evaluable patients was 151. The mean pre-GK symptom duration was 105 mos (range 4-468). The mean age at GK treatment was 69 years (range 19-98). Median follow-up was 15 mos, with 42% of cases followed for at least 24 mos. Over this time period we increased the mean maximum GK dose used: from 1997 through 2000 (25 pts) the average was 72.2 Gy (range 70-75) while from 2001 onward (126 pts) it was 79.6 Gy (range 75-85). Significant pain relief was seen in 84% of GK pts, with 73% pts achieving almost complete pain relief. There was a dose-response relationship exists with 39% of patients treated with 70-75 Gy (n=38) and 99% treated with $\geq$80 Gy (n=113) achieving significant pain control. There was also a significant decrease in the number of neuralgia medications following GK therapy. Among pts initially receiving neuralgia medications 51% were able to reduce their medication doses with 36% being able to discontinue all neuralgia medications. Nearly all patients with objective sensory deficits had almost complete or complete pain control.

CONCLUSIONS: Gamma Knife radiosurgery is an effective treatment for TN with nearly all patients achieving significant pain relief and half able to reduce pain medications when treated at a dose of 80 Gy. Better pain relief occurs when some objective sensory deficit is produced. These data provide important information in aiding clinicians and patients make decisions on surgical treatment for TN.

FRIDAY, NOVEMBER 6

10:45 - 11:00 AM **THE TECHNIQUE AND CLINICAL OUTCOMES USING MODEST INTRAVASCULAR HYPOTHERMIA AFTER ACUTE CERVICAL SPINAL CORD INJURY**

Allan D. Levi, MD, PhD, FACS, Gizelda Casella MD,PhD, Barth A Green MD, FACS, W. Dalton Dietrich PhD, Steven Vanni DO, Jonathan Jagid MD, Michael Y Wang MD

INTRODUCTION: There is widespread interest in the use of hypothermia in the treatment of central nervous system injury. While there is considerable experience in the use of cooling for a variety of brain pathologies, limited data exists after spinal cord injury. In the last few years, technological advances in the induction and maintenance of cooling have been achieved and can potentially allow more rapid evaluation of this form of treatment.

METHODS: We performed a retrospective analysis of the medical records including clinical outcomes and complications in 14 SCI patients presenting with a complete cervical spinal cord injury (AIS A). All patients who met inclusion criteria were treated with 48 hours of modest (33^0C) intra-vascular hypothermia. A comparison group was composed of 14 age and injury matched subjects treated at the same institution.

RESULTS: The average age of the patients was 39.4 years (range from 16 to 62). With regard to neurological level, 21.4% were C4, 50% C5 and 28.6% C6. There was an excellent correlation between intravascular and intrathecal cerebrospinal fluid temperature. The average time between injury and induction of hypothermia was 9.17 $\pm$ 2.24 h (mean $\pm$ SEM); the time to target temperature was 2.72 $\pm$ 0.42 h; the duration of cooling at target temperature was 47.6 $\pm$ 3.1 h; the average total length of time of cooling was 93.6 $\pm$ 4 h. There was a positive correlation between temperature and heart rate. Six of the 14 (42.8 %) cooled patients were incomplete at final follow-up (50.2 ± 9.7 weeks). Three patients improved to AIS B, 2 patients improved to AIS C and 1 patient improved to AIS D. Complications were predominantly respiratory (atelectasias, 85.7%; pneumonia, 57.1%; and Adult Respiratory Distress Syndrome, 14.4%) and infectious (urinary tract infection, 57.1% and sepsis 7.1%) in nature. However, in the control group a similar number of complications was observed.

CONCLUSION: We were able to effectively deliver systemic cooling using the cooling catheters with minimal variation in body temperature. The study represents the largest, modern series of hypothermia treatment of acute spinal cord injury with intravascular cooling techniques and provides needed baseline data for outcome studies to include larger multi-center, randomized trials.

FRIDAY, NOVEMBER 6

10:24 – 10:38 AM **Cavernous Malformation Radiosurgery: Seeking Détente**

L. Dade Lunsford, M.D. A. Naranjan,M BBS MS MCh,Hideyuki Kano, M.D., Ph.D., Aftab Khan, M.D., John C. Flickinger, M.D., Douglas Kondziolka, M.D.

OBJECT: The benefit and safety of stereotactic radiosurgery (SRS) in patients with solitary, high surgical risk cerebral cavernous malformations (CCM) are considered controversial. We reviewed our experience in order to seek detente among opposing factions. Methods and Materials:

Between 1988 and 2007 we performed SRS on 103 patients (57 males, 46 females) who had solitary, symptomatic CCM and multiple bleeding episodes confirmed by imaging.. The mean patient age was 39 years. Seventeen patients (16.5%) had undergone incomplete surgical resection before SRS. In all other patients surgical management was considered to have unsatisfactory risks related to the location ofthe CCM within the parenchyma of critical brain structures. Ninety-three (90010) were located in deep brain structures, and ten (9.7%) were in lobar regions ofthe brain (i.e. motor cortex, deep temporal lobe). The medium CCM volume was 1.31 mI., and the median marginal dose was 16 Gy. Results:

At a median of43 months the annual symptomatic, imaging-confirmed hemorrhage rate significantly decreased from 32% annually before SRS to 4.5% after radiosurgery (p<O.OOOl). After a two year latency interval the annual hemorrhage rate decreased to 1 % per year, which approximates the annual bleed rate for newly diagnosed CCM. Overall, new neurological deficits related to adverse radiation effects developed in 14 (13.5% ofpatients), primarily early in our experience. The hemorrhage rates before and after radiosurgery were not explained by temporal clustering. Targeting related to CCM radiosurgery has been enhanced by providing highly conformal and selective radiation delivery within the 1'2 signal volume defined by high resolution MR imaging.

CONCLUSIONS: Symptomatic CCM that repeatedly bleed should be considered for primary surgical resection. Most patients with lesions eligible for surgery will have CCM near a pial or ependymal surface. Providing that a low morbidity surgical corridor is feasible, surgical cure is possible (although in our experience 16% of patients had already failed surgical resection). For patients who have multiple bleeding events from deep seated CCM located in the parenchyma ofthe brainstem, diencephalon, or critical deep areas of functional white matter, SRS is a safe option that significantly reduces subsequent bleeding risks, especially after a two year interval has elapsed. We believe that gamma knife SRS should be considered an additional management option for properly selected CCM deemed ineligible for resection. For such patients the only other option is continued observation.

FRIDAY, NOVEMBER 6

10:40 – 10:58 AM **Academy Award Winner**
EGFRvIII Antibody Conjugated Iron Oxide Nanoparticles for Targeted Imaging and Therapy of Glioblastoma

Costas Hadjipanayis, M.D.

SATURDAY, NOVEMBER 7

8:25 – 8:37 AM **THE ROLE OF VEGF IN PEDIATRIC HYDROCEPHALUS**

Joseph R. Madsen MD, Joon W. Shim PhD, Johanna Sandlund MD PhD, Carin H. Han, Mustafa Hameed MD, Gani Abazi MD, Laurel Fleminig BA, Sandra Smith MS, Susan Connors, Michael Klagsbrun PhD, and Judah Folkman MD

INTRODUCTION/HYPOTHESIS: Vascular endothelial growth factor (VEGF) is a highly potent angiogenic and permeability-inducing factor. Elevated levels of VEGF or its receptor (VEGFR-2) have been associated with experimental and human hydrocephalus (HC), and we hypothesize that it may have a causal relationship with this neurological condition.

METHODOLOGY: Cerebrospinal fluid (CSF) was collected at the time of surgery from pediatric patients with hydrocephalus and other nonneoplastic conditions for analysis of VEGF levels. Enzyme-linked-immunosorbent serologic assay (ELISA) sensitive to splice variant isoforms of VEGF-A_{165} and $-A_{121}$ was conducted to detect this growth factor in CSF of the patient population in the absence or presence of HC. To explore a possible contributory role of VEGF in HC in an animal model without obstruction of CSF pathways, mini-osmotic pumps for intraventricular infusion of VEGF-A_{165} were implemented to assess the impact of intraventricular VEGF on ventricular size and function, and to test the efficacy of inhibitors for VEGF-A (bevacizumab) and VEGFR-2 (semaxanib).

RESULTS: The clinical data from ELISA demonstrate that VEGF-A is significantly elevated in CSF of pediatric HC. Specifically, seventy four CSF specimens were assayed blindly from hydrocephalic and nonhydrocephalic patients with nonneoplastic diagnoses (epilepsy, tethered cord, and Chiari malformations), with a median CSF VEGF-A of 56 pg/ml in cases of HC, and less than the threshold of 15 pg/ml in the controls ($p<0.001$, Mann-Whitney). Among HC patients, CSF VEGF-A is statistically higher in post-hemorrhagic than non-hemorrhagic cases ($p<0.05$). This prompted further study of the role of VEGF in the development of the hydrocephalic phenotype. Regulated infusions of VEGF-A_{165} for 7 d via osmotic pumps into the rat brain reveal that VEGF-A_{165} induces ventriculomegaly, angiogenesis, and hyperpermeability in the periventricular region. Expression of VEGFR-2, one of the receptors for VEGF-A_{165}, is limited to choroid plexus in the sham-operated control (vehicle infused). In contrast, exuberant VEGFR-2 immunoreactivity is visible in the ependyma, subependyma, and septum in the VEGF-induced HC rats. Inhibition of either a humanized form of VEGF-A by bevacizumab or endogenous VEGFR-2 by semaxanib was sufficient for the recovery of blood brain barrier exhibited by intravenous injection of Evans blue. Simultaneous delivery of VEGF-A_{165} and bevacizumab did not result in ventriculomegaly indicating VEGF-A_{165} was responsible for the development of HC in this animal model.

CONCLUSIONS: VEGF may plausibly play a role in the pathophysiology of HC and blocking its action by VEGF antagonists, especially for hydrocephalus with high CSF VEGF level or high vascular permeability in the central nervous system, may be a potential therapeutic approach. Further experiments will target VEGF blockers in other models of hydrocephalus.

SATURDAY, NOVEMBER 7

8:39 – 8:51 AM **THE ROLE OF CXCR4 (CHEMOKINE (C-X-C MOTIF) RECEPTOR 4) IN HUMAN INTRACRANIAL ANEURYSMS**

Changbin Shi, MD, PhD; Issam A. Awad, MD, FACS; H. Hunt Batjer, MD, FACS; Nadereh Jafari, PhD; Arun Kumar Sherma, MD; Markus Bredel, MD, PhD; ***Bernard R. Bendok, MD***

INTRODUCTION/HYPOTHESIS: Despite potential catastrophic consequences of intracranial aneurysms (IA), the exact pathogenesis remains elusive. Recently our functional microarray data demonstrated a dysregulation of the inflammatory and immune response pathway in IA, strongly suggesting a role for inflammation in the pathobiology of IA. The CXCR4 (chemokine (C-X-C motif) receptor 4) gene was noted to be located centrally in the functional microarray pathway analysis, indicating that it maybe a key regulator in the inflammatory and immune response pathway in IA. CXCR4 has not been previously investigated as a potential mediator in IA pathobiology.
METHODOLOGY: Intracranial aneurysm walls and the paired superficial temporal arteries (STA) surgically excised from four patients with IA were homogenized, and the high quality of total RNA was used for reverse transcription with a high-capacity cDNA reverse transcription kit. TaqMan quantitative real-time PCR was performed in triplicates and normalized with 18S rRNA endogenous control. All values were expressed relative to the level of 18S rRNA endogenous control whose expression was normalized to 100%. The immunofluorescent protein staining of CXCR4 in the surgically excised and fresh prefixed aneurysm wall and STA specimens was performed. The primary antibody used for the immunofluorescent staining was mouse anti-human CXCR4 (fusin) monoclonal antibody. Slides were viewed with a Zeiss LSM 510 META confocal microscope.
RESULTS: Our quantitative real time PCR data showed that CXCR4 mRNA was more highly expressed in four human IA walls than paired STA specimens, and fold changes were1.53, 4.88, 5.84, 6.04 respectively. There was a significant different expression between IA group and control-STA group (P=0.024). We have further shown greater CXCR4 immunofluorescent protein in IA walls as compared with the paired STA specimens.
CONCLUSIONS: Our preliminary results suggest CXCR4 could play a role in the pathogenesis of IA. The association of CXCR4 with growth and rupture of IA, and its mechanisms in the pathobiology IA warrant further investigation. Our laboratory is currently analyzing a larger number of samples to this end.

SATURDAY, NOVEMBER 7

8:53 – 9:05 AM **CHALLENGES IN DIAGNOSIS AND**

MANAGEMENT OF CUSHING DISEASE

Imad Kanaan, M.D., FACS, FRCSEd.

INTRODUCTION: Cushing disease is a rare entity caused by ACTH-producing pituitary adenoma and accounts for almost 15% of all pituitary adenomas. The evolution of pertinent biochemical and Neuro-imaging investigation during the past decades has enhanced diagnostic reliability of Cushing disease. The introduction of the microadenoma concept and the refinement of transsphenoidal surgery made by Hardy are the comer stone in the management of Cushing disease.

MATERIAL & METHOD: A retrospective review of patient material diagnosed to have pituitary tumor (1986-2008) is in progress. The total number of patients that has been dealt with during the past 24 years exceeds 1200 cases. The author has selected several cases of Cushing disease in order to highlight the diagnostic and therapeutic challenges that face the treating physician with focus on surgical approach, role of new technology and decision making regarding recurrences and Stereotactic radiotherapy.

CONCLUSION: The direct endonasal transsphenoidal approach coupled with experience in microsurgical and endoscopic dexterity has promoted minimal-invasiveness, improved patient safety and treatment outcome. However, difficult diagnostic confirmation/localization in some cases, tumor invasiveness, absent curative/ alternative medical treatment and variable response to stereotactic radiation therapy are among the challenges that the treating physicians are faced with.

SATURDAY, NOVEMBER 7

9:07 – 9:19 AM **MY EXPERIENCE IN 166**

INTRAMEDULLARY SPINAL CORD

EPENDYMOMAS

Jacques Brotchi, M.D., Ph.D.

INTRODUCTION: It is well known that the gold standard treatment for low grade intramedullary spinal cord ependymomas (ISCE) is complete surgical removal. That should be the ultimate goal.

RESULTS: Our experience is based on 166 ISCE among 425 operated intramedullary tumors. We did not succeed to totally remove all ISCE and we had recurrences in some patients with complete removal.

Complete removal has been achieved with success in 148 cases (89%). Subtotal or partial removal happened in 18 (11 %). In some cases indeed, we have been faced with a lack of a clear cleavage plane. Motor evoked potentials (MEPs) have been of great help in those situations to keep a good quality of life after surgery. But when a clear plane was present between the ependymoma and spinal cord, we did not stop surgery even when MEPs were lost, without facing neurological motor deficit postoperatively. It means that MEPs are not an absolute criteria.

In partial removal, we had to operate again several years later, sometimes in succeeding to perform a complete removal (7 cases) but in 6 patients, we failed and they regularly have to be operated again. In two patients, the remaining bud stays stable on yearly follow-up MRIs.

Our follow up runs from several months to 24 years. In 119 patients, we have a follow-up longer than 5 years. In all, we recommend a yearly MRI since in two cases with complete removal and clean MRI at 5 years, we observed a recurrence after 18 and 19 years respectively.

CONCLUSION: We should keep in mind that even after complete removal, ISCE may recur many years after a successful surgery. MRI follow-up of those patients should never stop. On the other hand, if in around 89% ISCE can be totally removed, in 11 % no clear plane of delineation should temper our surgical aggressivity to keep a *good* quality of *life* post-operatively. Surgery again is recommended when those tumors regrow.

SATURDAY, NOVEMBER 7

9:21 – 9:33 AM **INHIBITION OF MTOR MINIMIZES REACTIVE ASTROGLIOSIS FOLLOWING BRAIN INJURY**

Charles B. Mikell, M.D., Alexander A. Sosunov, M.D., Xiaoping Wu, M.D., Robert A. McGovern, B.S., Zachary G. Wright, B.S., Andrew Ducruet, M.D., E. Sander Connolly, M.D., and ***Guy M. McKhann, II, M.D.***

INTRODUCTION: Reactive astrogliosis is a conserved brain response to numerous CNS insults, including trauma, seizure, infection, and ischemia. Reactive astrocytes exhibit characteristic morphological features, including upregulation of intermediate filament (IF) proteins, hypertrophy, and proliferation. Functional abnormalities in reactive astrocytes, especially defects in potassium and glutamate uptake, contribute to continuing neurological deficits and seizures following brain injury. Reactive astrocytes have thus been proposed as a therapeutic target. However, no therapy to ameliorate reactive astrogliosis is currently available. The mammalian target of rapamycin (mTOR) is a critical kinase involved in numerous cell processes, including cell division, growth and differentiation. We have previously reported activation of the mTOR cascade in the astrogliosis that characterizes epileptogenic cortical tubers in the genetic condition tuberous sclerosis complex. In this study, we investigated mTOR in human and rodent acquired gliosis.

