THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY

72nd Annual Meeting

The Inn at Spanish Bay
Pebble Beach, California

November 3-6, 2010

American Association of Neurological Surgeons

Jointly Sponsored by AANS

FUTURE MEETINGS

2011

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

2012

September 26-29, 2012
TBD

Mark your calendars now!

GENERAL INFORMATION

HOTEL INFORMATION

The Inn at Spanish Bay
Pebble Beach Resorts
2700 17 Mile Drive
Pebble Beach, CA 93953

REGISTRATION DESK LOCATION AND HOURS:

Wednesday, November 3	Group Des	12:00 PM – 6:30 PM
Thursday, November 4	Group Desk	6:00 AM – 12:00 PM
Friday, November 5	Group Desk	6:00 AM – 12:00 PM
Saturday, November 6	Group Desk	6:00 AM – 12:00 PM

PROGRAM SUMMARY

WEDNESDAY, NOVEMBER 3

EVENTS	TIME	LOCATION
Registration	12:00 PM-6:30 PM	Group Desk
ABNS Advisory Council Meeting	1:30 PM-3:00 PM	Carnoustie Room
Academy Executive Comm. Mtg	3:00 PM-5:00 PM	Boardroom
Opening Reception	6:30 PM - 9:30 PM	St. Andrew's Room

THURSDAY, NOVEMBER 4

EVENTS	TIME	LOCATION
Registration	6:00 AM-12:00 PM	Group Desk
Continental Breakfast (Members)	6:30 AM-7:30 AM	St. Andrew's Room
Continental Breakfast (Spouse/Guest)	6:30 AM-10:30 AM	Peppoli
General Session	7:30 AM-1:00 PM	Ballroom
Pebble Beach Historian, Neil Hotelling	10:00 AM-10:30 AM	Peppoli
Point Lobos State Park	1:30 PM-4:30 PM	Ballroom Patio
Winery Tours of Monterey County	1:30 PM-5:00 PM	Ballroom Patio
Horseback Riding	2:00 PM-4:00 PM	1:00 p.m. Shuttle
Pebble Beach Golf Links	12:40 PM	Shuttle
Spyglass Hill Golf Course	12:20 PM	Shuttle
Reception	6:30 PM-7:30 PM	Fairway Troon Patio
Dinner	7:30 PM-10:30 PM	Ballroom

FRIDAY, NOVEMBER 5

Registration	**6:00 AM-12:00 PM**	**Group Desk**
Breakfast (Members)	**6:30 AM-7:30 AM**	**St. Andrew's Room**
Breakfast (Spouse and Guest)	**6:30 AM-10:30 AM**	**Peppoli**
General Session	**7:30 AM-1:00 PM**	**Ballroom**
Book Club, "Cannery Row" by John Steinbeck	**10:00 AM-11:00 AM**	**Peppoli**
Biking Tour	**2:00 PM-4:00 PM**	**Ballroom Gallery**
Sea Kayaking on Stillwater Cove	**1:30 PM-4:00 PM**	**Ballroom Gallery**
Whale Watching Cruise	**1:00 PM-4:30 PM**	**Ballroom Gallery**
Links at Spanish Bay	**12 noon**	**Shuttle**
Reception	**6:00 PM-7:00 PM**	**Beach Club**
Black Tie Optional Dinner	**7:00 PM-10:00 PM**	**Beach Club**

SATURDAY, NOVEMBER 6

Registration	**6:00 AM-12:00 PM**	**Group Desk**
Breakfast (all together)	**6:30 AM-10:30 AM**	**Peppoli**
General Session	**7:30 AM-1:00 PM**	**Ballroom**

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72nd Annual Meeting

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Mission Statement:
The purpose of the live Academy meeting shall be to promote scientific and social intercourse among its members, to foster neurological surgery as specialty of medicine, to encourage and sponsor basic and clinical research activity in the neurological sciences, and to promote the knowledge and skill of those who devote themselves to neurological surgery in accordance with the high ideals of the medical profession.

This activity will include live presentations from faculty to include case presentations, discussion, as well as time for questions and answers.

American Academy of Neurological Surgery

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Learning Objectives

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

Critique the safety, efficacy and overall value of surgical and non-surgical options in neurological surgery.

Discuss the potential applicability of new technologies to the treatment of complex cranial, spinal and peripheral nerve disorders.

Discuss the evolving landscape of resident education and its impact on neurosurgical training.

Evaluate the relevance of research methodologies and presented findings and their potential usefulness in clinical practice of neurological surgery

Accreditation Statement

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the American Association of Neurological Surgeons (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.

Intended Audience/background requirement
The scientific program presented is intended for neurosurgeons either in training or in active practice.

Designation Statement
The AANS designates this live educational activity for a maximum of 12.75 hours of *AMA PRA Category 1 Credit*s™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

AANS Disclaimer Statement
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Name	Conflict of Interest	Company
Adelson, PD	University Grants/Research Support	NIH
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Asher, A	Industry Grant Support	Salient Medical Technologies
	Consultant Fee	Salient Medical Technologies
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Barnett, G	Consultant fee	Monteris Medical
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Bendock, B	Industry Grant Support	MicroVention
Cauldwell, W	Honorarium	Dartmouth, Mayo Clinic, University of Maryland
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Gunel, T	University Grants/Research Support;	NIH

Hopkins, LN	University Grants/Research Support	Toshiba (for the Toshiba Stroke Research Center)
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Lawton, MT	Other Financial or Material Support	Mizuho America (Royalty)
Lee, K	University Grants/Research Support Consultant Fee Other Financial or Material Support	Mayo Clinic; Medtronic, inc licensed patent pending to St. Jude Medical, Inc
Lesniak, M.S.	University Grants/Research Support	National Institutes of Health
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Liu, C	Consultant Fee	Integra
MacDonald, L	University Grants/Research Support	Physicians Services Incorporated Foundation, Brain Aneurysm Foundation
	Industry Grant Support Stock or Shareholder Employee [any industry]	Actelion Pharmaceuticals Edge Therapeutics, Inc Edge Therapeutics, Inc
Midha, R	University Grants/Research Support	Canadian Institute for Health Research (CIHR)
	Industry Grant Support;	Integra Life Sciences Foundation Support for peripheral nerve fellowship

Newell, D	University Grants/Research Support	NIH, Life sciences Discovery fund
Rezai, A	Consultant fee	Educational consultant, Medtronic, ? Technologies
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Roberts, D	University Grants/Research Support	NIH
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Rutka, J	University Grants/Research Support	CIHR, CCSRI, PBTF
Siddiqui, A.H.	University Grants/Research Support	National Institutes of Health, University at Buffalo (Research Development Award)
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Sloan, A	Consultant fee;	Monteris
Spetzler, R	Consultant fee	Zeiss, Anspach, Medtronic, Codman
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Disclosures Pending

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AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
PRELIMINARY SCIENTIFIC PROGRAM AGENDA

THURSDAY, November 4, 2010		
Time	**Presentation**	**Presenter**
7:30 – 8:30 7:30 – 7:42 7:42 – 7:54 7:54 – 8:06 8:06 – 8:18 8:18 – 8:30	**VASCULAR FORUM** Update on Neurovascular Imaging CREST and Implications for CarotidRevascularization Outcomes, Complications, and Issues with the Pipeline Device BRAT and Implications for Aneurysm Surgery Questions and Discussion	 Meng Law, MD Nick Hopkins, MD Kim Nelson, MD Robert Spetzler, MD Panel
8:30 – 8:42	Results of AutoLITT First-in-Man Trial of Laser Interstitial Thermal Therapy for Recurrent Glioblastoma	Gene H. Barnett, MD
8:44 – 8:56	The Role of the Cofilin Pathway in Human Glioma Migration and Invasion	James T. Rutka, MD, PhD
8:58 – 9:10	The Functional Outcome and Morbidity Profile Associated with Resection of Gliomas in the Cingulate Gyrus	Mitchel S. Berger, MD
9:12 – 9:24	Safety and Efficacy of Superselective Intraarterial Cerebral Infusion of Bevacizumab after Osmotic Blood Brain Barrier Disruption for Recurrent Malignant Glioma	Howard A. Riina, MD
9:26 – 9:38	Indirubin-Mediated Reduction of GSK-3 Activity Leads to Reduced Tumor and Blood Vessel Cell Migration: A Novel Treatment in Experimental Glioblastoma Initiating Cell Neoplasms	E Antonio Chiocca, MD, PhD
9:40 – 9:52	Petroclival Meningiomas: Study On Outcomes, Complications and Recurrence Rates	Anil Nanda, MD
9:54 – 10:06	Staged Resection of Large Acoustic Neuromas: Indications, Surgical Results, Facial Nerve Outcomes, and Complications	Johnny B. Delashaw, MD
10:08 – 10:28	BREAK	

10:28 – 10:40	Pituitary Tumor Heritability	William T. Couldwell, MD
10:42 – 10:54	Outcomes of Endoscopic Transsphenoidal Surgery for Acromegaly: Rates of Remission, Complications, and Predictors of Outcome	John A. Jane, Jr, MD
10:56 – 11:08	Intraoperative CT Registration and Electromagnetic Neuronavigation for Transsphenoidal Pituitary Surgery: Accuracy and Time-Effectiveness	Marc Mayberg, MD
11:10 – 11:22	Closure of Intraoperative CSF Leaks in Trans-sphenoidal Surgery	Martin Weiss, MD
11:24 – 11:36	Effective Treatment of Disseminated Medulloblastoma with Modified Measles Virus in a Murine Model	Corey Raffel, MD, PhD
11:38 – 11:50	Medulloblastoma Comprises Four Distinct Diseases	Michael A. Taylor, MD
11:52 – 12:04	Whole Brain Radiotherapy and Stereotactic Radiosurgery in the Treatment of Brain Metastasis: The Randomized Clinical Trials	Phillip A. Tibbs, MD
12:06 – 12:18	Feasibility of Laser Intersititial Thermal Therapy With Real-Time MR Thermography for Critically Placed Brain Tumors	Michael Schulder, MD
12:20 – 12:32	Comparative Effectiveness Research in Spinal Surgery	Daniel K. Resnick, MD
FRIDAY, November 5, 2010		
7:30 – 7:42	Evacuation of Spontaneous Intracerebral Hemorrhage Using Sonothrombolysis	David W. Newell, MD
7:44 – 7 :56	Adenosine for Temporary Flow Arrest During Intracranial Aneurysm Surgery: A Single Center Retrospective Review	Bernard R. Bendok, MD
7:58 – 8:10	The Search for the Source of Brain Adenosine	H. Richard Winn, MD
8:12 – 8:24	Duel Regulation of Post Ischemic Neurogenesis by TFG-β1	Robert J. Dempsey, MD
8:26 – 8:38	Surgery for the Cavernous Angioma in the Brainstem	Kazuhiro Hongo, MD