METHODOLOGY: Using immunohistochemistry and electrophysiology, we studied human acutely reactive neocortex around implanted depth electrodes and chronically gliotic hippocampus resected from temporal lobe epilepsy patients. In parallel, we investigated rodent models of trauma, ischemia, and epilepsy. We determined whether activation of the mTOR cascade is found in reactive human and rodent astrocytes, and whether inhibition of mTOR could limit the reactive changes that occur in astrocytes in response to brain injury.

RESULTS The mammalian target of rapamycin (mTOR) cascade is activated in acutely reactive astrocytes and in astrocytes in areas of chronic gliosis in human brain. The mTOR pathway is similarly upregulated in rat models of various brain insults, including trauma, ischemia, and status epilepticus. Activation of mTOR precedes GFAP upregulation in reactive astrocytes. The mTOR inhibitor rapamycin minimizes reactive gliosis following rodent neocortical stab wound injury and in acute human tissue slices, as indicated by inhibition of expression of the downstream substrate of mTOR, phospho-S6; decreased expression of intermediate filaments GFAP and vimentin; and preservation of astrocytic glutamate transport and potassium uptake function in affected tissue. Ultrastructural alterations in astrocytes that accompany the conversion of a normal protoplasmic astrocyte to a reactive astrocyte are largely prevented by mTOR inhibition.

CONCLUSIONS: The mTOR pathway is activated in acute and chronic human and rodent gliosis. The mTOR inhibitor rapamycin ameliorates the anatomical and functional changes in astrocytes that occur in reactive astrogliosis. The mTOR cascade is a potential therapeutic target with a variety of human applications such as the prevention of epilepsy following brain injury and improving brain machine interface recording fidelity.

9:49 – 10:01 AM **MICROBALLOON ANGIOPLASTY IS SAFE AND EFFECTIVE TREATMENT IN SMALL DISTAL CEREBRAL VESSELS**

Alan S. Boulos, M.D., Doniel Drazin, M.D., Edward Gifford, John C. Dalfino, M.D., Anil K. Nair, M.D., A. John Popp, M.D.

INTRODUCTION: Mechanical angioplasty has been established to be an effective therapy in the treatment of peripheral, coronary, and large proximal cerebral vessels. In neurovascular interventions, angioplasty has been shown to be an effective treatment for vasospasm and ischemic stroke. This study was undertaken to examine the safety and effectiveness of angioplasty in the treatment of small distal cerebral vessel (<=2mm) with severe stenosis or occlusions. Historically, this group of patients was felt to be at too high risk for vessel rupture to safely angioplasty. Specifically, outcomes of patients in the following two groups were analyzed: (1) angioplasty of anterior cerebral artery (ACA) vasospasm in order to prevent ACA distribution infarcts and (2) angioplasty of distal (M2 or smaller) middle cerebral artery (MCA) occlusions to treat ischemic stroke.

METHODS: Patient charts were analyzed retrospectively using a prospectively maintained database. Seventeen consecutive patients were treated with mechanical angioplasty in either the ACA or distal MCA distribution for the treatment of either vasospasm or ischemic stroke from July 2003 to July 2008. Treatment was delivered following diagnostic angiography. All received microballoon angioplasty (PTA) to their affected vessels. In 15 of 17 patients, low compliant coronary angioplasty balloons were used for treatment. In 2 patients, high compliant balloons for ACA angioplasty were used. Stent placement followed in two patients with ischemic stroke in the MCA territory. All patients received intra-arterial (IA) medications for either thrombolysis in ischemic stroke or spasmolytics for vasospasm. Retrospective review of the patients' records included demographics, clinical grade, procedural details, complications, and clinical outcomes. For ischemic stroke patients, NIHSS and mRS were recorded at discharge and follow-up respectively. For vasospasm patients, Hunt-Hess grade, GOS, and mRS were recorded on admission, discharge and at follow-up.

RESULTS: PTA was successful in seventeen patients without treatment-associated intracerebral hemorrhage. For ischemic stroke patients, distal embolization occurred in one of nine patients. Full recanalization achieved in 5 of nine patients, near-complete recanalization (modified TIMI 3) in the remaining 4 patients. MRS of 0 or 1 occurred in 7 of 9 patients, one died from persistent significant neurological deficits despite full recanalization with angioplasty, and one patient suffered slight disability (mRS 2). For vasospasm patients, PTA successfully treated A1 or proximal A2 vasospasm without need for recurrent therapy. No procedural complication related to ACA angioplasty occurred; however, one patient suffered a vertebral artery dissection during coil embolization of a basilar artery aneurysm. Resolution of vasospasm was graded as complete in 6, mild in 1 and moderate in the remaining patient. Mean follow-up mRS was 2. Four patients had persistent neurological deficits at follow-up.

CONCLUSION: PTA for small distal cerebral vessels is safe and effective in the treatment of ischemic stroke and subarachnoid hemorrhage associated vasospasm. A larger multicenter prospective study is required to analyze these findings further. The advent of improved microwires and better balloons has resulted in improved treatments for stroke patients suffering from ischemia. The clinical outcomes for these patient groups can be substantially improved with this technology.

SATURDAY, NOVEMBER 7

9:49 – 10:01 AM **THE USE OF POSITIONAL MAGNETIC RESONANCE IMAGING TO ASSESS PATIENTS WITH LOW BACK PAIN**

Rick Edgar, M.D., Chris Karas, M.D., Mirza Baig, M.D., Ph.D., Andrew Shaw, M.D., Riley Splittstoesser, Sue Ferguson, Ph.D., William Marras, Ph.D., ***Ehud Mendel, M.D.***

PURPOSE: To investigate spinal anatomy in various postures using positional MRI in patients with low back pain. Radiographic findings, including intervertebral disk heights, thecal sac width, spondylolisthesis, and neural foraminal width, will be correlated with clinical symptoms and subjective pain severity scores.

BACKGROUND: 22.4 million people suffered from low back pain in the US in 2001 (NRC,2001). A specific diagnosis for its cause is unknown in 80-90% of cases (Spratt et. al.,1990). Functional physiological movements and pathologic conditions, such as lower back pain can alter the relationships of the lumbar structure, function and motion. MRI is widely used as the standard imaging modality to analyze the spinal canal, disk space, and neural contents. However, this has traditionally been done in a recumbent posture in which the axial spine is unloaded. Positional MRI is a relatively new option that allows assessment of the spine in a more physiologic manner. This has more limited availability and its efficacy in identifying relevant pathology in the lumbar spine is largely untested.

MATERIALS AND METHODS: One-hundred eighty-three patients with low back pain between 18 and 80 years of age will be included in the study. The only exclusion criteria are contraindications for MRI. Positional MRIs will be obtained using an open FONAR MR imager with a 0.6 T magnet. Non-contrast T2 images through the lumbar spine (axial and sagittal from T12-S1) will be acquired while supine, sitting, and standing. Further analysis will be performed with flexion at 60 degrees,and extension at 15 degrees in the upright and seated postures. Dependent measurements include thecal sac width, intervertebral foramina width, intervertebral disc height, and listhesis in axial and sagittal planes. These findings will be correlated with each subject's pain severity and disability scores.

RESULTS: Thus far, 65 patients have been enrolled in the study with imaging analysis complete in 28. Three patients have been unable to complete the necessary imaging requirements secondary to pain in dependent postures (10.7%). Of significance, thecal sac width has varied on average 1.6mm between flexion and extension postures with a range of 0-6.5mm within individual subjects (std dev=1.4mm). A 12.5% difference was detected when comparing thecal sac width in the sitting versus recumbent posture with a range of 8.1 to 15.6% depending upon intervertebral disk level.

CONCLUSIONS: Preliminary findings reveal positional MRI may enhance the ability to diagnose the cause of low back pain and/or identify surgically correctable pathology compared to traditional recumbent imaging. Additional research, including completion of this study, is warranted to determine clinical applicability of positional MRI as well as increasing the statistical power of these results and other dependent measures.

SATURDAY, NOVEMBER 7

10:23 – 10:35 AM **EXPERIMENTAL THERAPIES TARGETED TO MICRORNA EXPRESSION IN GLIOMA STEM CELLS**

E.A. Chiocca, M.D., Ph.D., Sean Lawler, Ph.D., Jakub Godlewski, Ph.D.

Two phenotypic hallmarks of malignant gliomas are its invasion and its sternness. We have been evaluating microRNA's (miR's) which are recently described small non-protein-coding RNA molecules that are involved in the modulation of protein synthesis, via binding to complementary mRNA. They are also known to modify the activity of oncogenes and tumor suppressor genes of tumors, such as glioblastoma multiforme (GBM), the most common and deadly form of brain cancer. Using microarray analysis and quantitative RT-PCR, we have noted that miR-128, which is normally abundant in normal brain tissue, is significantly under-expressed in GBM tumor samples (18.75-fold reduction). Ectopic expression of miR-128 using oligonucleotide precursor or lentiviral vector reduced glioma cells growth considerably - both in vitro (U87, U251) in vivo (flank), as well as in models that enrich for glioma "stem-like" cells. Direct effects on several target genes functionally linked to regulation of oncogenic growth related processes, as well as impact exerted on stem cell - like self-renewal will be discussed. In conclusion, miR's are important modulators of GBM proliferation, and have the potential to be used as diagnostic or therapeutic agents.

SATURDAY, NOVEMBER 7

10:37 – 10:40 AM **DEVELOPING A NEW GENERATION OF ACADEMIC NEUROSURGEONS**

Robert J. Dempsey, M.D.

INTRODUCTION: While considerable emphasis has been placed on development of neurosurgery students and residents, the future of academic neurosurgery will rest solely on the hands of the generation of young faculty that are entering their careers at this time. The successful strategies that mentor and develop their academic career are imperative.

HYPOTHESIS: In a time of economic uncertainty, conflicting service demands and difficult research funding, neurosurgery sponsored start-up awards, as well as a thoughtful and productive approach to mentoring, academic development, and early grant achievement will assure the future of our specialty. An assessment of the career success of past award winners will prove or disprove the utility of neurosurgery sponsored awards in faculty development.

The components of successful faculty development must include mentoring, collaborative research support, protective time and skills development. Mentoring must be a structured formal process with multi-departmental input and feedback independent of the department chair, providing influence and feedback to the chair on behalf of the faculty member. Academic and clinical expectations should be mutually agreed by distribution of effort and progress reviewed against this agreement biannually. Salary should be tied to achievement of clinical and academic goals as per pre-agreed targets. Equipment, support and space should be designed to facilitate efficient work and avoid unnecessary obstacles to success. Mentored-funded early academic awards are important to the process. We will present here the success of the awards: Van Wagenen, Joint Section Research Award and NREF in the development of young faculty by documenting the career success of past winners.

CONCLUSION: The growth of academic neurosurgery will take place only through proactive career planning and mentoring of our junior faculty.

SATURDAY, NOVEMBER 7

10:42 – 10:49 AM **MULTIPLE CRANIOTOMIES IN THE MANAGEMENT OF MULTIFOCAL-MULTICENTRIC HIGH GRADE GLIOMAS**

Raymond Sawaya, M.D.

OBJECT: Extent of resection has been evaluated as a predictor of survival in the management of glioblastoma; however, its role in the management of multifocal-multicentric high grade gliomas is undetermined.

METHODS: A retrospective review of patients with multifocal-multicentric high grade gliomas who underwent surgery for all lesions via multiple craniotomies during the same surgical session at The University of Texas M. D. Anderson Cancer Center between 1993 and 2008 was performed. Of these, 9 had two distinct lesions, no FLAIR connection, and no route of dissemination identified (Group A, multicentric). 11 had two or three distinct lesions, but did not fulfill the previous criteria (Group B, multifocal). 20 patients with solitary high grade gliomas who underwent resection (Group C) were selected to match Groups A & B with respect to the preoperative Karnofsky scale score, tumor functional grade, extent of resection, age at time of surgery and year of surgery. Clinical and neurosurgical outcomes and recurrence rates were evaluated.

RESULTS: Statistical analysis indicated that Groups A&B were homogeneous for age, sex, preoperative Karnofsky scale score, tumor necrosis, cysts, previous treatment, use of intraoperative navigation, and extent of resection. The median age for Groups A&B was 52 years (range 32-75 years). Seventy percent of patients in Groups A&B were male, while 65% were female in Group C. Aggressive resection of all lesions in Groups A&B was achieved via multiple craniotomies in the same session with median extent of resection of 100%. The median survival was 12.9 months for Group A, 9.6 months for Group B, and 10.5 months for Group C. No statistically significant difference in survival time between the three groups was found due to small sample size. Groups A &B (multiple craniotomies in the same session) and Group C (single craniotomy) had complication rates of 30% and 35% and 30-day mortality rates of 5% and 0%, respectively.

CONCLUSION: Aggressive surgical resection of all lesions in selected patients with multifocalmulticentric high grade gliomas results in similar survival time to that of patients undergoing surgery for a single lesion without an associated increase in postoperative morbidity.

SATURDAY, NOVEMBER 7

10:51 – 11:03 AM **OBSERVER BIAS IN BIO-SURGICAL TRIALS OF NOVEL PARKINSON'S DISEASE THERAPIES**

Ron L. Alterman, MD, Michele Tagliati, MD, H. Richard Winn, MD, C. Warren Olanow, MD

BACKGROUND/OBJECTIVE: Six times in the last 10 years, a surgically administered biological therapy for Parkinson's disease (PD) that appeared to be effective in an open-label feasibility trial (OLT), failed to generate similar results when tested subsequently in a prospective, double-blind sham surgery-controlled trial (SSCT). The prevailing consensus is that the Type I errors (ie false positive result) generated by the OLTs was due to the susceptibility of PD patients to the placebo effect, supporting the need for SSCTs in PD. In comparison, the role that observer bias played in generating these Type I errors has received little attention. The goal of the current study was to assess the relative importance of placebo effect and observer bias in generating these Type I errors.

METHODS: Review and meta-analysis of the clinical responses and complications reported from both the initial OLT and the subsequent SSCT of these six bio-surgical therapies. When available, the off medication Unified Parkinson's Disease Rating Scale Motor Sub-score (UPDRS-III) was recorded so that clinical responses could be compared across trials.

RESULTS: For the three fetal cell transplantation studies (two human, one porcine) a direct comparison could not be made between the OLT and SSCT because different clinical rating scales were employed. Nevertheless, the responses in the SSCTs were far less dramatic than those reported in the OLTs. For the three most recently tested therapies a striking and measurable trend is noted. In four OLTs of these three therapies (intraputaminal infusion of GDNF, retinal pigemented epithelial cell transplantation, and AAV2-mediated Neurturin gene therapy) an average 43% (range: 36-57%) improvement is reported in the off medication UPDRS-III as compared to a more modest 11% improvement in the subsequent SSCTs. Moreover, a number of therapy-related serious adverse events including runaway dyskinesia emerged during the SSCTs that were not observed during the OLTs.

CONCLUSIONS: Observer bias was a stronger factor than placebo effect in yielding the Type I errors generated by the OLTs of these six bio-surgical therapies for PD. These data call into question the value of OLTs in evaluating novel PD therapies. Moreover, the number of patients studied in these OLTs (5-12) was too small to provide a reasonable estimate of the risks posed by the new therapy, which, after all, is the primary purpose of Phase I study. We propose that future Phase I trials of novel bio-surgical therapies for PD be comprised of 15-20 patients and that the clinical response be measured in a blinded fashion with comparable patients receiving 'best medical therapy' or even deep brain stimulation serving as controls. Therapies that appear effective in such a study will be far more likely to be proven effective in a subsequent SSCT, thereby minimizing the chance that additional patients will be subjected to the risks of a biosurgical therapy that is ineffective.

SATURDAY, NOVEMBER 7

11:05 – 11:17 AM **THE GUANINE NUCLEOTIDE EXCHANGE**

FACTOR SWAP-70 MODULATES GLIOMA MIGRATION AND INVASION

Ho Jun Seol, M.D., Christian Smith, Ph.D., Bodour Salhia, Ph.D., ***James T. Rutka, M.D.***

The malignant glioma is the most common primary human brain tumor. Its invasiveness and tendency to migrate away from the primary tumor mass is considered a leading cause of tumor recurrence and treatment failure. The molecular pathogenesis of malignant glioma invasion is currently under investigation. Previously, we performed a gene expression array study comparing human gliomas to non-neoplastic controls and identified several Rac guanine nucleotide exchange factors with differential expression. Here, we report that the guanine nucleotide exchange factor SWAP-70 has increased expression in malignant gliomas which strongly correlates with decreased patient survival. SWAP-70 is a multi-functional signaling protein involved in membrane ruffling that works cooperatively with activated Rac. Using a glioma tissue microarray, we validated that SWAP-70 demonstrates higher expression in malignant gliomas compared to low-grade gliomas or non-neoplastic brain tissue. Through immunoflorescence and western blot analysis, SWAP-70 is inducibly localized to membrane ruffles in response to the rac-activating growth factor Epidermal Growth Factor (EGF). To assess the role of SWAP-70 in glioma migration and invasion, we inhibited its expression by siRNA and observed decreased glioma cell migration and invasion, even when stimulated with EGF.

SWAP-70 over-expression led to increased levels of active Rac even in low-serum conditions. In addition, when SWAP-70 was over-expressed in glioma cells and treated with EGF, we observed enhanced membrane ruffle formation followed by increased cell migration and invasiveness. Taken together, our findings suggest that the guanine nucleotide exchange factor SWAP-70 plays an important role in the migration and invasion of human glioma cells.

SATURDAY, NOVEMBER 7

11:19 – 11:31 AM **TRANSCUTANEOUS INDUCTIVE**

INTRACRANIAL PRESSURE MONITOR:

AN IMPLANTABLE WIRELESS DEVICE

FOR DIAGNOSING SHUNT MALFUNCTION

Josh Medow, M.D.

INTRODUCTION: Symptoms of shunt malfunction are frequently inconsistent and pose diagnostic dilemmas that have the potential for adverse consequences. Observation and exploratory surgery in patients that are found later to have had a functioning shunt are often expensive and may inflict unnecessary morbidity. A means of ascertaining intracranial pressure is necessary to best treat patients with shunted hydrocephalus. The technology exists to build a functioning implantable intracranial pressure monitor to scale. We describe our operational prototype and the next step in the downsizing process.

METHODS: A Medtronic Becker Burretrol system was raised and lowered changing a column of fluid between -5cmH20 and 30cmH20. The transmitter frequencies and receiver voltages on two digital multimeters were recorded. We then pushed and aspirated on a 10m1 syringe to qualitatively evaluate a waveform on a 50MHz analog oscilloscope.

RESULTS: Voltage and frequency output varied nearly linearly with changes in pressure. Every increase in pressure by 5cmH20 resulted in an approximate increase in oscillation by 10Hz. A similar .IV per 5cm.H20 increase was observed on the volt meters as well. A pressure waveform is qualitatively and quantitatively delineated with syringe .

CONCLUSION: An inductively powered intracranial pressure monitor has been built and demonstrates proof of concept. Additional research is necessary to further develop the device for implantation.

SATURDAY, NOVEMBER 7

11:33 – 11:45 AM **STATINS DECREASE EXCITOTOXIC CELL DEATH AND RESULTANT CORTICAL DYSFUNCTION**

Charles Hodge, M.D.

INTRODUCTION: A significant portion of the neurologic deficit from brain trauma, ischemia or hemorrhage is due to the shared mechanisms of excitotoxic neuronal cell death (END) from necrosis and apoptosis. Attempts to ameliorate this damage with calcium channel and NMDA glutamate receptor blockers in clinical situations have failed. Other potential neuroprotective agents therefore deserve evaluation. The statin drugs have been reported to be neuroprotective unrelated to their cholesterol lowering effects. This study is designed to determine if pravastatin (PS) protects neurons from END. END was produced in the rodent whisker barrel cortex with a cortical layer IV micro-injection of the excitotoxin kainic acid (KA), a glutamate analog.

METHODS: Experiments were done in pairs, one of the animals serving as control and the other as the test animal. The test animals received either pre-injury PS for one week and continued PS for a one week survival time or received only one week of post-injury PS. Neuronal cell death was determined by counting dead or dying neurons marked with fluoro jade (FJ). Cytochrome oxidase (CO) staining served as a marker of cortical metabolism, and intrinsic cortical signal imaging was used as a marker of cortical physiologic function.

RESULTS: KA injection reliably caused neuronal cell death at the injection site and surrounding barrel cortex. This was associated with decreased CO oxidase staining in the areas of the dead neurons. Similarly, the KA injected control animals had markedly disrupted ability to respond to contralateral myastatial whisker movement. Both the test animals receiving pre- and post-injury PS (group 1) and those receiving only post-injury PS (group 2) had decreased cell death. Group 1 had a 67-99 percent decrease in END while group 2 had a 50-60 percent decrease in cell death. The functional studies using CO staining demonstrated that there was less loss of CO intensity in both groups of PS treated animals. There was also less disruption of cortical responsiveness to contralateral whisker movement in the PS groups of animals.

CONCLUSIONS: This data indicates that the PS has potent neuroprotective effects in a model of excitotoxic cell death. The mechanisms of this protective effect are unclear. Statins have been reported to have multiple potentially important protective mechanisms including preservation of cerebral blood flow by upregulation of eNOS, decrease in inflammatory cytokine induction, alteration in glutamate receptor function and decrease in apoptotic cell death. The importance of these results, if confirmed in models of trauma and ischemia, are that statins, already commonly used in the clinic, might offer protection for patients in situations where END is expected such as patients undergoing cranial surgery, patients at risk for ischemia and even soldiers at risk for the effects of blast injury brain trauma.

SATURDAY, NOVEMBER 7

11:47 – 11:59 AM **LENGTH OF STAY AND TOTAL HOSPITAL COSTS OF CLIPPING VERSUS COILING FOR RUPTURED AND UNRUPTURED CEREBRAL ANEURYSMS IN THE NATIONWIDE INPATIENT SAMPLE DATABASE: 2002-2006**

Brian L. Hoh, M.D., Yueh-Yun Chi, Ph.D., Matthew F. Lawson, M.D., J. Mocco, M.D., Fred G. Barker II, M.D.

INTRODUCTION: We have previously reported the effect of clipping versus coiling of cerebral aneurysms on length of stay and hospital costs at our single institution. We now conducted this analysis at a national level using the Nationwide Inpatient Sample (NIS) Database.

METHODS: We obtained the NIS from the Healthcare Cost and Utilization Project, Agency for Healthcare Quality and Research. The NIS is the largest all-payer inpatient care database in the United States and represents approximately 20% of all inpatient admissions to nonfederal hospitals. Hospitalizations for clipping or coiling of ruptured and unruptured cerebral aneurysms from 2002-2006 were collected from the NIS by cross-matching ICD-9 codes for subarachnoid hemorrhage (430) or unruptured cerebral aneurysm (437.3) with clipping of aneurysm (39.51) or coiling (39.79; 39.72; or 39.52). Length of hospital stay and total hospital costs for clipping and coiling were compared using multivariate linear models adjusted for the following factors: gender, race, admission source, admission type, median income level of zip code, payor, and comorbidities.

RESULTS: There were a total of 9635 hospitalizations for ruptured aneurysms (6019 clipping, 3616 coiling); and 9399 hospitalizations for unruptured aneurysms (4700 clipping, 4699 coiling). For ruptured aneurysm patients, in the multivariate analysis, clipping was associated with significantly longer length of stay (19.9±16.1 vs. 17.9±14.1 days, $P<0.0001$), but similar total hospital costs ($173,197±$133,622 vs. $174,443±$136,707, $P=0.31$) compared to coiling, respectively. For unruptured aneurysm patients, in the multivariate analysis, clipping was associated with significantly longer length of stay (9.2±10.5 vs. 4.5±7.5 days, $P<0.0001$), and significantly higher total hospital costs ($82,726±$85,051 vs. $68,750±$67,217, $P=0.001$) compared to coiling, respectively.

CONCLUSIONS: The results of this nationwide analysis differed from the findings of our single institution study. Clipping is associated with longer lengths of stay for both ruptured and unruptured aneurysm patients, and higher total hospital costs for unruptured aneurysm patients, compared to coiling.

SATURDAY, NOVEMBER 7

12:01 – 12:13 PM **UNDERSTANDING AND ELIMINATING ONCOGENIC EGFR SIGNALING IN MALIGNANT GLIOMAS**

Jaime Acquaviva, Ph.D., Abraham Boskovitz, M.D, and ***Al Charest, M.Sc., Ph.D.***

BACKGROUND: Malignant gliomas account for the majority of primary brain cancers and are considered one of the most insidious malignancies in humans. Our research focuses on the use of animal models of glioblastoma multiforme (GBM) to understand the molecular and genetic contributors to disease initiation, maintenance and resistance to therapies. The epidermal growth factor receptor (EGFR) signaling pathway plays a crucial role in GBM pathogenesis: initiating the early stages of tumor development, sustaining tumor growth, promoting infiltration and mediating resistance to therapy. The importance of this pathway is highlighted in the fact that EGFR is mutationally activated in >50% of GBM tumors.

METHODS: Consistent with this observation, we have created a genetically engineered conditional EGFR mouse model of GBM and established that EGFR overexpression in adult mice promotes the development of GBMs. We hypothesized that specific gene mutations will confer resistance to targeted therapeutic agents and by eliminating the responsible gene(s), we can sensitize tumors to those drugs.

RESULTS: Using cells established from these GBM tumors we demonstrated that sensitivity to tyrosine kinase inhibitors (TKI) is dictated by the genetic makeup of the tumors. We performed cell growth and apoptosis assays on these cells and demonstrated, for example, that Iressa treatment of EGFR positive and PTEN null tumor cells result in cytostaticity whereas treatment of EGFR positive tumor cells is a cytotoxic effect. To better understand these differences at the molecular lever, we studied these results in the context of global phospho-proteomic analysis. We performed phospho-tyrosine proteomic scans using mass spectrometry on treated versus non treated cells and established system network pathways that relate to the observed behaviors of the cells.

CONCLUSIONS: Lack of PTEN expression directs EGFR signaling networks towards pathways that are no longer responsive to TKI action. In deciphering signaling components of TKI resistance, we strive to identify and overcome key members that are responsible for drug treatment resistance.

NOTES

SPECIAL GUESTS

Guests	Sponsors
Aviva Abosch Minneapolis, MN	Stephen Haines
Ron Alterman New York, NY	Richard Winn
Mustafa Baskaya Madison, WI	Robert Dempsey
Bernard Bendok Chicago, IL	Hunt Batjer
Alan Boulos Albany, NY	John Popp
Richard Byrne Chicago, IL	Vincent Traynelis
Al Charest Boston, MA	Carl Heilman
Michael Chicoine St. Louis, MO	Robert Grubb
Austin Colohan Loma Linda, CA	John Jane, Sr.
John Day San Antonio, TX	Howard Riina
Ian Dorward St. Louis, MO	Ralph Dacey
Constantinos Hadjipanayis Atlanta, GA	Academy Award Winner
Brian Hoh Gainesville, FL	William Friedman
Frederick Lang	Raymond Sawaya

Houston, TX

Joung Lee
Cleveland, OH

Johnny Delashaw

Maciej Lesniak
Chicago, IL

Issam Awad

Allan Levi
Miami, FL

Robero Heros

Charles Liu
Los Angeles, CA

Steven Giannotta

Michael McDermott
San Francisco, CA

Mitchel Berger

Joshua Medow
Madison, WI

Benny Iskandar

Ehud Mendel
Columbus, OH

Ennio Chiocca

Charles Prestigiacomo
New Jersey

Peter Carmel

Nader Sanai
Phoenix, AZ

Academy Award Runner-up

Andrew Sloan
Cleveland, OH

Warren Selman

Phillip Tibbs
Lexington, KY

Russell Travis

Clarence Watridge
Memphis, TN

Martin Camins

Michael Weaver
Philadelphia, PA

Christopher Loftus

Gregory Zipfel
St. Louis, MO

Ralph Dacey

ACADEMY AWARD WINNERS

Paul M. Lin 1955
Hubert L. Rosomoff 1956
Byron C. Pevehouse 1957
Norman Hill 1958
Jack Stern 1959
Robert Ojemann 1960
Lowell E. Ford 1962
Charles H. Tator 1963
Earle E. Crandall 1964
Stephen Mahaley, Jr. 1965
Chun Ching Kao 1966
John P. Kapp 1967
Yoshio Hosobuchi 1968
Gary G. Ferguson 1970
Richard L. Pressley 1971
David G. McLone 1972
Arden F. Reynolds, Jr. 1973
Richard L. Rapport 1974
Andrew G. Shetter 1975
John R. Howe 1976
Howard W. Blume 1977
Howard J. Senter 1978
Elisabeth M. Post 1979
David Dubuisson 1980
Dennis A. Turner 1981
Marc R. Mayberg 1982
David S. Baskin 1983
Kevin J. Kiwak 1984
Terry Lichtor 1985
Michael G. Nosko 1986
Joseph R. Madsen 1987
James T. Rutka 1988
Christopher D. Heffner 1989
Scott I. Gingold 1990
Mary Louise Hlavin 1991
Adam P. Brown 1992
Michael Tymianski 1993
David Garrett, Jr. 1994
L. Brannon Thomas 1995
John S. Yu 1996
Gregory Canute 1997
Nathan R. Selden 1998
Robert M. Friedlander 1999
Tien T. Nguyen 2000
Peng Chen 2001
Ganesh Rao 2002
Gelarch Zadeh 2003

Eric C. Leuthardt 2004
Manish Aghi 2005
Alfred T. Ogden 2006
Paul Kongkham 2007
Elias Rizk 2008
Costas Hadjipanayis. 2009

MEETINGS OF THE ACADEMY

Hotel Netherland Plaza, Cincinnati, Ohio …….October 28-29, 1938
Roosevelt Hotel, New Orleans, Louisiana …… October 27-29, 1939
Tudor Arms Hotel, Cleveland, Ohio ……..October 21-22, 1940
Mark Hopkins Hotel, San Francisco, California …… November 11-15, 1941
Ambassador Hotel, Los Angeles, California ……...November 11-15, 1941
The Palmer House, Chicago, Illinois ……..October 16-17, 1942
Hart Hotel, Battle Creek, Michigan…… September 17-18, 1943
Ashford General Hospital, White Sulphur Springs,
West Virginia ……..September 7-9, 1944
The Homestead, Hot Springs, Virginia ……September 9-11, 1946
Broadmoor Hotel, Colorado Springs,
Colorado ……October 9-11, 1947
Windsor Hotel, Montreal, Canada ……..September 20-22, 1948
Benson Hotel, Portland, Oregon …….October 25-27, 1949
Mayo Clinic, Rochester, Minnesota…… September 28-30, 1950
Shamrock Hotel, Houston, Texas ……October 4-6, 1951
Waldorf-Astoria Hotel, New York City,
New York ……..September 29-October 1, 1952
Biltmore Hotel, Santa Barbara, California ……October 12-14, 1953
Broadmoor Hotel, Colorado Springs, Colorado ……October 21-23, 1954
The Homestead, Hot Springs, Virginia …….October 27-29, 1955
Camelback Inn, Phoenix, Arizona ……..November 8-10, 1956
The Cloister, Sea Island, Georgia …….November 11-13, 1957
The Royal York Hotel, Toronto, Canada ……. November 6-8, 1958
Del Monte Lodge, Pebble Beach, California ……October 18-21, 1959
Copley Sheraton Plaza, Boston, Massachusetts ……October 5-8, 1960
Royal Orleans, New Orleans, Louisiana ……..November 7-10, 1962
El Mirador, Palm Springs, California ……October 23-26, 1963
The Key Biscayne, Miami, Florida…….November 11-14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio …….October 14-16, 1965
Fairmont Hotel & Towers, San Francisco,
California ……October 17-19, 1966
The Key Biscayne, Miami, Florida ……..November 8-11, 1967
Broadmoor Hotel, Colorado Springs, Colorado …….October 6-8, 1968
St. Regis Hotel, New York City ……..September 21, 1969
Camino Real, Mexico City, Mexico…… November 18-21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada ……September 26-30, 1971
New College, Oxford, England ……September 4-7, 1972
Huntington-Sheraton Hotel, Pasadena, California ……November 14-17, 1973
Southampton Princess Hotel, Bermuda…… November 6-9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona ……...November 5-8, 1975
Mills Hyatt House, Charleston, South Carolina …….November 10-13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii …….November 2-5, 1977
Hotel Bayerischer Hof, Munich, Germany ……October 22-25, 1978
Hyatt Regency, Memphis, Tennessee …….November 7-10, 1979
Waldorf-Astoria Hotel, New York City, New York …….October 1-4, 1980
Sheraton Plaza, Palm Springs, California …….November 1-4, 1981