8:40 – 8:52	Supratentorial Cavernous Malformations in Eloquent and Deep Locations: Surgical Approaches and Outcomes	Michael T. Lawton, MD
8:54 – 9:06	Thrombospondin-1 Modulates the Angiogenic Phenotype of Human Cerebral Arteriovenous Malformation Endothelial Cells	Charles Y. Liu, MD, PhD.
9:08 – 9:20	Intracranial Aneurysm Risk Genes Identified through Whole Genome Association Study	Murat Gunel, MD
9:22 – 9:34	Hemodynamic-Morphologic Discriminants for Intracranial Aneurysm Rupture	Adnan H. Siddiqui, MD, PhD
9:36 – 9:48	Effect of Drug Treatment on Vasospasm, Delayed Cerebral Ischemia and Functional Outcome After Aneurysmal Subarachnoid Hemorrhage – a Systematic Review and Meta-Analysis	R. Loch MacDonald, MD, PhD
9:50 – 10:02	Basilar Occlusion for Complex Basilar Artery Aneurysms: An Old Technique for New Age Neurosurgery	Robert A. Solomon, MD
10:04 – 10:16	Management of Cervical AVMs	Robert Spetzler, MD
10:18 – 10:38	BREAK	
10:38 – 10:50	Augmenting Adult Hippocampal Neurogenesis Using Targeted Brain Stimulation: Implications for Memory Networks	Scellig Stone, MD
10:50 – 11:50 10:50 – 11:05 11:05 – 11:20 11:20 – 11:35 11:35 – 11:50	**HALL OF FAME FORUM**	 Bennett Stein, MD Charles Wilson, MD John Jane, Sr, MD Panel
11:50 – 12:35	**Presidential Address**	Steven Giannotta, MD

SATURDAY, November 6, 2010		
7:30 – 8:30	**CONCUSSION FORUM**	
7:30 – 7:45 7:45 – 8:00 8:00 – 8:15 8:15 – 8:30	Traumatic Brain Injury and Concussion Science of Sports Injury Report on the NFL Committee Questions and Discussion	Geoff Manley, MD Kevin Guskiewicz, PhD Hunt Batjer, MD Panel
8:30 – 8:42	Experimental Traumatic Brain Injury Alters the Organization of the Mossy Fiber Projection in the Immature Rat Hippocampus	P. David Adelson, MD
8:44 – 8:56	Poloxamer-188 and Bumetanide: Potential Combinatorial Neuroprotective Therapy in an Experimental Model of Neural Injury	David M. Frim, MD, PhD
8:58 – 9:10	A Prospective Randomized Trial Comparing Expansile Cervical Laminoplasty Versus Cervical Laminectomy and Fusion for Multi-level Cervical Myelopathy	Allan D. Levi, MD, PhD
9:12 – 9:24	Temporal Lobectomy for Medically Intractable Epilepsy: Effect of the Extent of Hippocampal Resection in Patients with Hippocampal Sclerosis and with Normal Pathology	Richard Byrne, MD
9:26 – 9:38	Human Wireless Electrochemical Recordings During Deep Brain Stimulation Neurosurgery Using the wincs System in Parkinson's and Essential Tremor Patients	Kendall H. Lee, MD, PhD
9:40 – 9:52	Stereotactic Radiosurgery for Glossopharyngeal Neuralgia: Preliminary Report of 4 Cases	Bruce E. Pollock, MD
9:54 – 10:06	Deep Brain Stimulation (DBS) of the Ventral Capsule/Ventral Striatum (VC/VS) for the Treatment of Neurobehavioral Disorders	Ali R. Rezai, MD
10:08 – 10:28	BREAK	
10:28 – 10:40	Dynamics of the Deep-Brain Stimulator Tissue-Electrode Interface	Karl Sillay, MD

10:42 – 10:54	Deep Brain Stimulation of the Subthalamic Nucleus in Primary Cervical Dystonia: Results of a Pilot Trial	Philip A. Starr, MD, PhD
10:56 – 11:08	The Effects of Disease and Aging on Neural Grafts: Implications for Future Stem Cell Therapies	Thomas B. Freeman, MD
11:10 – 11:22	Dose and Duration of Nerve Growth Factor (NGF) Administration Determine Behavioral Recovery Following Peripheral Nerve Injury	Rajiv Midha, MD
11:24 – 11:36	A New Treatment Paradigm: Neo-Adjuvant Radiosurgery Prior to Surgical Resection of Brain Metastases with Analysis of Local Tumor Recurrence	Anthony L. Asher, MD
11:38 – 11:50	Bone Marrow Mesenchymal Stem Cells Loaded with an Oncolytic Adenovirus Suppress the Anti-Adenoviral Immune Response in an Immunocompetent Model	Maciej S. Lesniak, MD
11:52 – 12:04	CpG Island DNA Methylation Sites are Associated with Malignant Potential in Meningiomas	Andrew E. Sloan, MD
12:06 – 12:18	MP-MUS (I): A Novel MAO-B Activated Pro-Drug which Specifically Targets and Destroys Gliomal Mitochondria	David S. Baskin, MD
12:20 – 12:32	Intratumoral Hemorrhage and Fibrosis in Vestibular Schwannoma: A Possible Mechanism for Hearing Loss	Andrew T. Parsa, MD, PhD
12:34 – 12:46	Quantitative Fluorescence in Intracranial Tumor: Implications for Ala-Induced Ppix as an Intraoperative Marker	David W. Roberts, MD

PAPER PRESENTATIONS

8:30 – 8:42 AM RESULTS OF AUTOLITT FIRST-IN-MAN TRIAL OF LASER INTERSTITIAL THERMAL THERAPY FOR RECURRENT GLIOBLASTOMA

Gene H. Barnett, M.D., M.B.A., F.A.C.S.; Andrew E. Sloan, M.D., F.A.C.S.; Mark G. Torchia, Ph.D.

INTRODUCTION: The use of laser interstitial thermal therapy (LITT) as a treatment for human brain tumors dates to the early 1990s, but the recent introduction of two commercial LITT systems aimed at treatment of these disorders may bring the technology into the surgical armamentarium. The results of the first human investigation of the AutoLITT (Monteris Medical, Winnipeg, Canada) system for recurrent glioblastoma are presented.

METHODS: This was a Phase I dose-escalation trial in patients with biopsy-proved supratentorial recurrent glioblastoma of maximum 4cm cross sectional dimension. Patients underwent frequent clinical and radiographic examinations for two weeks, after which they could engage in additional glioblastoma therapies. All patients were followed to death. The AutoLITT system is comprised of a gas-cooled side-firing laser probe (coupled to a 1064 nm wavelength diode laser), positioning/manipulation device, and software predicting likelihood of thermal injury/ablation while the procedure is monitored using real-time magnetic resonance thermometry. The system allows the surgeon to control depth and rotation of the laser beam to provide conformal thermal coagulation during the procedure. The trial was conducted at Cleveland Clinic (CC) and University Hospitals Case Medical Center (UHCMC).

RESULTS: Between November 2008 and October 2009, eleven patients were enrolled and ten treated. Average age was 56.1 years and 78% were male. Average interval between diagnosis and treatment was 494 days (range 111 – 1056). Four (40%) improved, five (50%) stayed stable, and one (10%) worsened neurologically in the two-week follow-up period. All treated patients developed tissue necrosis apparent on 24 and 48-hour post procedure scans that correlated with the lines of thermal damage threshold generated during the procedures. One patient suffered hemiplegia which resolved gradually but incompletely, and two developed steroid-reversible neurologic deficits related to post-treatment brain edema. One developed a delayed intracerebral hemorrhage due to a treatment-related pseudoaneurysm.

CONCLUSIONS: The AutoLITT system provided accurate thermal coagulation of recurrent glioblastoma. More detailed results and strategies to prevent neurological complications will be discussed.

THURSDAY, NOVEMBER 4

8:44 – 8:56 AM **THE ROLE OF THE COFILIN PATHWAY IN HUMAN GLIOMA MIGRATION AND INVASION**

James T Rutka, MD, PhD, Orlando Moreno, MD, Shoichi Nagai, MD, PhD, Christian Smith, PhD

INTRODUCTION: The cofilin pathway plays a central role in the regulation of actin polymerization and the formation of protrusions that are essential for cell migration. Phosphorylation of cofilin is a key regulatory mechanism modulating cofilin activity. Cofilin expression is altered in a variety of cancers including ovarian, renal cell, and oral-squamous carcinomas. It is clear that the expression of cofilin and other proteins in the cofilin pathway such as Rac and LIMK are upregulated in invasive tumor cells, and that the activation status of cofilin may be directly linked to tumor invasion. To date, the role of cofilin in human glioma migration and invasion has not been studied or described.

MATERIALS AND METHODS: We examined the expression of cofilin by immunohistochemistry using a glioma tissue microarray (TMA) containing over 60 specimens. We performed immunocytochemistry for cofilin, phosphorylated cofilin, and LIMK1 in a panel of glioma cell lines. Knockdown of cofilin expression was reliably achieved using siRNAs. The migration and invasiveness of glioma cell lines before and after cofilin manipulation was determined *in vitro* and *in vivo* using several model systems.

RESULTS: Cofilin expression was increased on the glioma TMA, and correlated with increasing grade malignant astrocytoma. In addition, both cofilin and LIMK had elevated expression in glioma cell lines. Targeted knockdown of cofilin altered glioma cell morphology and inhibited glioma migration and invasion in vitro. In contrast, overexpression of a cofilin phosphorylation mutant in an in vivo xenograft model of brain tumors resulted in a marked accentuation of the invasive phenotype in 10/10 (100%) of mice. Invasive features found *in vivo* included spread to the contralateral cerebral hemisphere across the corpus callosum, penetration along arteriolar spaces, and diffuse leptomeningeal disease.