Ritz-Carlton Hotel, Boston, Massachusetts …….. October 10-13, 1982
The Lodge at Pebble Beach, California …….October 23-26, 1983
The Homestead, Hot Springs, Virginia ……..October 17-20, 1984
The Lincoln Hotel Post Oak, Houston, Texas ……October 27-30, 1985
The Cloister, Sea Island, Georgia ……..November 5-8, 1986
Hyatt Regency, San Antonio, Texas……October 7-10, 1987
Omni Netherland Plaza, Cincinnati, Ohio …….September 13-17, 1988
Loews Ventana Canyon, Tucson,
Arizona …….September 27-October 1, 1989
Amelia Island Plantation, Amelia Island, Florida ……..October 2-7, 1990
Salishan Lodge, Gleneden Beach, Oregon …….September 22-26, 1991
Ritz-Carlton Hotel, Naples, Florida ……October 21-25, 1992
The Wigwam, Phoenix, Arizona …….October 27-30, 1993
The Cloister, Sea Island, Georgia ……November 3-6, 1994
Loews Ventana Canyon Resort, Tucson, Arizona ……..November 1-5, 1995
The Greenbrier, White Sulphur Springs,
West Virginia ……September 18-22, 1996
Rimrock Resort, Banff, Alberta, Canada …….September 10-14, 1997
Four Seasons Biltmore, Santa Barbara, California ……..November 4-7, 1998
Ritz-Carlton, Amelia Island, Florida ……...November 10-13, 1999
The Broadmoor, Colorado Springs, Colorado …….October 11-14, 2000
The Breakers, Palm Beach, Florida……..November 14-17, 2001
The Phoenician, Scottsdale, Arizona . . . . . ……October 16-19, 2002
Colonial Williamsburg, Williamsburg, VA …….October 29-November 1, 2003
Four Seasons Berlin and
Taschenbergpalais Dresden Germany . . . . . ……October 3-8, 2004
Ritz-Carlton, Half Moon Bay, California . . . . . ……September 21-24, 2005
Ritz-Carlton, Reynolds Plantation, Greensboro, GA …..…… October 18-21, 2006
Ritz-Carlton, Lake Las Vegas, Nevada ……...October 31-November 3, 2007
Barrow Neurological Institute Phoenix and
Echantment Resort, Sedona Arizona ……September 10-13, 2008
The Breakers, Palm Beach, Florida ……November 4-7, 2009

PAST PRESIDENTS

Dean H. Echols..... 1938-39
Spence Braden..... 1940
Joseph P. Evans..... 1941
Francis Murphey 1942
Frank H. Mayfield..... 1943
A. Earl Walker 1944
Barnes Woodhall..... 1946
William S. Keith 1947
Howard A. Brown..... 1948
John Raaf..... 1949
E. Harry Botterell..... 1950
Wallace B. Hamby 1951
Henry G. Schwartz..... 1952
J. Lawrence Pool..... 1953
Rupert B. Raney..... 1954
David L. Reeves..... 1955
Stuart N. Rowe..... 1956
Arthur R. Elvidge..... 1957
Jess D. Herrmann 1958
Edwin B. Boldrey..... 1959
George S. Baker 1960
C. Hunter Shelden 1961-62
Samuel R. Snodgrass . 1963
Theodore B. Rasmussen1964
Edmund J. Morrissey 1965
George Maltby 1966
Guy L. Odom 1967
James G. Galbraith 1968
Robert H. Pudenz . 1969-70
William B. Scoville ... 1971
Robert L. McLaurin ... 1972
Lyle A. French 1973
Benjamin B. Whitcomb1974
John R. Green..... 1975
William H. Feindel 1976
William H. Sweet..... 1977
Arthur A. Ward 1978
Robert B. King 1979
Eben Alexander, Jr. ... 1980
Joseph Ransohoff II ... 1981
Byron C. Pevehouse .. 1982
Sidney Goldring 1983
Russel H. Patterson, Jr.1984
Thomas Langfitt..... 1985
Phanor L. Perot, Jr..... 1986
Shelley N. Chou 1987
James T. Robertson ... 1988
Thoralf M. Sundt, Jr. . 1989
Robert Ojemann 1990
Nicholas Zervas..... 1991
Henry Garretson 1992
George Tindall..... 1993
William A. Buchheit .. 1994
David L. Kelly, Jr. 1995
John M. Tew, Jr. 1996
Julian T. Hoff 1997
Edward Connolly..... 1998
J. Charles Rich..... 1999
George A. Ojemann …2000
Roberto C. Heros.……2001
Donald O. Quest……..2002
David G. Piepgras……2003
Volker K.H. Sonntag……...2004
Martin B. Camins……2005
L. Nelson Hopkins……...2006
Richard Morawetz……...2007
Robert F. Spetzler……2008
Ralph G. Dacey, Jr……...2009

PAST VICE-PRESIDENTS

Francis Murphey..... 1941
William S Keith..... 1942
John Raaf..... 1943
Rupert B Raney 1944
Arthur R Elvidge 1946
F Keith Bradford..... 1949
David L Reeves 1950
Henry G Schwartz..... 1951
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
Augustus McCravey.... 1969-70
Edward W Davis..... 1971
John R Green..... 1972
George J Hayes..... 1973
Richard L DeSaussure..... 1974
Ernest W Mack..... 1975
Frank E Nulsen..... 1976
Robert S Knighton..... 1977
Robert G Fisher 1978
H Thomas Ballantine, Jr.... 1979
George Ehni..... 1980
Courtland H Davis, Jr 1981
John F Mullan..... 1982
Hugo V Rizzoli..... 1983
James W Correll 1984
E Bruce Hendrick 1985
Griffith R Harsh, III 1986
Ellis B Keener..... 1987
Robert Grossman 1988
Jim Story 1989
John Jane, Sr..... 1990
Stewart Dunsker 1991
Burton M Onofrio..... 1992
Martin H Weiss..... 1993
John M Tew, Jr..... 1994
John C VanGilder 1995
Edward Connolly..... 1996
George Ojemann..... 1997
Charles H Tator 1998
Donald O Quest 1999
Howard M. Eisenberg 2000
Richard B. Morawetz……...2001
Martin B. Camins…….....2002
Arthur L. Day……2003
William F. Chandler…….2004
Steven L. Gianotta……2005
Robert F. Spetzler…….2006
Griffith R. Harsh IV……..2007
Ralph Dacey, Jr…….2008
M. Sean Grady ..……...2009

PAST SECRETARY-TREASURERS

Francis Murphey..... 1938-40
A. Earl Walker..... 1941-43
Theodore C. Erickson .. 1944-47
Wallace B. Hamby..... 1948-50
Theodore B. Rasmussen1951-53
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
Ralph G. Dacey, Jr…….2004-07
James Rutka……2008-11

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
James T. Rutka…….....2004-07
Griffith Harsh…….2008-11

HONORARY MEMBERS Elected

GUY LAZORTHES (Annick) 1973
Home: 5 Allee Charles Malpel
31300 Toulouse
FRANCE
Tel: 33-5-34-513215

KEIJI SANO (Yaeko) 1975
Office: Tokyo Neuro-Center
MT Building 2F
4-1-17 Toranomon
Minato-ku, Tokyo 105-0001
JAPAN
Tel: 81-3-5776-1200
Fax: 81-3-5776-1201
E-mail:none

SENIOR MEMBERS Elected

JAMES AUSMAN (Carolyn).....1979
69-844 Highway 111, Suite C
Rancho Mirage CA 92270
760-770-4646, fax 760-770-4647, jamesausman@mac.com

DONALD BECKER (Maria).....1990
Division of Neurosurgery, Room 74-129
UCLA Medical Center, box 956901
10833 Le Conte Avenue
Los Angeles, CA 90095-6901
310-825-3998, fax 310-794-5836, dbecker@mednet.ucla.edu

GILLES BERTRAND (Louise).....1967
Montreal Neurological Institute
3801 University Street, #109
Montreal, Quebec H3A 2B4
CANADA
514-398-1935, fax 514-398-2811, bertrandgilles@videotron.ca

JERALD BRODKEY (Arielle).....1977
13901 Shaker Boulevard
Cleveland, OH 44120
216-752-4545, fax 216-752-9455, jsb@brodkey.com

WILLIS BROWN, JR. (Elizabeth {Ann}).....1984
7523 Shadylane Drive
San Antonio, TX 78209
210-828-0023, fax 210-828-0385, willis_brown@sbcglobal.net

WILLIAM BUCHHEIT (Christa).....1980
6014 Cricket Road
Flourtown PA 19031
215-836-9295, fax 215-836-4634, wbuchheit@aol.com

MARTIN CAMINS (Joan).....1995
Neurological Surgery, Suite T1-C
205 East 68th Street
New York, NY 10065
212-570-0100, fax 212-570-0117, martin_camins@msn.com

PETER CARMEL (Jacqueline Bello).....1991
Neurosurgery, Suite 8100
New Jersey Medical School
90 Bergen Street
Newark, NJ 07103
973-972-2335, fax 973-972-8553, carmel@umdnj.edu

WILLIAM CHANDLER (Susan) 1989
Department of Neurosurgery, SPC 5338
University of Michigan Health System
3552 Taubman Health Care Center
1500 East Medical Center Drive
Ann Arbor, MI 48109-5338
734-936-5020, fax 734-936-9294, wchndlr@umich.edu

PAUL CHAPMAN 1983
Neurosurgery, Suite 745
Massachusetts General Hospital
15 Parkman Street
Boston, MA 02114
617-726-3887, chapman@helix.mgh.harvard.edu

WILLIAM COLLINS, JR......1963
11948 Adorno Place
San Diego, CA 92128
858-673-9025, wfcollin@aol.com

EDWARD CONNOLLY (Elise).....1972
18 Richmond Place
New Orleans, LA 70115
504-891-1159, fax 504-891-1128, escelc@bellsouth.net

PAUL COOPER (Leslie) 1995
320 East 72nd Street
New York, NY 10021
212-288-6778, paul.cooper@nyumc.org

COURTLAND DAVIS, JR. (Carrie).....1967
2525 Warwick Road
Winston-Salem, NC 27104-1943
336-723-7296, chdccdmd@triad.rr.com

DONALD DOHN (Carolyn).....1968
P.O. Box 998
Point Clear, AL 36564
251-928-7670, fax 251-928-7670 (call first), dohn@mchsi.com

STEWART DUNSKER (Ellen).....1975
551 Abilene Trail
Cincinnati, OH 45215
513-522-0330, fax 513-522-0333, dunsker@aol.com

MICHAEL EDWARDS (Linda Laughlin) 1992
Neurosurgery, Room R-211
Stanford University Medical Center
300 Pasteur Drive
Stanford, CA 94305-5327

650-497-8775, fax 650-725-5086, edwards9@stanford.edu

HOWARD EISENBERG (Doris Zografos) 1985
Neurosurgery, Suite 12D South
22 South Greene Street
Baltimore, MD 21201
410-328-3514, fax 410-328-1420, heisenberg@smail.umaryland.edu

MEL EPSTEIN (Lynn) 1992
411 Poppasquash Road
Bristol, RI 02809
401-254-5083, fax 401-253-6422, melepstein@earthlink.net

WILLIAM FEINDEL (Faith).....1959
Montreal Neurological Institute
3801 University Street
Montreal, Quebec H3A 2B4
CANADA
514-398-1939, fax 514-398-1375, william.feindel@bic.mcgill.ca

EUGENE FLAMM (Susan).....1979
Department of Neurosurgery
Montefiore Medical Center
Bronx, NY 10467
718-920-2339, fax 718-515-8235, eflamm@montefiore.org

ELDON FOLTZ (Catherine).....1960
2480 Monaco Drive
Laguna Beach CA 92651
949-494-3422, fax 949-494-8947, eldonfoltz@gmail.com

RICHARD FRASER (Sara Anne).....1976
75 Holly Hill Lane
Greenwich, CT
914-967-6867, safraser50@aol.com

JOHN GARNER (Candace) ……1971
2834 Dove Run Creek Drive
Las Vegas, NV 89135
702-243-3592, jtgrex@aol.com

PHILIP GORDY.....1968
3601 Carmel Drive
Casper, WY 82604-4949
307-265-7883, philipgordy@aol.com

ROBERT GROSSMAN (Ellin).....1984
Department of Neurosurgery
The Methodist Hospital
6560 Fannin, Suite 944
Houston, TX 77030

713-441-3810, fax 713-793-1004, rgrossman@tmhs.org

ROBERT GRUBB, JR. (Julia) 1985
Department of Neurological Surgery, Box 8057
Washington University Medical Center
660 South Euclid Avenue
St. Louis, MO 63110
314-362-3567, fax 314-362-2107, grubbr@nsurg.wustl.edu

JOSEPH HAHN (Andrea).....1993
Neurosurgery/H18
The Cleveland Clinic Foundation
9500 Euclid Avenue
Cleveland, OH 44195-1004
216-444-5802, fax 216-445-7100, hahnj@ccf.org

GRIFFITH HARSH, III (Craig) 1980
27 Arlington Avenue, # 24
Birmingham, AL 35205
205-933-2376, gharsh3@aol.com

MARK PETER HEILBRUN (Robyn).....1984
P.O.Box 920041
Snowbird, UT 84092
801-742-0516, fax 801-742-0516 (call first), mpeterheilbrun@msn.com

ROBERTO HEROS (Deborah) 1985
Department of Neurosurgery
University of Miami
1095 NW 14th Terrace
Miami, FL 33136
305-243-4572, fax 305-243-3180, rheros@med.miami.edu

CHARLES HODGE, JR. (Cathy) 1982
46 Harrison Street
Johnson City, NY 13790
607-729-4942, hodgec@upstate.edu

L. NELSON (NICK) HOPKINS, III (Ann {Bonnie}) ..1992
University at Buffalo Neurosurgery
Millard Fillmore Gates Hospital, Kaleida Health
3 Gates Circle
Buffalo, NY 14209
716-887-5200, fax 716-887-4378, lnhbuffns@aol.com

EDGAR HOUSEPIAN (Marion) 1976
The Neurological Institute
710 West 168th Street
New York, NY 10032
212-305-5256, fax 212-305-3250, emh4@columbia.edu

ALAN HUDSON (Susan).....1978
Cancer Care Ontario
620 University Avenue
Toronto, Ontario M5G 2L7
CANADA
416-971-9800 x1610, alan.hudson@cancercare.on.ca

JOHN JANE, SR. (Noella).....1982
Department of Neurosurgery
University of Virginia Health System
PO Box 800212
Charlottesville, VA 22908
434-982-3244, fax 434-243-2954, jaj6r@virginia.edu

PETER JANNETTA (Diana) 1994
Neurosurger, Suite 302
Allegheny General Hospital
420 East North Avenue
Pittsburgh, PA 15212
412-359-6200, fax 412-359-4811, pjannett@wpahs.org

ELLIS KEENER (Ann) 1978
915 East Lake Drive
Gainesville, GA 30506
770-532-5616, ebkeener@bellsouth.net

DAVID KELLY, JR. (Sarah {Sally}).....1975
Department of Neurosurgery
Wake Forest University
Baptist Medical Center
Medical Center Boulevard
Winston-Salem, NC 27157-1029
336-716-4049, fax 336-716-3065, dkelly@wfubmc.edu

PATRICK KELLY (Carol).....1992
Neurosurgery, 7S
Bellevue Medical Center
465 First Avenue
New York, NY 10016
212-263-6416, fax 212-263-8225, kellyp01@med.nyu.edu

GLENN KINDT (Charlotte).....1977
Neurosurgery, Box C307
University of Colorado
12631 East 17th Avenue
Denver, CO 80045
303-724-2292, fax 303-724-2300, glenn.kindt@ucdenver.edu

WOLFF KIRSCH (Marie-Claire).....1971
Neurosurgery Center for Research, Training, and Education
Loma Linda University
11175 Campus Street, Suite 11113
Loma Linda, CA 92350
909-558-7070, fax 909-558-0472, wkirsch@llu.edu

DAVID KLINE (Helen {Nell}).....1971
Department of Neurological Surgery
Louisiana State Univ. Health Science Center
2020 Gravier Street
New Orleans, LA 70112
504-568-6120,dkline@lsuhsc.edu

SANFORD LARSON (Jacquelyn)……1989
Department of Neurosurgery
Medical College of Wisconsin
9200 West Wisconsin Avenue
Milwaukee, WI 53226
414-805-5407

EDWARD LAWS (Margaret {Peggy}) 1983
Department of Neurosurgery, PBB3
Brigham & Women's Hospital
15 Francis Street
Boston, MA 02115
617-732-6600, fax 617-264-5114, elaws@partners.org

RAEBURN LLEWELLYN (Carmen Rolon).....1963
Unit 8B
3 Poydras Street
New Orleans, LA 70130-1665
504-523-3909, fax 504-649-9265

DON LONG (Harriett).....1983
Neurosurgery, Carnegie 466
The Johns Hopkins Hospital
600 North Wolfe Street
Baltimore, MD 21287-7709
410-614-3536, fax 410-955-6407, dmlong@jhmi.edu

L. DADE LUNSFORD (Julianne {Julie}) 1992
Neurosurgery, B-400
Univ. of Pittsburgh Medical Center
200 Lothrop Street
Pittsburgh, PA 15213
412-647-6781, fax 412-647-6483, lunsfordld@upmc.edu

ROBERT MARTUZA (Susan {Jill}).....1989
Neurosurgery Service/GRB 502
Massachusetts General Hospital
55 Fruit Street
Boston, MA 02114
617-726-8583, fax 617-643-0669, rmartuza@partners.org

ROBERT MAXWELL (Karen).....1992
12037 Brassie Circle #201
Fort Meyers, FL 33913
23-245-8439, fax same (call first), max2wally@yahoo.com

J. GORDON MCCOMB (Rhoda) 1998
Neurosurgery, Suite 1006
Children's Hospital of Los Angeles
1300 North Vermont Avenue
Los Angeles, CA 90027
323-663-8128, fax 323-363-3101, gmccomb@chla.usc.edu

ROBERT MCLAURIN (Sarah {Sally}) 1955
2412 Ingleside Avenue, 5C
Cincinnati, OH 45206
513-281-9782, McLaurin@one.net

RICHARD MORAWETZ (Mary Jean) 1990
1002 Faculty Office Tower
510 Twentieth Street South
Birmingham, AL 35294-3410
205-934-2918, fax 205-996-4674, mmorawetz@aol.com

JOHN MULLAN (Vivian).....1963
5844 Stony Island Avenue
Chicago, IL 60637
773-241-6546, jandvmullan@comcast.net

BLAINE NASHOLD, JR. (Irene).....1967
2701 Pickett Road, Apt. 4042
Durham, NC 27705-5653
919-489-9728, nasho002@aol.com

PAUL NELSON (Teresa).....1991
Neurosurgery, Emerson Hall 139
Indiana University
545 Barnhill Drive
Indianapolis, IN 46202
317-274-5725, fax 317-274-7351, pnelson1@iupui.edu

GEORGE OJEMANN (Linda).....1975
Neurological Surgery, Box 356470
University of Washington
1959 N.E. Pacific Street
Seattle, WA 98195-6470
206-543-3570, fax 206-543-8315, gojemann@u.washington.edu

ROBERT OJEMANN (Jean).....1968
85 Nobscot Road
Weston, MA 02493
781-899-3146, fax781-891-5336, rojemann@mindspring.com

EDWARD OLDFIELD (Susan) 1975
Department of Neurosurgery
P.O.Box 800212
University of Virginia Health System
Charlottesville, VA 22908
434-982- 0059, fax 434-924-9069, blc2v@virginia.edu

ANDRE OLIVIER (Nicole Poulin) 1989
Division of Neurosurgery
Montreal Neurological Hospital
3801 University Street, #109
Montreal, Quebec H3A 2B4
CANADA
514-398-1937, fax 514-398-2811, andre.olivier@mcgill.ca

BURTON ONOFRIO (Judith).....1975
1105 Tenth Street SW
Rochester, MN 55902
507-289-3684, fax 507-529-9469

TAE SUNG PARK (Meeaeng).....1996
Department of Neurosurgery
St. Louis Children's Hospital
One Children's Place
St. Louis, MO 63110
314-454-2810, fax 314-454-2818, park@wustl.edu

RUSSEL PATTERSON, JR. (Juliet {Julie}).....1971
Apartment #65A
146 West 57th Street
New York, NY 10019-3301
212-586-9237, fax 212-315-3877, patt10019@verizon.net

SYDNEY PEERLESS (Ann).....1977

2721 Hibiscus Court
Punta Gorda, FL 33950
941-833-5710, fax (same #), speerless@earthlink.net

PHANOR PEROT, JR.(Sandra) 1970
Neurosurgery, Suite 428
Medical Univ. of South Carolina
6 Jonathan Lucas Street
Charleston, SC 29425
843-792-7700, fax 843-792-0468, perotplj@musc.edu

BYRON CONE PEVEHOUSE (Lucy).....1964
13623 N.E. 32nd Place
Bellevue, WA 98005-1400
425-881-5531, fax 425-881-5831, byron.pevehouse@verizon.net

DAVID PIEPGRAS (Jane) 1987
Department of Neurologic Surgery
Mayo Clinic, Gonda 8-209
200 First Street SW
Rochester, MN 55905
507-284-2254, fax 507-284-5206, piepgras.david@mayo.edu

LAWRENCE PITTS (Mary).....1997
Interim Executive Vice President of Academic Affairs
UC Office of the President
1111 Franklin Street
Oakland, CA 94607
510-987-9020, lawrence.pitts@ucop.edu

ROBERT PORTER (Dean).....1962
6461 Bixby Hill Road
Long Beach, CA 90815
562-430-0788, rporter785@aol.com

KALMON POST (Linda Farber-Post).....1995
Neurosurgery, Box 1136
Mount Sinai Medical Center
One Gustave L. Levy Place
New York, NY 10029
212-241-0933, fax 212-423-9285, kalmon.post@mountsinai.org

DONALD QUEST (Ilona).....1986
Department of Neurological Surgery
The Neurological Institute, 4-440
710 West 168th Street
New York, NY 10032
212-305-5582, fax 212-305-2026, doq1@columbia.edu

ROBERT RATCHESON (Peggy).....1986
Department of Neurosurgery
University Hospitals of Cleveland
11100 Euclid Avenue
Cleveland, OH 44106
216-368-3360 or 216-844-3472, rar@case.edu

ALBERT RHOTON, JR. (Joyce).....1984
Department of Neurosurgery
University of Florida
PO Box 100265
Gainesville, FL 32610
352-273-9000, fax 352-392-8413, rhoton@neurosurgery.ufl.edu

J. CHARLES RICH, JR. (Jasmine) 1987
25 Columbia Drive (winter)
Rancho Mirage, CA 92270
760-324-2010, jcrichnsur@aol.com

2397 East 1300 South (summer)
Salt Lake City, UT 84108
801-583-4822

HUGO RIZZOLI 1973
Apartment 102
5100 Dorset Avenue
Chevy Chase MD 20815
301-654-6486, fax 301-654-3018, hrizzoli@comcast.net

JAMES ROBERTSON (Valeria) 1971
189 Crestview Drive
Brevard, NC 28712
828-884-4934, fax 828-884-4934, jrober52@gmail.com

JON ROBERTSON (Carol Anne) 1992
Neurosurgery, Suite 200
Semmes-Murphey Clinic
1211 Union Avenue
Memphis, TN 38104
901-259-5335, fax 901-259-5300, jrobertson@semmes-murphey.com

DUKE SAMSON (Patricia Bergen) 1994
Department of Neurological Surgery
Univ. of Texas, Southwestern Med. School
5323 Harry Hines Boulevard
Dallas, TX 75390
214-648-4551, fax 214-648-2282, dukesamson@utsouthwestern.edu

R. MICHAEL SCOTT (Susan)…….1991
Department of Neurosurgery

The Children's Hospital
300 Longwood Avenue
Boston, MA 02115
617-355-6011, fax 617-730-0906, michael.scott@childrens.harvard.edu

EDWARD SELJESKOG (Peg).....1992
Neurosurgical Associates
4141 5th Street
Rapid City, SD 57701-6021
605-341-2424, fax 605-341-4547, edskog@msn.com

CHRISTOPHER SHIELDS (Deborah) 1993
Department of Neurosurgery, Suite 1102
University of Louisville
210 East Gray Street
Louisville, KY 40202
502-629-5510, fax 502-629-5512, cbshields1@gmail.com

WILLIAM SHUCART (Laura).....1989
250 Beacon Street
Boston, MA 02116
617-267-1038, fax 617-636-7587, william.shucart@bmc.org

FREDERICK SIMEONE …………1981
6825 Norwitch Drive
Philadelphia, PA 19153
215-816-7000, fax 215-365-8230, fasimeone@comcast.net

JAMES SIMMONS (Vanita).....1975
190 South Grove Park Road
Memphis, TN 38117
901-522-7700

KENNETH SMITH, JR. (Marjorie).....1987
Division of Neurosurgery
St. Louis University
3635 Vista Avenue at Grand Boulevard
St. Louis, MO 63110-0250
314-577-8795, fax 314-577-8720, smithj5@slu.edu

VOLKER SONNTAG (Lynne).....1995
Barrow Neurosurgical Associates
2910 North Third Avenue
Phoenix, AZ 85013
602-406-3458, fax 602-406-6110, Debbie.nagelh@bnaneuro.net

DENNIS SPENCER (Susan) 1989
Department of Neurosurgery, TMP4

Yale University School of Medicine
333 Cedar Street
New Haven, CT 06520-8082
203-785-2285, fax 203-785-4161, dennis.spencer@yale.edu

BENNETT STEIN (Bonita).....1970
411 Claremont Road
Bernardsville, NJ 07924
908-696-0293, fax 908-696-0283

JIM STORY (Joanne).....1972
3135 Stonehaven Road
San Antonio, TX 78230
210-344-9082, fax 210-344-3633, jlstory@swbell.net

RONALD TASKER.....1971
Division of Neurosurgery, 4W-437
Toronto Western Hospital
399 Bathurst Street
Toronto, Ontario M5T 2S8
Canada
416-603-5771, fax 416-603-5298

CHARLES TATOR (Carol) 1991
Neurosurgery, Suite 4W-433
Toronto Western Hospital
399 Bathurst Street
Toronto, Ontario M5T 2S8
Canada
416-603-5889, fax 416-603-5298, charles.tator@uhn.on.ca

JOHN TEW, JR. (Susan).....1971
Mayfield Clinic, Suite #3100
222 Piedmont Avenue
Cincinnati, OH 45219
513-475-8643, fax 513-475-8664, jtew@mayfieldclinic.com

GEORGE TINDALL [Elizabeth Barringer(Wendy)] 1968
Mid Georgia Nursery
227 Rose Hill Road
Meansville, GA 30256
770-567-3874, fax 770-567-3746, gtindall@midgeorgiansy.com

RUSSELL TRAVIS (Jill).....1994
2343 Alexandra Drive
Lexington, KY 40504
859-224-2006, fax 859-224-2005, rltravis@mac.com

JOHN TYTUS (Virginia).....1967
3827 East Crockett Street
Seattle, WA 98112
206-325-9552

CLARK WATTS (Patricia).....1975
5922 Northwest Place
Austin, TX 78731
512-323-2295, fax 512-323-5891, cwattsmdjd@msn.com

BRYCE WEIR (Mary Lou).....1984
1262 Saturna Drive
Parksville, BC V9P 2X6
CANADA
250-951-2192, bkaweir@shaw.ca

MARTIN WEISS (Debby).....1981
Neurosurgery, Suite 5046
USC Medical Center
1200 North State Street
Los Angeles, CA 90033
323-226-7421, fax 323-226-7833, weiss@usc.edu

LOWELL WHITE, JR......1971
11009 East Villa Monte Drive
Mukilteo, WA 98275
425-315-8030, bud.white@verizon..net

ROBERT WILKINS (Gloria).....1973
Box 3807
Duke University Medical Center
Durham NC 27710
919-684-3034, rhwilkins@aol.com

CHARLES WILSON (Francie Petrocelli).....1966
3881 Washington Street
San Francisco, CA 94118
415-831-7449, fax 415-831-1947, cwilson@charleswilson.org

H. RICHARD WINN (Deborah).....1993
Annenberg Building 8-35
Mt. Sinai School of Medicine, Box 1136
One Gustave L. Levy Place
New York, NY 10029-6574
212-241-9128, fax 212-410-0603, richard.winn@mountsinai.org

FREMONT P. WIRTH (Lynn) 1993
4 Jackson Boulevard
Savannah, GA 31405-5895
912-355-1010, fax 912-629-9163, fpwirth@bellsouth.net

DAVID YASHON 1972
955 Eastwind Drive
Westerville, OH 43081
614-224-1720, fax 614-221-9805, dyashon@columbus.rr.com

A. BYRON YOUNG (Judith {Judy}) 1989
Division of Neurosurgery, Room MS101
University of Kentucky Medical Center
800 Rose Street
Lexington, KY 40536-0298
859-323-5864, fax 859-257-8011, afaul6@email.uky.edu

HAROLD YOUNG (M. Theresa) 1994
Department of Neurosurgery
Medical College of Virginia
Post Office Box 980631
Richmond, VA 23298-0631
804-828-9165, 804-828-0374, hfyoung@vcu.edu

NICHOLAS ZERVAS (Thalia) 1972
Department of Neurosurgery
Massachusetts General Hospital
55 Fruit Street
Boston, MA 02114
617-726-4141, fax 617-726-6789, nzervas@partners.org

ACTIVE MEMBERS Elected

EBEN ALEXANDER, III (Holley).....1999
Focused Ultrasound Surgery Foundation
213 Seventh Street NE
Charlottesville, VA 22902
434-220-4993 ext. 201, fax 434-220-4978, ealexander@fusfoundation.org

ISSAM AWAD (Catherine {Cathy}).....1996
Division of Neurosurgery, Burch 224
Northshore University Health Systems
2650 Ridge Avenue
Evanston, IL 60201
847-570-1440, fax 847-570-1442, iawad@northshore.org

JULIAN BAILES (Colleen).....2002
Department of Neurosurgery, Suite 4300
West Virginia University School of Medicine
One Medical Center Drive
Morgantown, WV 26506-9183
304-293-5041, fax 304-293-4819, jbailes@hsc.wvu.edu

NICHOLAS BARBARO (Sue Ellen).....2002
University of California San Francisco
Neurosurgery, Box 0112
San Francisco CA 94143-0112
415-353-3557, fax 415-353-3997, barbaron@neurosurg.ucsf.edu

GENE BARNETT (Cathy Ann Sila).....2000
Brain Tumor Institute, Neurosurgery/S80
Cleveland Clinic Foundation
9500 Euclid Avenue
Cleveland, OH 44195
216-445-1379, fax 216-444-9170, barnetg@ccf.org

DANIEL BARROW (Mollie).....1993
Section of Neurosurgery, Suite 6400
The Emory Clinic
1365 B Clifton Road NE
Atlanta, GA 30322
404-778-3895, fax 404-778-4472, daniel.barrow@emoryhealthcare.org

DAVID BASKIN (Julie)……...2006
Department of Neurosurgery, Suite #944
Methodist Neurological Institute
6560 Fannin Street
Houston, Texas 77030
713-441-3800, fax 713-793-1001, dbaskin@tmhs.org

H. HUNT BATJER (Janet) 1996
Department of Neurological Surgery, Suite 2210
Northwestern University Medical School
676 North St. Clair Street,
Chicago, IL 60611
312-695-6285, fax 312-695-0225, hbatjer@nmff.org

MITCHEL BERGER (Joan) 1997
UCSF Department of Neurosurgery
505 Parnassus Avenue, M-786
Box 0112
San Francisco, CA 94143-0112
415-353-3933, fax 415-353-3910, bergerm@neurosurg.ucsf.edu

KEITH BLACK (Carol Bennett) 1995
Cedars-Sinai Medical Center
Maxine Dunitz Neurosurgical Institute
8631 West Third Street, Suite 800 East
Los Angeles, CA 90048
310-423-1773, fax 310-423-1008, black@cshs.org

PETER BLACK (Katharine).....1988
Department of Neurosurgery
Brigham & Women's Hospital
75 Francis Street
Boston, MA 02115
617-525-7796, fax 617-734-8342, pblack@partners.org

LAWRENCE BORGES (Susan) 1993
Neurosurgery, White 1205
Massachusetts General Hospital
55 Fruit Street
Boston, MA 02114
617-726-6156, fax 617-724-7407

CHARLES BRANCH, JR. (Lesa).....1996
Department of Neurosurgery
Wake Forest University- Baptist Medical Center
Medical Center Boulevard
Winston-Salem, NC 27157-1029
336-716-4083, fax 336-716-3065, cbranch@wfubmc.edu

HENRY BREM (Rachel) 1996
Neurosurgery, Meyer 7-113
Johns Hopkins Hospital
600 N. Wolfe Street
Baltimore, MD 21287
410-955-2252, fax 410-955-8263, hbrem@jhmi.edu