CONCLUSION: These data show for the first time the role of cofilin in human glioma invasion. They also indicate that the cofilin pathway, which lies downstream of the small cytoskeletal Rho-GTPases, may represent a novel therapeutic target to ablate invasion in these highly malignant tumors.

THURSDAY, NOVEMBER 4

8:58 – 9:10 AM **ASSESSMENT OF MORBIDITY FOLLOWING RESECTION OF CINGULATE GYRUS GLIOMAS**

Matthew Tate, MD, PhD, Chae-Yong Kim, MD, PhD, Edward Chang, MD, Mei-Yin Polley, PhD, and ***Mitchel S. Berger, MD***

OBJECT: The morbidity associated with resection of tumors in the cingulate gyrus (CG) is not well established. The goal of this present study is to define the short- and long-term morbidity profile associated with resection of gliomas within this region.

METHODS: Ninety consecutive patients with gliomas involving the CG were analyzed. Resections were classified by zones corresponding to functionally defined regions of the CG as follows: Zone I (perigenual, anterior), Zone II (midcingulate), Zone III (posterior), and Zone IV (retrosplenial). Basic demographic, imaging, operative details, and pre-and postoperative neurological examinations were recorded for each patient. Patients in whom neurological morbidity was documented during their initial postoperative examination who did not completely improve by the 6-month follow-up examination were considered to have a permanent deficit. For each patient with surgery-related morbidity, postoperative MR imaging and operative notes were reviewed, and the cortical regions incorporated in the surgery trajectory were recorded. The analysis was carried out for tumors confined to the CG(>90% of tumor contained within the CG) as well as those involving the CG but extending into adjacent cortical structures.

RESULTS: Analysis of the entire patient cohort demonstrated that 29% of patients experienced a new or worsened neurological deficit immediately after surgery. The most common deficits were supplementary motor area (SMA) syndrome (20%), weakness (20%), and sensory changes (2&). All patients with an SMA syndrome in our series had intentional resection of SMA as part of the surgical approach. Patients with resections including Zone II or III had a higher rate of total morbidity and SMA syndrome than patients with Zone I resections (p>0.0.5). Only 4% of patients had a persistent neurological deficit at 6 months postoperatively. A similar morbidity profile was observed in the subset analysis of patients with tumors confined to the CG, with no additional morbidity related to known cingulate-specific functions.

CONCLUSIONS: Resection of gliomas involving the CG can be performed with minimal, predictable long-term morbidity (>5%). Surgical morbidity is primarily a function of surgical trajectory rather than the particular cingulate region resected. (DO1: 10.3171/2010.JNS10709)

THURSDAY, NOVEMBER 4

9:12 – 9:24 AM **SAFETY AND EFFICACY OF SUPERSELECTIVE INTRAARTERIAL CEREBRAL INFUSION OF BEVACIZUMAB AFTER OSMOTIC BLOOD BRAIN BARRIER DISRUPTION FOR RECURRENT MALIGNANT GLIOMA**

Howard A. Riina, MD, FACS., John A. Boockvar, MD

INTRODUCTION/HYPOTHESIS: Glioblastoma multiforme (GBM) is a uniformly fatal disease with a median survival of approximately 15 months. Recent monoclonal antibody therapies such as bevacizumab (Avastin) delivered intravenously (IV) have been shown to be active in GBM and to prolong survival in patients with recurrent malignant glioma. The objectives of this study were to determine the safety (maximum tolerated dose or MTD) and activity of transient blood brain barrier disruption with IA mannitol followed by super-selective intraarterial cerebral infusion (SIACI) of bevacizumab for recurrent malignant glioma. A secondary objective was to determine radiographic response utilizing MR imaging and MR perfusion.

METHODS: Patients with recurrent glioblastoma multiforme (GBM) and anaplastic astrocytomas (AA) received osmotic blood brain barrier disruption with intraarterial (IA) mannitol (25% in 10cc over 60 seconds) followed by superselective intraarterial cerebral infusion (SIACI) of bevacizumab starting at a dose of 2 mg/kg (3 patients per cohort for a total of 5 cohorts) with dose escalation up to a dose of 10 mg/kg. Dose limiting toxicity (DLT) was assessed over a four-week period. Treatment response was assessed on MR imaging using the WHO-based MacDonald criteria and volumetric analysis four weeks after the infusion and prior to starting the standard biweekly IV bevacizumab (10mg/kg) protocol. Changes in intratumoral MR perfusion and MR spectroscopy were included in the post infusion imaging assessment. Response to IA chemotherapy was correlated to previous exposure to IV bevacizumab and to tumor expression levels of VEGF on immunohistochemistry.

RESULTS: Fifteen patients received a single dose of IA mannitol followed by SIACI of bevacizumab prior to starting biweekly bevacizumab therapy. No DLT was observed during the 28-day observation period after using SIACI bevacizumab up to a dose of 10mg/kg. No patients discontinued treatment because of Grade 3 central nervous system intratumoral hemorrhage, wound dehiscence, or bowel perforation. . MR imaging within 28 days of treatment prior to the initiation of IV chemotherapy showed that 2 patients (13%) had a partial response, 11 patients (73%) had stable disease, and 2 patients (13%) had progressive disease of the targeted neoplasm. Post-infusion MRI at 28 days showed a mean tumor area reduction of 12.1% (SD 43.3%) and volume increase of 11.3% (SD 93.8%). MRI perfusion demonstrated that IA bevacizumab concurrently diminished locoregional relative cerebral blood volume (rCBV) by 20.7% (SD 29.7).
Conclusion: SIACI of mannitol followed by bevacizumab for recurrent malignant glioma (up to 10mg/kg) is safe and well tolerated. Radiographic responses suggest that this delivery method can act locally and after a single dose in some patients with recurrent malignant glioma.

THURSDAY, NOVEMBER 4

9:26 – 9:38 AM **INDIRUBIN-MEDIATED REDUCTION OF GSK-3 ACTIVITY LEADS TO REDUCED TUMOR AND BLOOD VESSEL CELL MIGRATION: A NOVEL TREATMENT IN EXPERIMENTAL GLIOBLASTOMA INITIATING CELL NEOPLASMS**

Shanté P Williams, BS, Michal O Nowicki, PhD, Fang Liu, BS, Rachael Press, ***E Antonio Chiocca, MD PhD,*** & Sean E Lawler, PhD

The invasive nature of gliomas is a major obstacle to effective therapy, and anti-invasive therapeutic strategies are in demand. Here we report that treatment of both glioma cells and glioma initiating cell-enriched neurospheres with GSK-3 inhibitors of the indirubin family leads to reduced migration both *in vitro* and *in vivo* and improves survival in tumor bearing mice. These effects on migration were mediated at least in part by effects on □-catenin. Treatment of mice bearing invasive intracranial human glioma initiating cell xenografts with 6-bromo-indirubin acetoxime (BIA) led to reduced invasion of surrounding normal brain tissue. Interestingly, BIA treatment also led to decreased tumor growth, with a significant improvement in animal survival. Histologic examination showed a substantial decrease in blood vessel density in tumors from treated animals. *In vitro* studies showed that BIA was also able to block migration of endothelial cells. This data suggests that *in vivo* anti-invasive glioma therapy with GSK-3 inhibitors, not only inhibits invasion of tumor cells, but also blocks angiogenesis, slowing tumor growth, and provides a novel treatment paradigm for invasive gliomas.

THURSDAY, NOVEMBER 4

9:40 – 9:52 AM **PETROCLIVAL MENINGIOMAS: STUDY ON OUTCOMES, COMPLICATIONS AND RECURRENCE RATES**

Anil Nanda MD, FACS, Vijayakumar Javalkar MD, Anirban Deep Banerjee MD

OBJECT: Petroclival meningiomas are notoriously difficult lesions to manage surgically, given the critical neurovascular structures intimately associated with the tumors. The aim of the study was to review our series of patients with petroclival meningiomas who underwent surgical treatment; emphasis was placed on evaluating modes of presentation, postoperative neurological outcome, complications, and recurrence rates.

METHODS: Fifty patients underwent surgical treatment for petroclival meningiomas. We retrospectively reviewed their medical records, imaging studies and pathology reports to analyze presentation, surgical approach, neurological outcomes, complications and recurrence rates.

RESULTS: Majority of them were women (72%). Headache was the commonest presentation (58%). The most commonly used approaches were transpetrous (n=16) followed by orbito-zygomatic approach (n=13). Gross total resection was performed in 14 patients (28%) and in the remaining cases a residual was left behind (72%). 18 patients with residual were treated with gamma knife. New postoperative cranial neuropathies were noted in 22 patients (44%). The most common cranial nerve deficit following surgery was third cranial nerve dysfunction (n=11) and facial weakness (n=10). In 9 patients cranial nerve dysfunction was transient (41%). 7 patients had permanent dysfunction (32%). 8 patients developed hydrocephalus and all required placement of 3 ventriculoperitoneal (VP) shunt. CSF leak was noted in only two cases (4%). Wound dehiscence in one patient. Both CSF leaks and the wound dehiscence occurred in patients who were undergoing re-operations. Adequate radiographic follow-up (minimum 6 months) was present for 31 patients (62 %). Mean follow-up was 22.1 months. In six patients, tumor progression or recurrences were noted. Time to recurrence averaged 62.3 months (range 24-156 months). At the time of discharge, 92% of the patients had good outcome (GOS 5, 4). Three patients died due to causes not directly related to the surgery.

CONCLUSION: Petroclival meningiomas still pose a formidable challenge to neurosurgeons. In our series, we used multiple skull base approaches and careful micro neurosurgical technique to achieve a good functional outcome (GOS 4 or 5) in 92 % of patients. Our primary surgical goal was to achieve maximal tumor resection while maintaining or improving neurological function. We favor the treatment of residual tumor or recurrent tumor with SRS

THURSDAY, NOVEMBER 4

9:54 – 10:06 AM

STAGED RESECTION OF LARGE ACOUSTIC NEUROMAS: INDICATIONS, SURGICAL RESULTS, FACIAL NERVE OUTCOMES, AND COMPLICATIONS

Johnny B. Delashaw, Jr. M.D. **and Sean O. McMenomey, M.D.**

INTRODUCTION: Staged resection of large acoustic neuromas (ANs) as a strategy to improve facial nerve outcomes and morbidity has been proposed. We report our experience with two-staged resection of large ANs and analyze the indications, facial nerve outcomes, surgical results, and complications. These results were compared to a similar cohort of patients who underwent single-staged resection.

METHODS: A retrospective study was performed on 30 patients who underwent surgery for large ANs (size ≥ 3 cm) at OHSU. From 2002 to 2006, 18 patients underwent two-staged resection. A first stage retrosigmoid approach (without meatal drilling) was performed to remove the cerebellopontine angle portion of the tumor and to decompress the brainstem. A decision to stage the operation was made intraoperatively if there was cerebellar/brainstem edema, excessive tumor adherence to the facial nerve or brainstem, a poorly stimulation facial nerve, or thinned/splayed facial nerve. A second stage translabyrinthine approach was performed at a later date to remove remaining tumor. The remaining 12 patients who underwent single-staged resection underwent a retrosigmoid approach with meatal drilling. Patients were evaluated for tumor size, extent of resection, tumor recurrence, House-Brackmann (HB) facial nerve function, and complications.

RESULTS: In the two-stage group, the average tumor size was 4 cm (range, 3 to 7 cm) with mean follow-up of 27 months (range, 4 to 52 months). Gross or near total resection was achieved in 94.4%. Anatomic facial nerve preservation was achieved in all but one patient (94.4%). There were no recurrences on follow-up imaging. Fifteen patients (83.3%) had a HB grade 1-2, one (5.6%) had a HB grade 4, two (16.7%) had a HB grade 3-4, and three (25%) had a HB grade 5-6. There were no significant differences in complications between the two groups. There were no postoperative strokes, hemorrhages, or deaths in either group.

CONCLUSIONS: Staged resection of large ANs is an effective strategy to improve facial nerve outcomes. There does not appear to be added neurologic morbidity with staged resections. Since this preliminary data was analyzed, the Oregon Skull base team has routinely performed a two-staged operation for ANs ≥ 3 cm in size. The results of facial nerve function for this group (2007-2010) of patients will also be presented.

THURSDAY, NOVEMBER 4

10:28 – 10:40 AM **PITUITARY TUMOR HERITABILITY**

William T. Couldwell, Lisa Cannon-Albright

Pituitary tumors are prevalent in the general population, with a frequency of nearly 20 percent. The authors have analyzed the Utah Population Data Base (UPDB), a resource combining a computerized genealogy of the Utah population with a statewide tumor registry, to investigate familial clustering of pituitary tumors. Data from the UPDB has been used to evaluate the genetic contribution to specific phenotypes using different methods. The first method, which was developed specifically for use with the UPDB, looks at the estimation of the average relatedness among affected individuals who share a specific phenotype, also termed the Genealogical Index of Familiality (GIF). In addition, relative risk (RR) of developing disease in relatives of affected individuals can be estimated, and high-risk pedigrees can be identified. This work has demonstrated that the relative risks for first- and third-degree relatives were significantly elevated (RR = 2.83 and 1.63, respectively) among a group of 714 individuals with pituitary tumors. The average pairwise relatedness of pituitary tumor cases was significantly higher than expected, even when close relationships were ignored. The significantly elevated risks to relatives as well as the significant excess distant relatedness observed in cases provide strong support for a genetic contribution to predisposition to pituitary tumors, which helps with counseling family members of affected individuals. Multiple high-risk pedigrees can be identified in the UPDB, and study of such pedigrees is ongoing to identify gene(s) responsible for this heritability. An update on the high risk pedigree study will be presented, with search for candidate genes responsible for the development of these tumors.

THURSDAY, NOVEMBER 4

10:42 – 10:54 AM **OUTCOMES OF ENDOSCOPIC TRANSSPHENOIDAL SURGERY FOR ACROMEGALY: RATES OF REMISSION, COMPLICATIONS, AND PREDICTORS OF OUTCOME**

John A. Jane, Jr., M.D

OBJECT: The outcomes of the endoscopic approach for growth hormone adenomas have not been clearly defined.

METHODS: Sixty patients with Growth Hormone adenomas were identified who underwent pure endoscopic transsphenoidal surgery. Their medical records and imaging were reviewed. Surgical remission was defined as a normal IGF-1 or GH suppression to less than 1 ng/ml during OGTT. Clinical covariates predicting remission with a univariate p-value <0.10 were included in multivariate analysis.

RESULTS: Biochemical remission was achieved in 49 of 60 patients (82%), including all 14 microadenomas and 35 of 46 (76%) of macroadenomas. Knosp 0-2 tumors were associated with a significantly increased rate of remission (43/45, 95.5%) compared to Knosp 3-4 tumors (6/15, 40%). In multivariate analysis pre-operative variables predictive of remission include age (OR=0.86, 95% CI 0.77-0.98, p=0.022), Knosp (OR=3.21, 95% CI 1.38-7.44, p=0.007), and pre-operative growth hormone level (OR 1.03, 95% CI 1.01-1.06, p=0.023). Postoperative GH levels <3 ng/ml provided the best prediction of remission (p<0.001) with a sensitivity of 98% and a specificity of 82%.

New postoperative diabetes insipidus occurred in 6.8%, and 5.3% of patients required new steroid replacement beyond two months. New hypogonadism occurred in 25% of men and 14% of women. However, 41% of men had normalized testosterone levels and 67% of amenorrheic women regained menses postoperatively. The most common complaints at follow up were sinonasal (36/60, 60%). All but 2 patients ultimately experienced resolution of their sinonasal symptoms. Major complications occurred in 3 patients including one CSF leak, one pseudoaneurysm, and one postoperative meningitis.

CONCLUSIONS: The endoscopic transsphenoidal resection of GH adenomas is associated with high rates of remission and low incidence of new endocrinopathy. In spite of the panoramic views, cavernous sinus invasion predicts a lower rate of remission. The EAS identifies patients at high risk of failure, but further prospective analysis is warranted.

Table 1. Overall Cohort Characteristics

Variable	**N=60 (%)**
Percent Male	33 (55)
Age	48±13.3 (14-73)
Knosp	
0	27 (44.3)
1	15 (24.6)
2	4 (6.6)
3	7 (11.5)
4	8 (13.1)
Prior surgery	10 (16.7)
Bitemporal hemianopsia	7 (11.7)
Pre-operative GH	23
Pre-operative IGF	727
Tumor Size	
0-9mm	14
10-19mm	24
20-29mm	16
30-40mm	4
>40mm	2

THURSDAY, NOVEMBER 4

10:56 – 11:08 AM **INTRAOPERATIVE CT REGISTRATION AND ELECTROMAGNETIC NEURONAVIGATION FOR TRANSSPHENOIDAL PITUITARY SURGERY: ACCURACY AND TIME-EFFECTIVENESS**

Marc Mayberg, MD, Paula Eboli , MD, Bob Shafa , MD

OBJECTIVE: To assess the feasibility, anatomical accuracy and cost effectiveness of frameless electromagnetic (EM)-guided neuronavigation in conjunction with portable intraoperative CT (iCT) registration for transsphenoidal adenomectomy (TSA).

METHODS: A prospective database was established for 208 consecutive patients who underwent TSA using iCT/EM, and compared to a retrospective cohort of 65 consecutive preceding patients who underwent fluoroscope-assisted TSA by the same surgeon. All patients in both groups had trans-nasal removal of pituitary adenomas or neuroepithelial cysts using identical surgical technique with an operating microscope. The iCT/EM patients had a portable iCT scan performed immediately prior to surgery for registration to the EM navigation system, which did not require rigid head fixation. Pre-existing (non-navigation protocol) MRI scans were fused to the iCT images to enable three-dimensional navigation based upon MRI data. Accuracy of the navigation system was determined in the first 50 iCT/EM cases by visual concordance of the navigation probe location to 5 pre-selected bony landmarks. For all patients in both cohorts , total operating room time, incision-to-close time and relative costs of imaging and surgical procedures were determined from hospital records.

RESULTS: In every case, intraoperative registration using iCT images was successful and pre-operative MRI images were fused to iCT without affecting navigation accuracy. There was 100% concordance between probe tip location and pre-determined bony loci in the first 50 iCT/EM cases. Total OR time was significantly less in the iCT/EM cases (mean=108.9 +/-24.3 min; N=208) compared to the fluoroscopy group (mean=121.1 +/-30.7 min; N=65; $p<0.001$). Similarly, incision-to-close time was significantly less for the iCT/EM cases (mean=61 .3+/-18.2 min) versus the fluoroscopy group (mean = 71.75 +/-19.0 min; $p<0.001$). Relative overall costs for iCT/EM and intraoperative C-arm
fluoroscopy were comparable; increased costs for navigation equipment were offset by savings in operating room costs for shorter procedures.

CONCLUSION: The use of iCT/MRI guided neuronavigation for transsphenoidal surgery is time-effective, cost-efficient, safe and technically beneficial.

THURSDAY, NOVEMBER 4

11:10 – 11:22 AM **CLOSURE OF INTRA-OPERATIVE CSF LEAKS IN TRANS-SPHENOIDAL SURGERY**

Martin H. Weiss, MD, Charles Liu, MD, PhD, & William T. Couldwell, MD, PhD

Post operative CSF fistulas are the most common serious complications of trans-sphenoidal surgery whether performed by the sublabial, endonasal or endoscopic routes. Efforts to obliterate intra-operative CSF leaks include the use of autologous fat grafts, suture repair of the dural opening, artificial membranes, various sealants, autologous cartilage and both biodegradable and permanent mechanical devices. Approximately 8 years ago, the authors adopted a technique employing autologous rectus abdominis fascia applied directly in apposition to the open dural edges, anchored by a single pledget of surgicel and then buttressed by autologous harvested abdominal fat filling the sphenoid.