JEFFREY BRUCE (Rebecca) 2002
Neurological Institute, Rm. 434
Columbia University Medical Center
710 W. 168th Street
New York, NY 10032
212-305-7346, fax 212-305-2026, jnb2@columbia.edu

KIM BURCHIEL (Debra) 1992
Dept of Neurosurgery
Oregon Health & Science University
3303 SW Bond Avenue
Portland, OR 97201
503-494-7978, fax 503-494-7161, burchiek@ohsu.edu

FADY CHARBEL (Alexandra) 2003
Department of Neurosurgery, (MC 799)
University of Illinois at Chicago
912 South Wood Street
Chicago, IL 60612
312-996-4842, fax 312-966-9018, fcharbel@uic.edu

E. ANTONIO CHIOCCA (Charlotte)……...2005
Ohio State University Medical Center
Department of Neurosurgery
N-1021 Doan Hall
410 W. 10th Avenue
Columbus, OH 43210
614-293-9312, fax 614-293-4024, ea.chiocca@osumc.edu

ALAN COHEN (Shenandoah Robinson**)**.....1999
Division of Neurosurgery
Rainbow Babies and Children's Hospital
Room B-501
11100 Euclid Avenue
Cleveland, OH 44106
216-844-5741, fax 216-844-5710, alan.cohen@uhhs.com

E. SANDER CONNOLLY, Jr (Christine) 2004
Department of Neurosurgery
Columbia University, Room 435
710 West 168th Street
New York City, NY 10032
212-305-0376, fax 212-305-2026, esc5@columbia.edu

GARTH REES COSGROVE (Karen).....1997
Lahey Clinic Medical Center
Department of Neurosurgery
41 Mall Road
Burlington, MA 01805

781-744-1990, fax 781-744-1147, g.rees.cosgrove@lahey.org

WILLIAM COULDWELL (Marie Simard) 1999
Department of Neurosurgery
University of Utah
175 North Medical Drive East
Salt Lake City, UT 84132
801-581-6908, fax 801-581-4385, william.couldwell@hsc.utah.edu

RALPH DACEY, JR. (Corinne).....1990
Department of Neurological Surgery, Campus Box 8057
Washington University School of Medicine
660 South Euclid
St. Louis, MO 63110
314-362-5039, fax 314-362-2107, daceyr@wustl.edu

ARTHUR DAY (Dana) 1990
Department of Neurosurgery, Room PB301
Brigham & Women's Hospital
75 Francis Street
Boston, MA 02115
617-732-6600, fax 617-264-5114, aday1@partners.org

JOHNNY DELASHAW (Fran) 2004
Department of Neurological Surgery, CH8N
Oregon Health Sciences University
3303 SW Bond Avenue
Portland, OR 97239
503-494-4314, fax 503-494-0870, Delashaw@ohsu.edu

ROBERT DEMPSEY (Diane).....1996
Department of Neurological Surgery, Room K4/822
University of Wisconsin
600 Highland Avenue
Madison, WI 53792
608-263-9585, fax 608-263-1728, dempsey@neurosurg.wisc.edu

JAMES DRAKE (Elizabeth Jane)……2005
Division of Neurosurgery
Roy C. Hill Wing, Suite 1504
The Hospital for Sick Children
555 University Avenue
Toronto, Ontario, M5G 1X8
Canada
416-813-6125, fax 416-813-4975, james.drake@sickkids.ca

MICHAEL FEHLINGS (Darcy).....2004
Neurosurgery, Suite 4W-449
Toronto Western Hospital
339 Bathurst Street

Toronto, Ontario M5T 2S8
Canada
416-603-5627, fax 416-603-5298, mchael.fehlings@uhn.on.ca

RICHARD FESSLER (Carol).....2004
Department of Neurosurgery, Suite 2210
Northwestern University
676 North St. Clair,
Chicago, IL 60611
312-695-6200, fax 312-695-0225, rfessler@nmff.org

KEVIN FOLEY (Lynn).....1999
Image-Guided Surgery Research Center
Semmes-Murphey Clinic, Suite 200
1211 Union Avenue
Memphis, TN 38104
901-259-5340, fax 901-259-2058, kfoley@usit.net

ROBERT FRIEDLANDER (Eugenia)……..2006
Department of Neurosurgery
Brigham & Women's Hospital
75 Francis Street
Boston, MA 02115
617-525-7775, fax 617-734-8342, rfriedlander@rics.bwh.harvard.edu

ALLAN FRIEDMAN (Elizabeth Bullitt).....1994
Division of Neurological Surgery
Duke University Medical Center
Box 3807
Durham, NC 27710
919-684-3271, fax 919-681-7973, fried010@mc.duke.edu

WILLIAM FRIEDMAN (Ransom).....1995
Department of Neurosurgery
University of Florida Health Sciences Center
P.O. Box 100265, MBI
Gainesville, FL 32610-0265
352-273-9000, fax 352-392-8413, friedman@neurosurgery.ufl.edu

DANIEL FULTS, III (Carol).....1997
Clinical Neurosciences Center, Room 5229
University of Utah
175 North Medical Drive East
Salt Lake City, UT 84132-2303
801-581-6908, fax 801-581-4385, daniel.fults@hsc.utah.edu

STEVEN GIANNOTTA (Sharon).....1992
Department of Neurosurgery, Suite 5046
University of Southern California
1200 North State Street

Los Angeles, CA 90033-4525
323-226-7421, fax 323-226-7833, giannott@usc.edu

M. SEAN GRADY (Debra).....2003
Department of Neurosurgery
University of Pennsylvania
Silverstein Pavilion, 3rd Floor
3400 Spruce Street
Philadelphia, PA 19104
215-349-8325, fax 215-349-5108, gradys@uphs.upenn.edu

MARK HADLEY (Lori).....2001
Division of Neurosurgery
University of Alabama
1030 Faculty Office Tower
510 20th Street South
Birmingham AL 35294
205-934-1439, fax 205-975-6081, mhadley@uabmc.edu

STEPHEN HAINES (Jennifer Plombon).....1994
Department of Neurosurgery
University of Minnesota Medical School
D429 Mayo Memorial Building, MMC 96
420 Delaware Street, SE
Minneapolis MN 55455
612-626-5767, fax 612-624-0644, shaines@umn.edu

ROBERT HARBAUGH (Kimberly)… 2001
Department of Neurosurgery
Penn State University–Milton S. Hershey Medical Center
30 Hope Drive
Hershey PA 17033-0850
717-531-4383, fax 717-531-3858, rharbaugh@psu.edu

HAYNES LOUIS HARKEY, III (Alison).....2002
Department of Neurosurgery
University of Mississippi Medical Center
2500 North State Street
Jackson, MS 39216-4505
601-984-5714, fax 601-815-9658, lharkey@neurosurgery.umsmed.edu

GRIFFITH HARSH, IV (Meg Whitman) 2001
Department of Neurosurgery, CC2222
Stanford University Medical Center
875 Blake Wilbur Drive
Stanford, CA 94305-5826
650-725-0701, fax 650-498-4686, gharsh@stanford.edu

CARL HEILMAN (Carolyn).....2002

Department of Neurosurgery, # 178
Tufts Medical Center
800 Washington Street
Boston, MA 02111
617-636-5860, fax 617-636-7587, cheilman@tuftsmedicalcenter.org

MATTHEW HOWARD, III (Delia).....2004
Department of Neurosurgery, 1840 JPP
University of Iowa Hospitals & Clinics
200 Hawkins Drive
Iowa City, IA 52242
319-356-8468, fax 319-353-6605, matthew-howard@uiowa.edu

BERMANS J. ISKANDAR (Jenny)…… 2007
Department of Neurological Surgery, K4/832
University of Wisconsin Hospitals & Clinics,
600 Highland Avenue
Madison, WI 53792
608-263-9651, fax 608-263-1728, iskandar@neurosurg.wisc.edu

IAIN KALFAS (Holly) ……..2003
Department of Neurosurgery (S-80)
Cleveland Clinic Foundation
9500 Euclid Avenue
Cleveland, OH 44195
216-444-9064, fax 216-636-3174, kalfasi@ccf.org

DOUGLAS KONDZIOLKA (Susan).....1998
Department of Neurological Surgery
University of Pittsburgh Medical Center, Suite B-400
200 Lothrop Street
Pittsburgh, PA 15213
412-647-6782, fax 412-647-0989, kondziolkads@upmc.edu

WILLIAM E. KRAUSS (Joan) 2007
Department of Neurologic Surgery,
Mayo Clinic, Gonda 8-209
200 1st Street SW
Rochester, MN 55905
507-284-3331, fax 507-284-5206, krauss.william@mayo.edu
MICHAEL LAWTON (Suzanne) 2003
Department of Neurosurgery
UCSF, M-780C
505 Parnassus Avenue
San Francisco, CA 94143-0112
415-353-3998, fax 415-353-3907, lawtonm@neurosurg.ucsf.edu

ELAD I. LEVY (Cynthia {Cindy}) ……2008
Department of Neurosurgery
State University of New York at Buffalo

3 Gates Circle
Buffalo, NY 14209
716-887-5200, fax 716-887-4672, eoconnor@ubns.com

MICHAEL LEVY (Karen) 2003
Department of Neurosurgery, Suite 502
University Childrens Medical group
8010 Frost Street
San Diego, CA 92123
858-966-8574, fax 858-966-7930, mlevy@chsd.org

CHRISTOPHER LOFTUS (Sara Sirna).....1992
Department of Neurosurgery
Temple University
3401 North Broad Street
Philadelphia PA 19140
215-707-2620, fax 215-707-3831, cloftus@temple.edu

ANDRES LOZANO (Marie Slegr).....2004
Neurosurgery, Rm 4-447 West Wing
Toronto Western Hospital
399 Bathurst Street
Toronto, Ontario Canada M5T 2S8
416-603-6200, fax 416-603-5298, lozano@uhnres.utoronto.ca

R. LOUGHLIN MACDONALD (Sheilah).....2000
Division of Neurosurgery
St. Michael's Hospital
30 Bond Street
Toronto, ON M5B 1W8
416-864-5452, fax 416-864-5634, macdonaldlo@smh.toronto.on.ca

ROBERT MACIUNAS (Ann Failinger).....1999
Department of Neurological Surgery, HH5
University Hospitals-Case Medical Center
11100 Euclid Avenue
Cleveland, OH 44106
216-844-5743, fax 216-844-3014, robert.maciunas@uhhospitals.org

JOSEPH MADSEN (Ilonna Rimm).....2003
Department of Neurosurgery
Children's Hospital of Boston
300 Longwood Avenue
Boston, MA 02115
617-355-6005, fax 617-734-2628, joseph.madsen@tch.harvard.edu

TIMOTHY MAPSTONE (Barbara).....2004
Department of Neurosurgery

University of Oregon Health Science Center
Suite 400
1000 North Lincoln Blvd.
Oklahoma City, OK 73104
405-271-4912, fax 405-271-3091, timothy-mapstone@ouhsc.edu

JAMES MARKERT (Laili).....2002
Neurosurgery, University of Alabama at Birmingham
1050 Faculty Office Towers
510 20th Street South
Birmingham, AL 35294-3410
205-934-2918, fax 205-996-4674, jmarkert@uabmc.edu

MARC MAYBERG (Teresa {Terry}) 1995
Swedish Neuroscience Institute, Suite 500
550 17th Avenue
Seattle, WA 98122
206-320-2805, fax 206-320-2827, marc.mayberg@swedish.org

PAUL MCCORMICK (Doris) 1998
Department of Neurosurgery
Neurological Institute
710 West 168th Street
New York, NY 10032
212-305-7976, fax 212-342-6850, pcm6@columbia.edu

CAMERON G. MCDOUGALL (Inga Wiens)…… 2007
Barrow Neurologic Institute
2910 N. 3rd Avenue
Phoenix, AZ 85013
602-406-3964, fax 602-406-7137, cgm@bnaneuro.net

GUY McKHANN (Lianne de Serres McKhann)…… .2006
Neurological Institute, NI-42
Columbia University Medical Center
710 West 168th Street
New York, NY 10032
212-305-0052, fax 212-305-3629, gm317@columbia.edu

FREDRIC MEYER (Irene)..... 1995
Department of Neurologic Surgery
Mayo Clinic, Gonda 8-209
200 First Street SW
Rochester, MN 55905
507-284-5317, fax 507-284-5206, meyer.fredric@mayo.edu

RAJIV MIDHA (Vandy) ……2007
Clinical Neurosciences
Foothills Medical Centre, Room 1195

1403 29th Street N.W.
Calgary, Alberta T2N 2T9
403-944-1259, fax 403-270-7878, rajmidha@ucalgary.ca

JACQUES MORCOS (Fiona).....2003
Department of Neurological Surgery (D4-6)
Lois Pope Life Center
1095 NW 14th Terrace
Miami, FL 33136
305-243-4675, fax 305-243-3337, jmorcos@med.miami.edu

KARIN M. MURASZKO (Scott Van Sweringen)……2007
Department of Neurosurgery
University of Michigan, 3470 Taubman Center
1500 E. Medical Center Drive
Ann Arbor, MI 48109-5338
734-936-5015, fax 734-647-0964, karinm@umich.edu

ANIL NANDA (Laura) …….2008
Department of Neurological Surgery
Louisiana State University HSC-Shreveport
1501 Kings Highway
Shreveport, LA 71130
318-675-6404, fax 318-675-6867, ananda@lsuhsc.edu

RAJ NARAYAN (Tina)……..2005
University of Cincinnati, College of Medicine
Department of Neurosurgery, Room 4314
P.O. Box 670515
231 Albert Sabin Way
Cincinnati, OH 45267-0515
513-558-3556, fax 513-558-7702, raj.narayan@uc.edu

DAVID NEWELL (Shirley).....2002
Swedish Neuroscience Institute, Suite 500
550 17th Avenue
Seattle, WA 98122
206-320-2800, fax 206-320-2827, david.newell@swedish.org

W. JERRY OAKES (Jean) 1999
Pediatric Neurosurgery, ACC 400
The Children's Hospital of Alabama
1600 7th Avenue South
Birmingham, AL 35233 - 1711
205-939-6914, fax 205-939-9972, wjomd@uab.edu

CHRISTOPHER OGILVY.....2000
Neurosurgery, Wang 745
Massachusetts General Hospital
55 Fruit Street
Boston, MA 02114
617-726-3303, fax 617-726-7501, cogilvy@partners.org

ALESSANDRO OLIVI (Luisa) ……. 2007
Department of Neurosurgery, Phipps 1-100
The Johns Hopkins Hospital
600 N. Wolfe Street
Baltimore, MD 21287
410-955-0703, fax 410-614-9877, aolivi@jhmi.edu

NELSON OYESIKU (Lola)……..…2005
Department of Neurosurgery, Suite #6200
Emory University School of Medicine
1365-B Clifton Road, N.E.
Atlanta, GA 30322
404-778-4737, fax 404-778-4472, noyesik@emory.edu

STEPHEN PAPADOPOULOS (Penny).....2000
Barrow Neurological Institute
2910 N. Third Avenue
Phoenix, AZ 85013
602-406-3159, fax 602-406-3167, stvpapa@bnaneuro.net

BRUCE POLLOCK (Kristen).....2004
Department of Neurologic Surgery
Mayo Clinic, Gonda 8-209
200 First Street SW
Rochester, MN 55905
507-284-5317, fax 507-284-5206, pollock.bruce@mayo.edu

A. JOHN POPP (Margaret Vosburgh).....2001
Department of Neurosurgery, PBB3
Brigham & Women's Hospital
15 Francis Street
Boston, MA 02115
617-525-9419, fax 617-734-8342, jpoppl@partners.org

COREY RAFFEL (Kathy) 1998
Division of Pediatric Neurosurgery
Nationwide Children's Hospital
The Ohio State University
700 Children's Drive
Columbus, OH 43205
614-722-2014, fax 614-722-2041, corey.raffel@nationwidechildrens.org

HOWARD A. RIINA (Anne) ……2008
Department of Neurological Surgery
Weill Medical College of Cornell Universtiy,
New York Presbyterian Hospital
525 East 68th Street
New York, NY 10021
212-746-5953, fax 212-746-8416, har9005@med.cornell.edu

DAVID ROBERTS (Kathryn).....1996
Section of Neurosurgery
Dartmouth-Hitchcock Medical Center
One Medical Center Drive
Lebanon, NH 03756
603-650-8734, fax 603-650-7911, david.w.roberts@dartmouth.edu