A review of our experience of the past 5 years involving 1021 cases of conventional trans-sphenoidal surgery in addition to extended trans-sphenoidal surgery involving the clivus and planum sphenoidale reveals 3 cases of post operative CSF leaks requiring surgical repair. None of the cases had intra-operative or post-operative lumbar drainage. In cases in which the arachnoid is partially preserved, the fascial graft is applied intradurally between the arachnoid and dural opening. In cases in which residual arachnoid is virtually absent (particularly in cases of penetrating clival chordomas and anterior extended trans-sphenoidal procedures for craniopharyngiomas and dermoids) the fascial graft straddles the dural opening once the surgeon confirms that bone has been removed circumferentially sufficiently to have dural edges available throughout the operative field. In the latter situations, the extradural fascia is tucked under the edges of surrounding bone, reinforced by a single pledget of surgical also tucked under the bone and then buttressed with the fat as described above.

The authors believe that this represents a simple and cost effective technique that is most effective.

THURSDAY, NOVEMBER 4

11:24 – 11:36 AM **EFFECTIVE TREATMENT OF DISSEMINATED MEDULLOBLASTOMA WITH MODIFIED MEASLES VIRUS IN A MURINE MODEL**

Corey Raffel, M.D., Ph. D., Adam Studebaker, Ph.D., Brian Hutzen, B.A.,

INTRODUCTION: Dissemination of medulloblastoma in the cerebrospinal fluid is present in 20% of patients at presentation and 40% of patients at recurrence. CSF dissemination carries a particularly grave prognosis with less than 20% of patients surviving 5 years. Effective new treatments for dissemination are needed. We present here our results in treating a xenograft model of disseminated medulloblastoma with modified measles virus. We demonstrate statistically increased survival in animals treated with the virus.

METHODOLOGY: Nude mice were injected with 10E6 D283MED medulloblastoma cells stereotaxically into the lateral ventricle. The cells were transfected with a luciferase expression cassette to allow monitoring of tumor by imaging over time. Three days later, modified measles virus (2x10E5 pfu) was injected in to the lateral ventricle every other day for 5 treatments. Animals were followed with serial biolumuminescent imaging. Survival was determined for treated and control animals. Autopsies were performed on animals showing signs of progressive disease.

RESULTS: Tumor in the spinal canal was detected in all animals by imaging. Untreated mice died in an average of 37 days. Survival in the treated animals averaged 82 days, p=0.0004. Tumors either stabilized or shrank in treated animals, as visualized by bioluminescent imaging, but tumors eventually progressed in all but one treated animal. This one animal, of 8 treated animals, appears to have been cured of disseminated disease. Autopsy revealed extensive intraventricular, intracranial subarachnoid, and spinal subacrachnoid disease in untreated animals. Treated animals that died of progressive disease had similar findings.

CONCLUSION: We have demonstrated effective treatment of disseminated medulloblastoma with measles virus in a new murine model of CSF dissemination. Ongoing experiments in our lab to determine optimal dosing of virus in preparation for a phase 1 trial are underway.

THURSDAY, NOVEMBER 4

11:38 - 11:50 AM **MEDULLOBLASTOMA COMPRISES FOUR DISTINCT DISEASES**

Paul A. Northcott, Andrey Korshunov, Hendrik Witt, Thomas Hielscher, Charles Eberhart, Stephen Mack, Eric Bouffet, Steven C. Clifford, Cynthia Hawkins, Pim French, James T. Rutka, Stefan Pfister, ***Michael D. Taylor***

Prior attempts to subgroup medulloblastoma using genomics have identified 4-6 distinct subtypes, including distinct groups driven by Wnt and Shh signaling. We analyzed a cohort of 103 primary medulloblastomas using Affymetrix Exon expression arrays and Affymetrix 500k SNP arrays to determine how many subgroups of the disease exist, how they differ, and the extent of overlap between subgroups.

Both unsupervised hierarchical clustering and principal component analysis of expression data on 103 medulloblastomas reveals very high confidence for existence of four medulloblastoma subgroups: WNT, SHH, Group C, and Group D. Further bioinformatic analyses using Prediction Analysis of Microarrays (PAM), Nonnegative Matrix factorization (NMF), and Subclass Mapping (SubMap) all support the existence of four subgroups. The SHH group tumors occur in infants and adults, Group C tumors occur only in children, and Wnt and Group D tumors are found across all age groups. We identified 'signature' genes over-expressed in each subgroup for which there are high quality commercial antibodies. We stained two separate medulloblastoma tissue microarrays containing 294 non-overlapping tumors for DKK1 (WNT), SFRP1 (SHH), NPR3 (Group C), and KCNA1 (Group D) demonstrating that 288/294 (98%) of tumors stained for only a single marker. A multivariate analysis of age, extent of resection, histology, M stage and subgroup revealed that only LCA histology and Group C were prognostic.

Our data highly support the existence of four independent types of medulloblastoma that differ in their demographics, rate of metastases, transcription, genetic events, and clinical outcome. Our novel '4 antibody' technique is capable of determining medulloblastoma subgroup through immunohistochemistry on formalin fixed, paraffin embedded material suggesting that it will be broadly generalizable across the globe.

THURSDAY, NOVEMBER 4

11:52 – 12:04 PM **WHOLE BRAIN RADIOTHERAPY AND STEREOTACTIC RADIOSURGERY IN THE TREATMENT OF BRAIN METASTASIS: THE RANDOMIZED CLINICAL TRIALS**

Phillip A. Tibbs, MD., Roy L. Patchell, MD.

INTRODUCTION: Class I evidence from our studies established craniotomy followed by whole brain radiotherapy (WBRT) as the standard of care for the treatment of operable cerebral metastases. Stereotactic radiosurgery (SRS) is now frequently used, initially without level I evidence, as primary therapy for solitary or multiple brain metastases. In the past decade, further experience with these modalities has helped to clarify the specific, often complementary, roles of WBRT and SRS.

METHODOLOGY: The authors analyzed class I data from their own and other randomized clinical trials (RCTs) to identify the relative roles of WBRT and SRS in the treatment of cerebral metastases. A treatment algorithm was developed.

RESULTS: The combination of craniotomy and WBRT yields recurrence rates of less than 10%, a difficult standard to surpass. For a single metastasis <3cm in diameter or in surgically inaccessible tumors, the combination of SRS and WBRT gives comparable results although there is a need for salvage surgery due to radiation necrosis in some cases. RCT's have consistently demonstrated a statistically significant pattern of increased risk of early tumor recurrence for SRS alone versus craniotomy plus WBRT. Neurocognitive testing reveals a definite transient decline in function 3-4 months after WBRT not seen with SRS; however, recurrent or uncontrolled tumor growth is a greater threat to neurological function than WBRT-related neurocognitive effects.

CONCLUSIONS:

1. Currently available class I evidence indicates that patients with single cerebral metastases should receive craniotomy or SRS with WBRT adjuvant therapy in most cases.
2. Patients with multiple or inoperable metastases should receive WBRT, supplemented by SRS in certain cases.
3. WBRT produces a significant reduction in the rate of tumor recurrence versus SRS alone.
4. Where SRS alone is elected as primary therapy for brain metastases, regular MRI surveillance is mandatory for early detection of recurrence to allow salvage therapy.
5. The neurotoxicity of WBRT is transient in most cases and pales beside the deleterious effects of recurrent disease.
6. Salvage therapy is necessary more often following SRS alone than after WBRT.
7. WBRT and SRS should not be considered either/or therapies since combined use of the modalities often offers the best outcome for patients.

A treatment algorithm will be presented.

THURSDAY, NOVEMBER 4

12:06 – 12:18 PM **FEASIBILITY OF LASER INTERSITITIAL THERMAL THERAPY WITH REAL-TIME MR THERMOGRAPHY FOR CRITICALLY PLACED BRAIN TUMORS**

Michael Schulder, MD, Peter Kingsley, PhD, Ashok Gowda, PhD

INTRODUCTION: Laser interstitial thermal therapy (LITT) is a hyperthermic technique that has been described as a treatment for patients with cerebral metastatic tumors. We explored the application of this technique for patients with various tumors in critical locations and report our preliminary results.

METHODS: Three patients are included in this report. Two had tumors in the brainstem (1 metastatic and the other ependymoma), and the third had a prolactinoma compressing the optic chiasm. Each patient had progressive neurological symptoms from tumor growth, despite maximal attempts at prior surgery, radiation therapy or radiosurgery, and chemotherapy.
Laser fibers (Visualase, Houston, TX) were stereotactically placed in the operating room under guidance with a 0.15 Tesla (T) intraoperative MRI. An MRI-compatible guide tube was imaged during surgery to confirm accurate targeting. The laser fibers were then inserted and the patients transferred to a 1.5T MRI for LITT. Imaging was repeated to confirm laser placement within the tumors.
LITT was then begun and controlled with real-time MRI thermography (MRT). MRT was accomplished using a 2D RF-spoiled gradient recalled echo sequence, which required approximately 5 seconds for a single acquisition and which was run repeatedly during the treatment.
During each treatment, an Arrehenius-based model, which accounts for the time and temperature dependency of protein denaturation, was used to estimate irreversible cell death. Laser therapy was terminated when ablation zones reached desired sizes or automatically when critical structures exceeded preset safety limit temperatures. Contrast-enhanced T1 weighted MRI was then done to confirm the volume of treatment. After conclusion of LITT, patients were returned to the operating room for laser fiber removal and emergence from anesthesia.

RESULTS: LITT was successfully concluded in 2 patients (with pontine metastasis and prolactinoma). Laser depth was adjusted to improve geometric coverage of the lesions, and 1-3 treatment doses were applied at each depth. Laser on times ranged from 30 sec to 150 sec, and temperatures at the ablation margins were between 55° C and 60°C. Laser energy ranged from 6 W to 10 W. In these 2 patients the final MRI confirmed loss of contrast in the target volume. The patient with pontine metastasis had a temporary slight increase in ataxia, which recovered within 2 weeks. The third patient had had 2 prior craniectomies and irradiation for an ependymoma of the medulla. Accurate laser insertion was hindered by the steep angle and missing suboccipital bone. Laser test doses at 6 W did not yield satisfactory zones of ablation, and full therapeutic doses therefore were not applied.

CONCLUSIONS: LITT can be used for ablation of tumors in surgically critical locations, in patients who have exhausted other options. Technical improvements will allow improved visualization of laser fibers with iMRI and smaller openings for laser insertion.