GERALD (Rusty) RODTS (Kelly) 2003
Neurosurgery, Suite 3000
Emory Spine Center
59 Executive Park South
Atlanta, GA 30329
404-778-6227, fax 404-778-6310, grodts@emory.edu

ROBERT ROSENWASSER (Deborah August).....1996
Neurosurgery, 3rd Floor
Thomas Jefferson University Hospital
909 Walnut Street
Philadelphia, PA 19107
215-503-7022, fax 215-503-2452, robert.rosenwasser@jefferson.edu

JAMES RUTKA (Mari)……... 1996
Division of Neurosurgery, Suite 1503
The Hospital for Sick Children
555 University Avenue
Toronto, Ontario M5G 1X8
Canada
416-813-6425, fax 416-813-4975, james.rutka@sickkids.ca

RAYMOND SAWAYA 2003
Department of Neurosurgery, Unit 442
The University of Texas M.D.
Anderson Cancer Center
1515 Holcombe Boulevard
Houston, TX 77030
713-563-8749, fax 713-563-1804, rsawaya@mdanderson.org

MICHAEL SCHULDER (Lu Steinberg)…….2005
Department of Neurosurgery, 9 Tower
North Shore University Hospital

300 Community Drive
Manhasset, NY 11030
516-562-3065, fax 516-562-3631, schulder@nshs.edu

WARREN SELMAN (Diana).....1995
Department of Neurosurgery, HAN 5042
University Hospitals Case Medical Center
11100 Euclid Avenue
Cleveland, OH 44106
216-844-7600, fax 216-844-3014, warren.selman@uhhospitals.org

CHRISTOPHER SHAFFREY (Catherine)……..2006
Department of Neurological Surgery
University of Virginia Health System
P.O. Box 800212
Charlottesville, VA 22908-0212
434-243-9714, fax 434-243-9248, cis8Z@virginia.edu

MARK E. SHAFFREY (Caroline Smith Shaffrey) ……..2008
Department of Neurological Surgery
University of Virginia Health System
P.O. Box 800212
Charlottesville, VA 22908- 0212
434-924-1843, fax 434-982-0264, mes8c@virginia.edu

J. MARC SIMARD (Monique Bellefleur).....1999
Neurosurgery, Suite S12D04B
University of Maryland
22 South Greene Street
Baltimore, MD 21201
410-328-0850, fax 410-328-0756, msimard@smail.umaryland.edu

ROBERT SOLOMON (Barbara) 1996
The Neurological Institute of New York
710 West 168th Street
New York, NY 10032
212-305-4118, fax 212-305-2026, ras5@columbia.edu

ROBERT SPETZLER (Nancy).....1997
Barrow Neurological Institute
350 West Thomas Road
Phoenix, AZ 85013
602-406-3489, fax 602-406-4402, rspetzler@thebni.com

PHILIP STARR (Chantal**)** 2004
Department of Neurosurgery, Box 0445
University of California, San Francisco
533 Parnassus Avenue
San Francisco, CA 94143
415-353-7500, 415-353-2889, starrp@neurosurg.ucsf.edu

GARY STEINBERG (Sandra Garritano)…… ……....2006
Department of Neurosurgery, Room R281
Stanford University Medical Center
300 Pasteur Drive
Stanford, CA 94305
650-723-5575, fax 650-723-2815, gsteinberg@stanford.edu

PHILIP STIEG (Janet**)**.....2001
Neurological Surgery, Box 99
Weill Medical College – Cornell University
525 E. 68th Street
New York, NY 10065
212-746-4684, fax 212-746-6607, pes2008@med.cornell.edu

B. GREGORY THOMPSON (Ramona).....2004
Department of Neurosurgery, 3470 TC 3552
University of Michigan Medical Center
1500 East Medical Center Drive
Ann Arbor, MI 48109-5338
734-936-7493, fax, 734-936-9294, gregthom@med.umich.edu

VINCENT TRAYNELIS (Joan).....2001
Department of Neurosurgery, Suite 1115
Rush University Medical Center
1725 West Harrison
Chicago, IL 60612
312-942-6628, fax 312-563-3358 vincent_traynelis@rush.edu

ALEX B. VALADKA (Patty)……2007
Neurosurgery, Suite 1020
University of Texas-Houston
6410 Fannin Street
Houston, TX 77030
832-325-7090, fax 713-512-2238, alex.valadka@uth..tmc.edu

HARRY VAN LOVEREN (Jeffrie Hood).....1995
Department of Neurosurgery
South Tampa Center, 7th floor
University of South Florida
2 Tampa General Circle
Tampa, FL 33606
813-259-0965, fax 813-259-0858, hvanlove@health.usf.edu

DENNIS VOLLMER (Dorothy).....2001
Colorado Brain & Spine Institute, Suite #220

499 E. Hampden Ave.,
Englewood, CO 80113
303-783-8844, fax 303-783-2002, dvollmer@coloradoneurosurgery.com

RAND VOORHIES (Terry).....1996
Neurosurgery, Suite 510
Southern Brain and Spine
4228 Houma Blvd
Metairie, LA 70006
504-454-0141, fax 504-889-7205, voorhies@sbsdocs.net

M. CHRISTOPHER WALLACE (Katie) 2003
Division of Neurosurgery WW 4-450
The Toronto Western Hospital
399 Bathurst Street
Toronto, Ontario, Canada M5T 2S8
416-603-5428, fax 416-603-5298, chris.wallace@uhn.on.ca

RONALD WARNICK (Ana).....2000
Neurosurgery, # 3100
222 Piedmont Avenue
Cincinnati, OH 45219
513-475-8629, fax 513-475-8664, nsgymd@mac.com

ERIC ZAGER (Marirosa Colon) …… 2006
Department of Neurosurgery
Silverstein Building, 3rd Floor
University of Pennsylvania Hospital
3400 Spruce Street
Philadelphia, Pennsylvania 19104
215-662-3497, fax 215-349-5534, zagere@uphs.upenn.edu

SENIOR CORRESPONDING MEMBERS Elected

HIROSHI ABE (Yoko) 1999
Medical Scanning Sapporo Clinic
N-4, W-5, Chuoku
Sapporo, Hokkaido 060-0004
JAPAN
81-11- 208-3501, fax 81- 11-208-3502, hiroshiABE@aol.com

JOAO (JOHN) ANTUNES (Maria do Ceu Machado)...2001
Hospital de Santa Maria
Servico de Neurocirurgia
Av. Prof Egas Moniz
1649-035, Lisbon
PORTUGAL
351-21-797-2855, fax (same #), jlobo.antunes@mail.telepac.pt

R. LEIGH ATKINSON (Noela).....1989
201 Wickham Terrace
Brisbane, Queensland 4000
AUSTRALIA
61-7- 3839-3393, fax 61- 7-3832- 2005, leighatkinson@optusnet.com.au

ARMANDO BASSO (Milva).....1996
Ayacucho 1342
Buenos Aires, 1111
ARGENTINA
54-11- 4806-3635, fax 54-11-4806-6531, armandojbasso@aol.com

ALBINO BRICOLO (Annapaola Zandomeneghi).....2002
Department of Neurosurgery
University Hospital of Verona
Piazzale Stefani 1
Verona 37126 ITALY
39-045-8122007, fax 39- 045- 916790, albino.bricolo@univr.it

MARIO BROCK (Christina) 2001
Pueckler Strasse 10
D-14195
Berlin, GERMANY
49-177-825-2571, fax 49-89-727-324, prof.m@riobrock.de

JACQUES BROTCHI (Rachel) 2003
Department of Neurosurgery
Erasme Hospital, Universite Libre de Bruxelles
808, Route de Lennik
B-1070 Brussels
BELGIUM

32-2-555- 3694, fax 32-2-555- 3755, jbrotchi@skynet.be

LUC CALLIAUW (Dora**)**……..1988
Sint-Annarei 19
B-8000, Brugge
BELGIUM
32-50-344-377, fax 32-50-344-377, luccalliauw@hotmail.com

H. ALAN CROCKARD (Caroline)……1992
Department of Surgical Neurology
The National Hospital for Neurology and Neurosurgery
Queen Square
London, England WC1N 3BG
UNITED KINGDOM
44-20-7 829- 8714, fax 44-20-7676- 2044, alan.crockard@tiscali.co.uk

GIUSEPPE DALLE ORE (Guisi Scimone).....1970
Casa di Cura Villa Lieta
Via Anzani n. 12
Verona 37126
ITALY
39-045-914430, fax 39-045-914430, dalleore@libero.it

NOEL G. DAN (Adrienne).....1989
Specialist Medical Centre
235 New South Head Road
Edgecliff, N.S.W. 2029
AUSTRALIA
61-2-9327-8133, fax 61-2- 9327-5807, noeld@med.usyd.edu.au

EVANDRO DE OLIVEIRA (Marina) 2002
Praca Amadeu
Amaral 27 Andar 5
01327-010 Sao Paulo, SP
BRAZIL
55-11-288-8635, fax 55-11-251-1766, icne@uol.com.br

NICOLAS DE TRIBOLET (Veronique) 1995
Cour St. Pierre 7
CH-1204 Geneva
SWITZERLAND
41-795400844, nicolas.detribolet@unige.ch

JACQUES DE VILLIERS (Jeanne Marie Erica).....1986
7 Finsbury Avenue
Newlands
Cape Town, 7700
REPUBLIC OF SOUTH AFRICA
27-21-674- 3828, fax (same #), jcdevill@iafrica.com

HANS ERICH DIEMATH (Karoline**)** 1970
Maxglaner Hauptstrasse 6
A-5020, Salzburg
AUSTRIA
43-662-62-28-50, fax 43-662-62- 28-501, diemath@inode.at

HERMANN DIETZ (Elfrun) 1980
An Der Trift 10/B
D-30559, Hannover
GERMANY
49-511-525-686, fax (same #)

VINKO DOLENC(Anabel**)**.....1988
Neurosurgical Department
University Hospital Center - Ljubljana
Zaloska cesta 7
Ljubljana, SI-1525
SLOVENIA
38 6-1-522- 2218, fax (same #), vinko.dolenc@kclj.sl; janja.boh@kclj.si

RUDOLF FAHLBUSCH 1991
International Neuroscience Institute
Rudolf-Pichlmayr-Str. 4
D-30625 Hannover
GERMANY
49-511-27092-828, fax 49-511-27092-987, fahlbusch@ini-hannover.de

F. JOHN GILLINGHAM (Judy) 1962
Unable to locate contact information

HECTOR GIOCOLI (Maria Cristina Garcia).....2000
Address unknown

JAIME GOMEZ (Lucy) 1975
148 Newcastle Drive
Jupiter, FL 33458-3021
561-694-2853, drgomezmd@gmail.com

SALVADOR GONZALEZ-CORNEJO (Rosa).....1982
Address unknown

ERNST H. GROTE (Julianna).....1984
Ob der Grafenhalde 7
D-72076 Tuebingen
GERMANY
49-7071-408993, fax 49-7071-408994, je.grote@web.de

DAE HEE HAN (Sung Soon Cho).....1991
#39 Boramae-Gil
Dongjak-Gu
Seoul, 156-707
SOUTH KOREA
82-2-870-2305, fax 82-2-766-3322, daehan@snu..ac.kr

HAJIME HANDA (Hiroko).....1985
228-136 Naka-machi
Iwakura Sakyo-ku
Kyoto, 606-0025
JAPAN
81-75-701-8470

NOBUO HASHIMOTO (Etsuko**)** 2003
5-7-1 Fujishiro-dai
Suita, Osaka 565-8565
JAPAN
81-6-6833-5012, fax 81-6-6833-9865, hashimot@hsp.ncvc.go.jp

FABIAN ISAMAT (Maria Victoria {Marivi}).....1989
Neurogroup
Clinica Sagrade Familia
Ronda eneral Mitre 95
08022 Barcelona
SPAIN
34-932118991, fax 34- 932531879, 3345 fir@comb.cat

SHOZO ISHII (Akiko).....1975
5-24-16, Nakamachi
Setagaya-ku
Tokyo, 158-0091
JAPAN
81-3- 3703-7928, fax 81- 3-3703-7928

TAKESHI KAWASE (Mieko) 1997
Department of Neurosurgery
Keio University, School of Medicine
35 Shinanomachi, Shinjuku-ku
Tokyo 160-8582
JAPAN
81- 3-5363-3807, fax 81- 3-3358- 0479, kawase@sc.itc.keio.ac.jp

HARUHIKO KIKUCHI (Yuriko**)……**1993
Kobe City Medical Center
4-6 Minatojima-Nakamachi, Chuo-ku
Kobe 650-0046
JAPAN

81-78-302-4321, fax 81-78-302-8123

SHIGEAKI KOBAYASHI (Hideko) 1998
Medical Education and Research Center
Aizawa Hospital
Honjo 2-5-1
Matsumoto 390-8510
JAPAN
81-163-33-8600, fax 81- 263- 33-8716, skb0305@wa2.so-net.ne.jp

RAUL MARINO, JR (Angela).....1977
Instituto Neurologico De Sao Paulo
Rua Maestro Cardim, 808
Sao Paulo, SP 01323001
BRAZIL
55-11-3287-1130, fax 55-11-3141-9556, raulmarino@uol.com.br

A. DAVID MENDELOW (Michelle Davis)……..2005
Department of Neurosciences, Ward 31
Newcastle General Hospital
Westgate Road
Newcastle Upon Tyne NE4 6BE
UNITED KINGDOM
0191-256-3151, fax 0191-256-3262, a.d.mendelow@ncl.ac.uk

JORGE S. MENDEZ (Soledad) 1997
Marcoleta 367
Santiago
CHILE
562-770-950, fax 562- 639-5534,jorgemendez@manquehue.net

JOHN DOUGLAS PICKARD [Charlotte (Mary)].....2001
Academic Neurosurgery Unit
Box 167, Level A4, Addenbrookes Hospital
Cambridge, England CB2 2QQ
UNITED KINGDOM
44-1223- 336-946, fax 44-1223- 216-926, prof.jdp@medschl.cam.ac.uk

HANS-JUERGEN REULEN (Ute).....1998
Kastellstr. 5
81247 Munich
GERMANY
49-89-864-2524, hjreulen@gmx.de

MADJID SAMII (Mahsdrid).....1996
International Neuroscience Institute - Hannover
Rudolf-Pichlmayr-Str.4
30625, Hannover
GERMANY

49-511-270-92-700, fax 49-511-270- 92-706, samii@ini-hannover.de

JOHANNES SCHRAMM (Dorothea).....2002
Neurochirurgische
Universitats.-Klinik
Sigmund-Freud Str. 25
D-53127 Bonn
GERMANY
49-228-287- 6500, fax 49- 228-287- 6573, Johannes.Schramm@ukb.uni-bonn.de

CHARAS SUWANWELA (Nitaya) 1972
Chulalongkorn University Council
Chulalongkorn University
Phyathai Road
Bangkok, 10330
THAILAND
66-2-218-3305, fax 66-2 -218-3309, charas.s@chula.ac.th

LINDSAY SYMON (Pauline).....1982
Maple Lodge
Rivar Road
Shalbourne, Marlborough
Wiltshire, England SN8 3QE
UNITED KINGDOM
44-1672-870- 501, lindsaysymon@tixali.co.uk

KINTOMO TAKAKURA (Tsuneko).....1988
Institute of Advanced Biomedical Sciences
Tokyo Women's Medical University
8-1, Kawadacho, Shinjukuku
Tokyo 162-8666
JAPAN
81-3-5367-9945ext. 6302, fax 81- 3- 5361-7796, ktakakura@abmes.twmu.ac.jp

GRAHAM TEASDALE (Evelyn) 2004
NHS Quality Improvement Scotland
Delta House
50 West Nile Street
Glasgow, Scotland G12NP
United Kingdom
011-44-141-225-5566, graham.teasdale@nhs.net

DAVID THOMAS (Hazel).....1995
The National Hospital for Neurology & Neurosurgery
Private Consulting Rooms – Box 147
Queen Square
London, England WC1N 3BG
UNITED KINGDOM

44-207-391-8993, fax 44-207-391-8816, marcel.yazbeck@uclh.nhs.uk; roseann.mccrea@uclh.nhs.uk

E. SYDNEY WATKINS (Susan).....1975
Belmont House
Coldstream
Berwickshire, England TD 12 4ET
UNITED KINGDOM

M. GAZI YASARGIL (Dianne) 1975
Neurosurgery, #507
University of Arkansas for Medical Sciences
4301 West Markham
Little Rock, AR 72205-7199
501-686-6979, fax 526-5205, stellkathrynj@uams.edu