THURSDAY, NOVEMBER 4

12:20 – 12:32 PM **COMPARATIVE EFFECTIVENESS RESEARCH IN SPINAL SURGERY**

Daniel K. Resnick, MD MS

BACKGROUND: As evidenced by recent legislation, comparative effectiveness research is going to play a major role in the determination of what therapies are available for patients with disease processes where more than one treatment option exists. The goal of such research is to improve the value of medical care. Many spinal disorders are treated by a variety of practitioners using a variety of treatment modalities making spinal disorders a prime target for such research, and the use of surgery to treat spinal disorders has been under intense scrutiny for several years with a perception that such treatment is overused, expensive, and dangerous.

PURPOSE: In order to accurately describe the value of any treatment for lumbar spine disorders it is necessary to accurately describe the population of patients treated, employ validated outcomes measures that reflect clinically important variables, and to perform appropriate risk stratification. Ideally, multiple treatments could be compared if similar patient populations were treated and similar outcomes measures obtained.

METHODS: Multiple medical societies, the Agency for Health Care Research and Quality (AHRQ), the Center for Medicare Services (CMS), private third party payers, employers, patient advocates, and quality improvement organizations (NCQA, NQF) participated in a public forum to develop a rationale framework for outcomes research in lumbar spine disorders.

RESULTS: The multi-stakeholder workgroup has developed a template for outcomes research in order to facilitate communication between multiple medical societies, employers, third party payers, quality improvement organizations, and the government. The development process and important features of the template will be presented.

FRIDAY, NOVEMBER 5

SCIENTIFIC PROGRAM

American Academy of Neurological Surgery Annual Meeting The Inn at Spanish Bay, Pebble Beach, CA, November 3-6, 2010

PAPER PRESENTATIONS

7:30 – 7:42 AM **EVACUATION OF SPONTANEOUS INTRACEREBRAL HEMORRHAGE USING SONOTHROMBOLYSIS**

David W Newell MD, Mohsin Shah MD, Daniel F Hanley MD

Catheter based hemorrhage evacuation is a novel surgical approach for the treatment of brain hemorrhage. The objective of this study was to evaluate the safety and efficacy of ultrasound in combination with recombinant tissue plasminogen activator (rt-PA) delivered through a microcatheter directly into spontaneous intraventricular (IVH) or intracerebral (ICH) hemorrhage in humans.

METHODS: A total of 33 patients presenting to Swedish Medical Center, in Seattle Washington with ICH and IVH were screened between 11/21/2008 and 7/13/2009 for entry into the study. Entry criteria included the spontaneous onset of intracranial hemorrhage ≥ 25cc and or intraventricular hemorrhage producing ventricular obstruction. Nine patients (ages 38-83, average = 63, 6 male, 3 female) who met entry criteria were consented and entered into the trial. A ventricular drainage catheter and an ultrasound microcatheter were stereotactically delivered together, directly into the IVH or ICH. Recombinant tissue plasminogen activator (rt-PA) and 24 hours of continuous ultrasound were delivered to the clot. Gravity drainage was performed. In patients with intraventricular hemorrhages, a total of 3 mg of rt-PA was injected, and in patients with intraparenchymal hemorrhages a total of 0.9 mg rt-PA was injected, in three doses over 24 hours.

RESULTS: All patients had significant volume reductions of the treated hemorrhage. The mean percentage volume reduction after 24 hours of treatment, compared to the pre-treatment stability scans, as determined by CT were 59 % ± 5 (sem) for ICH, and 45.1% ±13 (sem) for IVH (1 ICH patient was excluded from analysis due to catheter breakage). There were no intracranial infections and there were no significant episodes of re-bleeding by clinical or CT assessment. There was 1 death by 30 days after admission. Clinical improvements as determined by a decrease in the National Institutes of Health Stroke Score (NIHSS) were demonstrated at 30 days in 7/9 patients. The rate of hemorrhage lysis was compared between 8 patients who completed treatment, to cohorts of patients treated using identical doses of tPA and catheter drainage without ultrasound for IVH and ICH (courtesy of MISTIE and CLEAR studies). Compared to data we observed a faster rate of lysis during treatment for IVH and for ICH in the patients treated with sonolysis + tPA versus tPA alone.

CONCLUSIONS: Lysis and drainage of spontaneous ICH and IVH with reduction of mass effect can be accomplished rapidly and safely by sonothrombolysis using stereotactically delivered drainage and ultrasound catheters via a burr hole. A larger clinical trial with catheters specifically designed for brain blood clot removal is warranted

FRIDAY, NOVEMBER 5

7:44 – 7:56 PM **ADENOSINE FOR TEMPORARY FLOW ARREST DURING INTRACRANIAL ANEURYSM SURGERY: A SINGLE CENTER RETROSPECTIVE REVIEW**

Bernard R. Bendok, MD, FACS; Rudy J. Rahme, MD; Dhanesh K. Gupta, MD; Christopher S. Eddleman, MD, PhD; Joseph G. Adel, MD; Arun K. Sherma, MD; Daniel L. Surdell, MD; John F. Bebawy, MD; Antoun Koht, MD; H. Hunt Batjer, MD, FACS

INTRODUCTION: Temporary occlusion with clips remains an integral technique for intracranial aneurysm clipping. Clip application for temporary occlusion is not always practical or feasible. Adenosine is an alternative which provides brief periods of flow arrest which can be used to advantage in aneurysm surgery but little has been published on its utility for this indication.

METHOD: We retrospectively reviewed our clinical database between May, 2007, and December, 2009. All patients who underwent microsurgical clipping of intracranial aneurysms under adenosine-induced asystole were included. Aneurysm characteristics, reasons for adenosine utilization, post operative angiographic and clinical outcome, cardiac complications and long term neurological follow-up using the modified Rankin Scale were noted.

RESULTS: Adenosine was used for 40 aneurysms (10 ruptured/ 30 unruptured). The most common indications for adenosine were “aneurysm softening” in 17 cases and “paraclinoid location” in 14 cases, followed by “broad neck” in 12 cases and intraoperative rupture in 6 cases. Troponin levels were elevated postoperatively in 2 patients. Echocardiography did not show acute changes in either. Transient cardiac arrhythmias were noted in 5 patients. 27 patients were available for follow-up. Mean follow-up was 3.9 months. The mRS score was 0 for 23 patients at time of last follow-up. 2 patients had a mRS score of 1, and scores of 2 and 3 were found in 1 patient each.

CONCLUSION: Adenosine appears to be a safe and effective method to achieve transient deep hypotension and asystole during microsurgical clipping of intracranial aneurysms. Further study of adenosine for this indication is warranted.

FRIDAY, NOVEMBER 5

7:58 – 8:10 **THE SEARCH FOR THE SOURCE OF BRAIN ADENOSINE**

Tobias Kulik, MD, PhD, ***H. Richard Winn, MD***

INTRODUCTION: Like the search for the origin of the Nile, we have been on a multi-decade hunt for the source of Adenosine (Ado) in the brain and believe we have identified astrocytes as the primary producers of this regulator of CBF and modulator of neuronal activity.

METHODS: Astrocytes were uniquely grown on microcarrier beads and then subject to step (measured in min) and abrupt (seconds) oxygen deprivation as well as reoxygenation. dO2 was monitored using an online fluorometric technique.

RESULTS: Fig 1 illustrates the changes in Ado with step changes in O2. Note the two phase with the most steep relationship occurring when O2 is less than 30 mmHg, precisely the level of oxygen found in brain tissue during normoxia (PaO2=100 mmHg). In addition, the levels of Ado are comparable to those found in vivo in CSF and interstitial fluid. With abrupt oxygen (Fig 2) deprivation and reoxygenation, 1000 fold changes in Ado concentration occur within 30 s. These changes are equal to those observed in vivo with hypoxia and are of sufficient magnitude to affect CBF and neuronal activity. Moreover, the time scale is identical to the changes in CBF with oxygen deprivation and reoxygenation

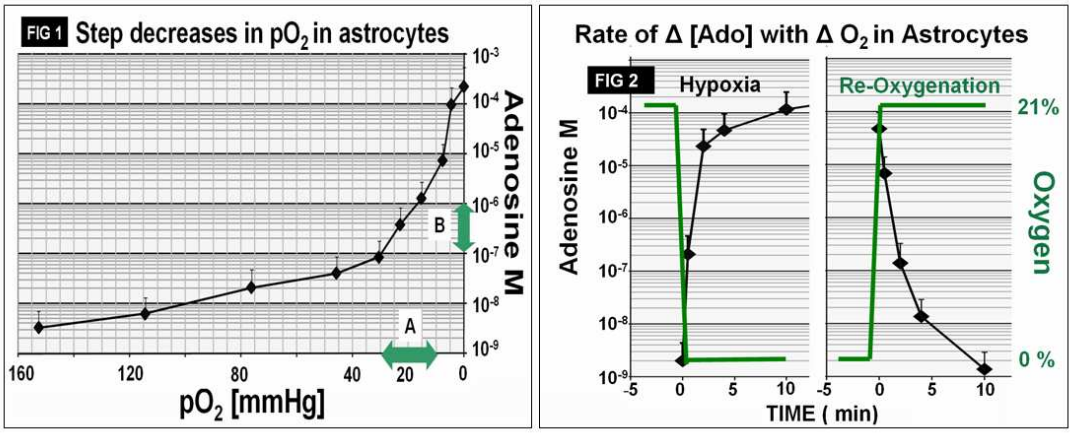

Fig 1: **Step changes in O_2:⟺** comparable resting O_2 (A) and Ado (B) *in vivo*.
Fig 2 Abrupt Oxygen Deprivation and Re-oxygenation: X1000 changes with 30s.

SUMMARY: The concentrations of Ado in astrocytes are similar to those found in brain in vivo and change with sufficient rapidity to account for the observed alterations in Ado concentrations, CBF, and neuronal activity in vivo during hypoxia.
CONCLUSION: We believe that, like Henry Morton Stanley, we have found the "source" for adenosine in the brain: astrocytes.
(Disclaimer: if chosen, the presenter will not wear a pith helmet.)

FRIDAY, NOVEMBER 5

8:12 – 8:24 AM **DUAL REGULATION OF POST ISCHEMIC NEUROGENESIS BY TGF-ß1**

Robert J. Dempsey, MD, Yiping Yan, PhD, Haviryaji Kalluri, PhD, Raghu Vemuganti, PhD

INTRODUCTION: Harnessing neurogenesis may be a future therapy for brain repair after stroke. Normally after cerebral ischemia, neurogenesis is increased by enhancing the proliferation and differentiation of neural progenitor cells already present in the normal brain.

HYPOTHESIS: We hypothesize that the growth factors that regulate such neurogenesis may be used to therapeutically alter such neurogenesis to enhance clinical recovery. Our studies have shown that focal cerebral ischemia upregulates the expression of the transforming growth factor TGF-ß1 after three days of brain reperfusion. In turn, this growth factor is co-localized with the macrophages and inflammatory cells which show ED1 +ve markers in the ischemic area. It is important to understand that the processes which increase proliferation and differentiation in the brain, must themselves be regulated to avoid the unchecked neoplastic growth of tumors. It is possible that the agents which increase differentiation of the neurons must also decrease or regulate what would otherwise be unchecked proliferation of cells. We know from other studies that TGF-ß1 is involved in the differentiation of neurons. In this study, we analyze the hypothesis that TGF-ß1 also regulates the process by decreasing proliferation of the progenitor cells (NPC) in vitro.

METHODOLOGY: We incubated NPC's with TGF-ß1 (0.1, 1, 10 ng/ml). Neural progenitor cells were isolated from subventricular zone of adult rats and cultured in neurobasal medium containing B27 and FGF2 (20 ng/ml) for 4 days in the presence of varying concentrations of TGF-β1 (0.1, 1, 10ng/ml). Cell division was assessed by cell proliferation assay & BrdU incorporation.

RESULTS: Incubation of neural progenitor cells with TGF-β1 (1, 10 ng/ml) reduced the formation of neurospheres, which was consistent with a decline in the metabolic activity of the cells. This result was not seen at lower dose of TGF-β1 (0.1ng/ml). Metabolic activity showed a 40-50 % ($P<0.001$) decrease in the proliferation of cells at the higher concentrations of TGF-β1 (1, 10 ng/ml). Likewise, BrdU labeling also demonstrated a 50% decrease ($P < 0.05$) in the proliferation of cells as compared to control in the two higher dose groups.

CONCLUSION: These results suggest that TGF-ß1 may play dual essential roles in regulating post ischemic neurogenesis. The implications are that if these agents are used therapeutically, they have to be carefully regulated to avoid unchecked proliferation of cells. By promoting differentiation and turning down proliferation, TGF-ß1 may be a natural way to avoid neoplasia. This dual role makes TGF-ß1 one of several important growth factors which will need to be modified sequentially to achieve the result of enhanced clinical improvement after stroke through neurogenesis.

FRIDAY, NOVEMBER 5

8:26 – 8:38 AM **SURGERY FOR THE CAVERNOUS ANGIOMA IN THE BRAINSTEM**

Kazuhiro Hongo, Tetsuya Goto, Hisashi Muraoka, Kunihiko Kodama, Yukinari Kakizawa, Keiichi Sakai

INTRODUCTION: Direct surgery for cavernous angiomas in the brainstem is indicated when symptomatic or causing repeated hemorrhage. In this report, our surgical strategy , use of the intraoperative brainstem monitoring/mapping and results will be reported.

METHODS: Between April 2000 and May 2010, 15 patients (4 men and 11 women, age ranged between 19 and 64 years, average age of 44.4 years, follow-up period of 4.7 years) were surgically treated. Lesions were midbrain in 6 patients, pons in 8, medulla oblongata in 1, cerebellar peduncle in 1 (in one patient there were two lesions in the midbrain and pons). For the fourth-ventricular floor approach, brainstem mapping/monitoring was utilized especially for preserving facial nerve function.

RESULTS: For the 15 patients, 17 surgeries were conducted: in one patient intentional two-stage surgery was performed. As surgical approaches, 2 occipital transtentorial, 2 subtemporal, one supracerebellar intratentorial approaches were taken for the midbrain lesion; trans-fourth ventricular floor approach was taken for all the 10 patients with the lesions in the midbrain, pons and medulla oblongata; lateral suboccipital approach for the cerebellar peduncle lesion, respectively. For one patient with double lesions in the pons, total removal was achieved in a two-staged surgery with the trans-fourth ventricular floor approach. Total removal was achieved in 13 patients, gross total removal in 2. There was no mortality. Postoperative Karnofsky performance state was equal or more than 90% in 13 patients and 80%; in two patients cerebellar ataxia worsened postoperatively, the remaining had better neurological status than that before surgery. There were no recurrences.

CONCLUSIONS: With selecting a suitable surgical approach with brainstem monitoring/mapping, the lesions were safely resected with minimum neurological deficits.

FRIDAY, NOVEMBER 5

8:40 – 8:52 AM **SUPRATENTORIAL CAVERNOUS MALFORMATIONS IN ELOQUENT AND DEEP LOCATIONS: SURGICAL APPROACHES AND OUTCOMES**

Michael T. Lawton, MD

OBJECTIVE: Surgical resection of cavernous malformations located in functionally eloquent areas of the supratentorial compartment is controversial. Hemorrhage from untreated lesions can result in devastating neurological injury, but surgery has potentially serious risks. We hypothesized that an organized system of approaches can guide operative planning and lead to acceptable neurological outcomes in surgical patients.

METHODS: We reviewed the presentation, surgery, and outcomes of 79 consecutive patients that underwent microresection of supratentorial cavernous malformations in eloquent and deep brain regions (basal ganglia (n=27), sensorimotor cortex (n=23), language cortex (n=3), thalamus (n=6,), visual cortex (n=10), and corpus callosum (n=10)). A total of 13 different microsurgical approaches were organized into 4 groups: superficial, lateral trans-Sylvian, medial interhemispheric, and posterior approaches.

RESULTS: The majority of patients (93.7%) were symptomatic, Hemorrhage with resulting focal neurological deficit was the most common presentation in 53 patients (67%). Complete resection, as determined by postoperative MRI, was achieved in 76 patients (96.2%). Overall, the functional neurological status of patients improved following microsurgical dissection at hospital discharge and at follow-up. At 6 months, 64 patients (81.0%) were improved relative to their preoperative condition and 14 patients (17.7%) were unchanged. Good outcomes (modified Rankin Score ≤ 2, living independently) were achieved in 77 patients (97.4%). Multivariate analysis of demographic and surgical factors revealed that pre-operative functional status was the only predictor of post-operative modified Rankin Scale score (OR=4.6, P=0.001). Six patients (7.6%) had transient worsening of neurological exam after surgery and one patient (1.3%) was permanently worse. There was no surgical mortality.

CONCLUSIONS: We present a system of 13 microsurgical approaches to 6 location targets with 4 general trajectories to facilitate safe access to supratentorial cavernous malformations in eloquent brain regions. Favorable neurological outcomes following microsurgical resection justify an aggressive surgical attitude towards these lesions.

FRIDAY, NOVEMBER 5

8:54 – 9:06 AM **THROMBOSPONDIN-1 MODULATES THE ANGIOGENIC PHENOTYPE OF HUMAN CEREBRAL ARTERIOVENOUS MALFORMATION ENDOTHELIAL CELLS**

Christopher J Stapleton, B.S.; Don L Armstrong, Ph.D.; Raphael Zidovetzki, Ph.D.; ***Charles Y Liu, M.D., Ph.D.***; Steven L Giannotta, M.D.; Florence M Hofman, Ph.D.

BACKGROUND: The management of cerebral AVM is challenging, and invasive therapies place vital intracranial structures at risk for injury. The development of noninvasive, pharmacologic approaches relies upon identifying factors that mediate key angiogenic processes. Prior studies indicate that endothelial cells derived from cerebral AVM (AVM-EC) are distinct from control brain endothelial cells (BEC) with regard to important angiogenic characteristics.

OBJECTIVE: The goal of this study is to determine whether thrombospondin-1 (TSP-1), a potent angiostatic factor, regulates critical angiogenic features of AVM-EC and to identify factors that modulate TSP-1 production in AVM-EC.

METHODS: Endothelial cell proliferation, migration, and tubule formation were evaluated with BrdU incorporation, Boyden chamber, and Matrigel studies, respectively. TSP-1 and inhibitor of DNA binding/differentiation 1 (Id1) mRNA levels were quantified with microarray and quantitative RT-PCR analyses. TSP-1 protein expression was measured using Western blotting, immunohistochemical, and ELISA techniques. The mechanistic link between Id1 and TSP-1 was established through siRNAmediated knockdown of Id1 in AVM-EC followed by Western blot and ELISA experiments assessing TSP-1 production.

RESULTS: (1) AVM-EC proliferate faster, migrate more quickly, and form disorganized tubules as compared to BEC. (2) TSP-1 is significantly downregulated in AVM-EC. (3) Addition of TSP-1 to AVMEC cultures normalizes the rate of proliferation and migration and the efficiency of tubule formation, whereas BEC are unaffected. (4) Id1 negatively regulates TSP-1 expression in AVM-EC.

CONCLUSIONS: These data highlight a novel role for TSP-1 in the pathobiology of AVM angiogenesis and provide a context for its use in the clinical management of brain AVM.

FRIDAY, NOVEMBER 5

9:08 – 9:20 AM **INTRACRANIAL ANEURYSM RISK GENES IDENTIFIED THROUGH WHOLE GENOME ASSOCIATION STUDY**

Murat Gunel, MD** for the Genetics of Intracranial Aneurysm Trial group * Yale University

INTRODUCTION: Saccular intracranial aneurysms (IAs) are balloon-like dilations of the intracranial arterial wall; their hemorrhage commonly results in severe neurologic impairment and death. We recently reported a genome-wide association study (GWAS) including 2,075 cases and 6,952 controls that identified 3 significant loci with odds ratios (ORs) ranging from 1.24 to 1.36 (Bilguvar et al., Nat Genet 40: 1472 – 1477, 2008). This analysis had limited power and explained only a small fraction of the risk of IA.