CORRESPONDING MEMBERS Elected

HELMUT BERTALANFFY (Atsuko)……2008
Department of Neurosurgery
University Hospital Zurich
Frauenklinikstr.10
CH-8091,Zurich
SWITZERLAND
41-44-255-2660, fax 41-44-255-4505, helmut.bertalanffy@usz.ch

A. GRAHAM FIEGGEN.(Karen) ……2008
Division of Neurosurgery
H53 Old Main Building
Groote Schuur Hospital
Observatory 7925
Cape Town
SOUTH AFRICA
27-21-406-6213, fax 27-21-406-6555, graham.fieggen@uct.ac.za

KIYOHIRO HOUKIN (Hiromi) ……..2006
Department of Neurosurgery
South-1, West-16
Sapporo Medical University
Sapporo 060-8543
JAPAN
81-11- 611- 2111, fax 81-11- 614-1662, houkin@sapmed.ac.jp

HEE-WON JUNG (Kyung Hee Park) ……..2006
Department of Neurosurgery
Seoul National University Hospital
28, Yongon-dong, Jongno-gu
Seoul 110-744
SOUTH KOREA
82-11-391-2355, fax 82- 2- 831-0721, hwnjung@snu.ac.kr

IMAD N. KANAAN (Huda)…….....2008
Department of Neurosciences, MBC-76
King Faisal Specialist Hospital & Research Centre
P.O. Box 3354
Riyadh 11211
KINGDOM OF SAUDI ARABIA
966-1- 464-7272 Ext 32770, fax 966-1- 442- 4763, dr.imad.kanaan@gmail.com

ANDREW KAYE (Judith) 1996
Department of Neurosurgery, Room 426, 4 East
The Royal Melbourne Hospital
Grattan Street
Parkville, Victoria 3050

AUSTRALIA
61- 3- 9342- 8218, fax 61- 3- 9342-7273, andrew.kaye@mh.org.au

BYUNG DUK KWUN (Eun Joo Lee)……2005
Department of Neurological Surgery
ASAN Medical Center
86 Asanbyeongwon-gil, Songpa-gu
Seoul 138-736
KOREA
82-2-3010-3552, fax 82-2-476-6738, bdkwun@amc.seoul.kr

EDWARD MEE (Jane Elliott)……..…2005
Department of Neurosurgery
Auckland City Hospital
Private Bag
Auckland
NEW ZEALAND
649-520-9672, fax 649-520-9673, edward.mee@xtra.co.nz

BASANT MISRA (Sasmita) …….2008
P.D. Hinduja National Hospital & MRC
V.S. Marg
Mahim, Mumbai 400 016
INDIA
91-22-24447204 or 24447214, fax 91-22-24447220 or 24440425, basantkmisra@gmail.com

MICHAEL MORGAN (Elizabeth).....1999
Australian School of Advanced Medicine
Level 1 Dow Corning Building
3 Innovation Road
Macquarie University, N.S.W. 2109
AUSTRALIA
61- 2- 9850- 4012, fax 61-2- 9850-4010, michael.morgan@mq.edu.au

M.NECMETTIN PAMIR (Feriha)……2006
Department of Neurosurgery
Inonu Cad. Okur Sok. No. 20
34742, Kozyatagi/Kadikoy
Istanbul
TURKEY
90-216-571-4483, fax 90-216-658-8456, pamirmn@yahoo.com

WAI SANG POON (Gillian Kew) **…….**2008
Division of Neurosurgery
Prince of Wales Hospital
Shatin, New territories
HONG KONG
852-2632-2624, fax 852-2637-7974, wpoon@surgery.cuhk.edu.hk

GABRIELE SCHACKERT (Hans) 2003
Klinik und Poliklinik fur Neurochirurgie
Fetscherstrasse 74
D-01307 Dresden
GERMANY
49- 351-458-2883, fax 49-351-458- 4304,Gabriele.Schackert@uniklinikum-dresden.de

YONG-KWANG TU (Charlotte) 2007
Department of Neurosurgery
National Taiwan University Hospital
7 Chung-Shan South Road
Taipei 100
TAIWAN
886-2-2312-3456 EXT. 65078, 886-2- 2341-7454, yktu@ntu.edu.tw

DECEASED MEMBERS

	Elected	Deceased
EBEN ALEXANDER, JR.	1950	2004
Winston-Salem, North Carolina		
(Senior)		
JAMES R. ATKINSON	1970	1978
Phoenix, Arizona		
(Active)		
PERCIVAL BAILEY	1960	1973
Evanston, Illinois		
(Honorary)		
GEORGE BAKER	1940	1993
Litchfield Park, Arizona		
(Senior)		
H. THOMAS BALLANTINE, JR.	1951	1996
Boston, Massachusetts		
(Senior)		
WILLIAM F. BESWICK	1959	1971
Buffalo, New York		
(Active)		
EDWIN B. BOLDREY	1941	1988
San Francisco, California		
(Senior)		
E. HARRY BOTTERELL	1938	1997
Kingston, Ontario, CANADA		
(Senior)		
ROBERT BOURKE	1983	1996
Rockville, Maryland		
(Senior)		
SPENCER BRADEN	Founder	1969
Cleveland, Ohio		
(Active)		
F. KEITH BRADFORD	1938	1971
Houston, Texas		
(Active)		
JEAN BRIHAYE	1975	1999
Bruxelles, BELGIUM		

(Senior Corresponding)

KARL-AUGUST BUSHE 1972 1999
Wurzburg, GERMANY
(Senior Corresponding)

HOWARD BROWN 1939 1990
San Francisco, California
(Senior)

FERNANDO CABIESES……1966……..2009
Lima, PERU
(Senior Corresponding)

JUAN CARDENAS..... 1966 1996
Mexico City, MEXICO
(Senior Corresponding)

HARVEY CHENAULT……...1949……..2006
Lexington, Kentucky
(Senior)

SHELLEY CHOU..... 1974 2001
Rio Verde, Arizona
(Senior)

JUAN CARLOS CHRISTENSEN1970 2003
Buenos Aires, ARGENTINA
(Senior Corresponding)

GALE CLARK..... 1970 1996
Oakland, California
(Senior)

W. KEMP CLARK……1970……2007
Dallas, TX 75205-3103
(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)

RICHARD DESAUSSURE, JR……..1962……..2008
Memphis, Tennessee
(Senior)

PEARDON DONAGHY.....1970.....1991
Burlington, Vermont
(Senior)

CHARLES DRAKE.....1958.....1998
London, Ontario, CANADA
(Senior)

FRANCIS ECHLIN.....1944.....1988
New Paltz, New York
(Senior)

DEAN ECHOLS.....Founder.....1991
New Orleans, Louisiana
(Senior)

GEORGE EHNI.....1964.....1986
Houston, Texas
(Senior)

ARTHUR ELVIDGE.....1939.....1985
Montreal, Quebec, CANADA
(Senior)

THEODORE ERICKSON....1940.....1986
Madison, Wisconsin
(Senior)

JOSEPH EVANS .....Founder.....1985
Kensington, Maryland
(Senior)

ROBERT FISHER ……..……..1955……..2003
Granada Hills, CA
(Senior)

JOHN FRENCH.....1951.....1989
Los Angeles, California
(Senior)

LYLE FRENCH 1954 2004
Scottsdale, Arizona

(Senior)

JAMES GALBRAITH.....1947.....1997
Birmingham, Alabama
(Senior)

HENRY GARRETSON…….1973……2007
Louisville, KY
(Senior)

SIDNEY GOLDRING.....1964.....2004
St. Louis, Missouri
(Senior)

EVERETT GRANTHAM.....1942.....1997
Louisville, Kentucky
(Senior)

JOHN GREEN.....1953.....1990
Phoenix, Arizona
(Senior)

JAMES GREENWOOD, JR. 1952.....1992
Houston, Texas
(Senior)

WESLEY GUSTAFSON.....1942.....1975
Jensen Beach, Florida
(Senior)

WALLACE HAMBY.....1941.....1999
Pompano Beach, Florida
(Senior)

HANNIBAL HAMLIN.....1949.....1982
Providence, Rhode Island
(Senior)

JOHN HANBERY.....1959.....1996
Palo Alto, California
(Senior)

JOHN HANKINSON……1973……..……..2007
Northumberland, England
(Senior Corresponding)

MAJOR GEN. GEORGE HAYES…1962.....2002
Washington, D. C.
(Senior)

E. BRUCE HENDRICK.....1968.....2001
Toronto, Ontario, CANADA
(Senior)

JESS HERRMANN .....1938.....1994
Oklahoma City, Oklahoma
(Senior)

HENRY HEYL.....1951.....1975
Hanover, New Hampshire
(Senior)

JULIAN HOFF……1975……..2007
Ann Arbor, MI
(Senior)

HAROLD HOFFMAN …….1982 2004
Toronto Ontario, Canada
(Senior)

WILLIAM HUNT.....1970.....1999
Columbus, Ohio
(Senior)

OLAN HYNDMAN.....1942.....1966
Iowa City, Iowa
(Senior)

KENNETH JAMIESON .....1970.....1976
Brisbane, AUSTRALIA
(Corresponding)

SIR GEOFFREY JEFFERSON1951 1961
Manchester, ENGLAND
(Honorary)

HANS-PETER JENSEN .....1980.....2000
Kiel, GERMANY
(Senior Corresponding)

RICHARD JOHNSON.....1974.....1997
Manchester, ENGLAND
(Senior Corresponding)

WILLIAM KEITH .....Founder.....1987
Toronto, Ontario, CANADA
(Senior)

ROBERT KING…….1958……2008
Syracuse, New York

(Senior)

KATSUTOSHI KITAMURA ……..1970……..2005
Japan
(Senior Corresponding)

ROBERT KNIGHTON 1966 2004
Cherry Valley, California
(Senior)

RICHARD KRAMER 1978 2001
Durham, North Carolina
(Inactive)

HUGO KRAYENBUHL..... 1974 1985
Zurich, SWITZERLAND
(Honorary)

KRISTIAN KRISTIANSEN . 1967 1993
Oslo, Norway
(Senior Corresponding)

THEODORE KURZE 1967 2002
Newport Beach, California
(Senior)

LAURI LAITINEN…….1972……...2007
FINLAND
(Senior Corresponding)

THOMAS LANGFITT 1971 2005
Philadelphia, Pennsylvania
(Senior)

WALPOLE LEWIN 1973 1980
Cambridge, ENGLAND
(Corresponding)

VALENTINE LOGUE 1974 2000
London, ENGLAND
(Honorary)

H.C. RUEDIGER LORENZ ……...1998……2008
Frankfurt, GERMANY
(Senior Corresponding)

HERBERT LOURIE 1965 1987
Syracuse, New York
(Senior)

JOHN LOWREY……1965…….2005
Kamuela, Hawaii
(Senior)

ALFRED LUESSENHOP ……..1977…….……..2009
Washington, DC
(Senior)

WILLEM LUYENDIJK.....1973.....1995
Oegstgeest, NETHERLANDS
(Senior Corresponding)

ERNEST MACK.....1956.....2000
Reno, Nevada
(Senior)

M. STEPHEN MAHALEY ...1972.....1992
Birmingham, Alabama
(Active)

LEONARD MALIS……..…….1973……..…….2005
Hollis Hills, New York
(Senior)

GEORGE MALTBY .....1942.....1988
Scarsborough, Maine
(Senior)

FRANK MARGUTH.....1978.....1991
Munich, GERMANY
(Senior Corresponding)

DONALD MATSON.....1950.....1969
Boston, Massachusetts
(Active)

FRANK MAYFIELD.....Founder.....1991
Cincinnati, Ohio
(Senior)

AUGUSTUS McCRAVEY ....1944.....1990
Chattanooga, Tennessee
(Senior)

KENNETH McKENZIE .....1960.....1964
Toronto, Ontario, CANADA
(Honorary)

J. MICHAEL MCWHORTER1989.....2004
Winston-Salem, North Carolina

(Senior)

WILLIAM MEACHAM 1952 1999
Nashville, Tennessee
(Senior)

JAMES MEREDITH..... 1946 1962
Richmond, Virginia
(Active)

J. DOUGLAS MILLER..... 1988 1995
Edinburgh, SCOTLAND
(Corresponding)

W. JASON MIXTER..... 1951 1968
Woods Hole, Massachusetts
(Honorary)

EDMUND MORRISSEY 1941 1986
San Francisco, California
(Senior)

FRANCIS MURPHEY .....Founder 1994
Naples, Florida
(Senior)

GOSTA NORLEN 1973 1985
Goteborg, SWEDEN
(Honorary)

FRANK NULSEN 1956 1994
Naples, Florida
(Senior)

SIXTO OBRADOR..... 1973 1978
Madrid, SPAIN
(Honorary)

GUY ODOM..... 1946 2001
Durham, North Carolina
(Senior)

PIETRO PAOLETTI..... 1989 1991
Milan, ITALY
(Corresponding)

WILDER PENFIELD..... 1960 1976
Montreal, Quebec, CANADA
(Honorary)

HELMUT PENZHOLZ 1978 1985
Heidelberg, WEST GERMANY
(Corresponding)

BERNARD PERTUISET 1986 2000
Paris, FRANCE
(Honorary)

HANS-WERNER PIA 1978 1986
Giessen, WEST GERMANY
(Corresponding)

J. LAWRENCE POOL 1940 2004
Canaan, CT
(Senior)

ROBERT PUDENZ 1943 1998
South Pasadena, California
(Senior)

JOHN E. RAAF ..... Founder 2000
Portland, Oregon
(Senior)

B. RAMAMURTHI 1973 2003
Tharamani, Chennai, INDIA
(Senior Corresponding)

AIDAN RANEY 1946 2002
Los Angeles, California
(Senior)

RUPERT B. RANEY 1939 1959
Los Angeles, California
(Active)

JOSEPH RANSOHOFF 1965 2001
Tampa, Florida
(Senior)

THEODORE RASMUSSEN . 1947 2002
Montreal, Quebec, CANADA
(Senior)

BRONSON RAY 1992 1993
New York, New York
(Honorary)

DAVID REEVES 1939 1970
Santa Barbara, California

(Active)

DAVID REYNOLDS 1964 1978
Tampa, Florida
(Active)

THEODORE ROBERTS……1976……2007
Seattle, Washington
(Senior)

R. C. L. ROBERTSON 1946 1985
Houston, Texas
(Senior)

STEWART ROWE 1938 1984
Pittsburgh, Pennsylvania
(Senior)

RICHARD SCHNEIDER..... 1970 1986
Ann Arbor, Michigan
(Senior)

KURT-FRIEDRICH SCHURMANN1978……2005
Mainz, GERMANY
(Senior Corresponding)

HENRY SCHWARTZ..... 1942 1998
St. Louis, Missouri
(Senior)

WILLIAM SCOVILLE..... 1944 1984
Hartford, Connecticut
(Senior)

R. EUSTACE SEMMES 1955 1982
Memphis, Tennessee
(Honorary)

C. HUNTER SHELDEN 1941 2003
Pasadena, California
(Senior)

ROBERT SMITH 1989 2003
Jackson, Mississippi
(Senior)

SAMUEL SNODGRASS..... 1939 1975
Galveston, Texas
(Senior)

GLEN SPURLING..... 1942 1968
La Jolla, California
(Honorary)

C. WILLIAM STEWART..... 1948 1948
Montreal, Quebec, CANADA
(Corresponding)

KENICHIRO SUGITA 1988 1994
Nagoya, Japan
(Senior Corresponding)

THORALF SUNDT, JR. 1971 1992
Rochester, Minnesota
(Active)
ANTHONY SUSEN……..1965…….2008
Burgess, Virginia
(Senior)

HENDRIK SVIEN 1957 1972
Rochester, Minnesota
(Active)

HOMER SWANSON..... 1949 1987
Atlanta, Georgia
(Senior)

WILLIAM SWEET 1950 2001
Brookline, Massachusetts
(Senior)

ALFRED UIHLEIN..... 1950 1990
Rochester, Minnesota
(Senior)

JOHN VAN GILDER (Kerstin)……1980……2007
Iowa City, IA
(Senior)

A. EARL WALKER 1938 1995
Albuquerque, New Mexico
(Senior)

EXUM WALKER (Nellie)……1938……2001
Atlanta, GA
(Senior)

ARTHUR WARD, JR...... 1953 1997
Seattle, Washington
(Senior)

THOMAS WEAVER, JR. 1943 1985
Dayton, Ohio
(Senior)

W. KEASLEY WELCH 1957 1996
Waban, Massachusetts
(Senior)

BENJAMIN WHITCOMB ... 1947 1998
Surrey, Maine
(Senior)

BARNES WOODHALL 1941 1985
Durham, North Carolina
(Senior)

FRANK WRENN 1973 1990
Greenville, South Carolina
(Senior)