METHODOLOGY: In order to increase the power to detect new IA loci, we conducted a second genome-wide association study with discovery and replication cohorts from Europe and Japan comprising 5,891 cases and 14,181 controls with ~832,000 genotyped and imputed SNPs across discovery cohorts.

RESULTS: We identified three new loci showing strong evidence for association with IA in the combined data set, including intervals on 18q11.2 (P=1.1×10^{-12}), on 13q13.1 (P=2.5×10^{-9}) and on 10q24.32 (P=1.2×10^{-9}) (Yasuno et al., Nat Genet, 42(5):420-5, 2010). We also confirmed prior associations near *SOX17* (8q11.23-q12.1; P=1.3×10^{-12}) and *CDKN2A/B* (9p21.3; P=1.5×10^{-22}). Further follow-up of 14 regions that had posterior probability of IA association between 10 and 50% in the discovery cohort, identified additional novel genes.

CONCLUSIONS: Several of the putative IA risk genes play a role in cell-cycle progression, potentially affecting proliferation and senescence of progenitor cell populations that are responsible for vascular formation and repair. These findings have implications for pre-clinical diagnosis, biology and therapy of intracranial aneurysms.

FRIDAY, NOVEMBER 5

9:22 – 9:34 AM **HEMODYNAMIC MORPHOLOGIC DISCRIMINANTS FOR INTRACRANIAL ANEURYSM RUPTURE**

***Adnan H. Siddiqui MD PhD*, L. Nelson [Nick] Hopkins MD**

INTRODUCTION: To identify significant morphologic and hemodynamic parameters that discriminate intracranial aneurysm (IA) rupture status using 3D angiography and computational fluid dynamics (CFD).

METHODS: 119 IAs (38 ruptured, 81 unruptured) were analyzed from 3D angiographic images and CFD. Six morphologic and seven hemodynamic parameters were evaluated for significance with respect to rupture. Receiver-operating characteristic (ROC) analysis identified area under the curve (AUC) and optimal thresholds separating ruptured from unruptured aneurysms for each parameter. Significant parameters were examined by multivariate logistic regression analysis in 3 predictive models—morphology only, hemodynamics only, and combined—to identify independent discriminants, and the AUC-ROC of the predicted probability of rupture status was compared among these models.

RESULTS: Morphologic parameters (*Size Ratio [SR], Undulation Index, Ellipticity Index,* and *Nonsphericity Index*) and hemodynamic parameters (*Average Wall Shear Stress [WSS], Maximum intra-aneurysmal WSS, Low WSS Area, Average Oscillatory Shear Index [OSI], Number of Vortices,* and *Relative Resident Time*) achieved statistical significance ($p<0.01$). Multivariate logistic regression analysis demonstrated *SR* to be the only independently significant factor in the morphology model (AUC=0.83, 95% confidence interval [CI] 0.75-0.91), whereas *WSS* and *OSI* were the only independently significant variables in the hemodynamics model (AUC=0.85, 95% CI 0.78-0.93). The combined model retained all three variables, *SR, WSS,* and *OSI* (AUC=0.89, 95% CI 0.82-0.96).

CONCLUSION: All three models—morphological (based on *SR*), hemodynamic (based on *WSS* and *OSI*), and combined—discriminate IA rupture status with high AUC values. Hemodynamics is as important as morphology in discriminating aneurysm rupture status.

FRIDAY, NOVEMBER 6

9:36 – 9:48 AM **EFFECT OF DRUG TREATMENT ON VASOSPASM, DELAYED CEREBRAL ISCHEMIA AND FUNCTIONAL OUTCOME AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE - A SYSTEMATIC REVIEW AND META-ANALYSIS**

R. Loch Macdonald, M.D., Ph.D., F.R.C.S.(C), F.A.C.S., Nima Etminan, M.D., Mervyn D.I. Vergouwen, M.D., Ph.D.

INTRODUCTION: Delayed cerebral ischemia (DCI) is a common cause of poor outcome in patients with subarachnoid hemorrhage (SAH). Since it is often assumed that DCI is caused by vasospasm, clinical trials in the last few decades focused on the prevention of vasospasm with the aim to improve functional outcome. However, it could be that the role of vasospasm in the pathogenesis of DCI and functional outcome is smaller than previously assumed. To further investigate the relationship between vasospasm and functional outcome, we decided to pool all randomized placebo-controlled trials that investigated pharmaceutical interventions as a preventive strategy in SAH patients.

METHODS: For this systematic review, the Cochrane Collaboration format was used. We included all randomized, double-blind, placebo-controlled trials that studied the efficacy of pharmaceutical preventive strategies in SAH patients, and had vasospasm, DCI and functional outcome as outcome events. Vasospasm was defined by catheter angiography or transcranial Doppler criteria. DCI was defined as clinical deterioration from cerebral ischemia after exclusion of other causes. Functional outcome was assessed on the dichotomous Glasgow outcome or modified Rankin scales. Effect sizes were expressed in (pooled) risk ratio estimates with corresponding 95% confidence intervals (CI).

RESULTS: In 14 studies, 4235 patients were included. Despite a reduction of vasospasm (RR 0.80 (95% CI 0.70-0.92)) and DCI (RR 0.76 (95% CI 0.67-0.87), no statistically significant effect on poor outcome was observed (RR 0.94 (95% CI 0.84-1.04)). No high risk of bias was observed in any of the studies.

CONCLUSION: Our results do not lend support to the assumption that a reduction of vasospasm or DCI results in better functional outcomes in patients with aneurysmal SAH

FRIDAY, NOVEMBER 5

9:50 - 10:02 AM **BASILAR OCCLUSION FOR COMPLEX BASILAR ARTERY ANEURYSM: AN OLD TECHNIQUE FOR NEW AGE NEUROSURGERY**

Christopher P Kellner MD, **Raqeeb Haque MD**, **Philip M Meyers MD**, **Sean D Lavine MD**, **E Sander Connolly, Jr. MD FACS**, ***Robert A Solomon, MD FACS***

INTRODUCTION: Surgical clipping of large and complex aneurysms of the basilar artery apex has always been accompanied by high risk and relatively poor results. Since the introduction of the Guglielmi detachable coil in 1991, the focus on treating basilar aneurysms shifted dramatically in favor of endovascular techniques. However, outcomes with endovascular techniques including coiling and stent-coiling have also been less than optimal for large and complex basilar aneurysms. Although surgical occlusion of the proximal basilar artery has been recognized for decades as a viable treatment option, deep hypothermic circulatory arrest surgery and more recently complex stent-coil procedures have dominated the discussion. We are therefore reporting our current 22 year experience with surgical basilar occlusion for complex basilar aneurysms with long-term outcomes.

METHODS:: Fifteen patients underwent surgical basilar artery occlusion at Columbia University Medical Center for complex basilar apex aneurysms between 1987 and 2009. With IRB approval, the clinical records of each patient were retrospectively reviewed for details of presentation, hospital course, operative intervention, and outcome.

RESULTS: Post-operatively, all patient encounters were recorded at discharge, one month, one year, and long-term outcome. Twelve of 15 patients (80%) had no new post-operative neurological deficits. Three patients presenting with severe neurologic impairment (mRS >3) made excellent recoveries (mRS 1-2) at long-term follow-up. One patient died, one patient suffered a stroke during the post-operative angiogram resulting in hemiparesis, and one patient suffered inter-nuclear ophthalmoplegia that resolved by 1 month follow-up. Long-term follow-up occurred at an average of 3 years (SD±4.5), ranging from 4 months for a recently treated patient to 18 years. Average mRS at long-term follow-up was 1 (SD±1.5). No patients experienced post-operative hemorrhage, rebleed, or delayed neurological deterioration.

CONCLUSION: Surgical occlusion of the basilar artery is an effective treatment option offering a high rate of angiographic cure in a single procedure for patients with complex basilar artery aneurysms. The ability to surgically perform point occlusion of the basilar artery without impairment of brainstem perforators while maintaining collateral blood flow to the posterior circulation branch vessels may provide an advantage over endovascular treatments.

FRIDAY, NOVEMBER 5

10:04 – 10:16 AM **MANAGEMENT OF CERVICAL AVMS**

Robert Spetzler MD, Cameron McDougall MD, and Felipe Albuquerque MD.

Cervical AVMs are uncommon vascular lesions that may present with hemorrhage, vascular steal or compression. We are presenting nine cases where single or combined treatment let to the cure of the AVM. The lessons learned have changed our approach to these daunting entities. The current management strategy includes selective angiographic exploration, functional testing followed with appropriate embolization. Surgical resection consists of removing the nidus that is extrapial. Much to our pleasant surprise these lesions can be resected while leaving the intramedullary component intact. The interruption of the channels with the removal of the extrapial component can eliminate flow through the intramedullary residual. This allows the subpial normal vasculature to stay intact while interrupting the abnormal channels that go in and out of the spinal cord. ICG angiography is most helpful in locating feeding vessels and establishing absence of shunting. By recognizing that the intra/extra medullary vascular channels can be interrupted and controlled with bipolar coagulation without uncontrolled bleeding or swelling we have been able to angiographically obliterate these AVM's while maintaining neurological function.

FRIDAY, NOVEMBER 5

10:38 – 10:50 AM **Academy Award Winner**

AUGMENTING ADULT HIPPOCAMPAL NEUROGENESIS USING TARGETED BRAIN STIMULATION: IMPLICATIONS FOR MEMORY NETWORKS

Scellig S.D. Stone MD, Kirill Zaslavsky BSc, Cátia M. Teixeira PhD, Andres M. Lozano MD, Paul W. Frankland PhD

During adulthood, new neurons are continuously added to the dentate gyrus (DG), a subregion of the hippocampus essential to memory formation. These adult-generated cells mature, integrate into the hippocampal circuitry, and eventually contribute to hippocampus-dependent memory formation. Given that hippocampal-degeneration occurs in Alzheimer's and related pathologies, promoting adult neurogenesis therefore offers the potential for restoring mnemonic function in the diseased brain. Limbic structures harbor many DG connections, including the entorhinal cortex (EC) via the perforant pathway. Moreover, targeted electrical stimulation of these regions can increase hippocampal neurogenesis. Deep brain stimulation (DBS), an established therapy, could possibly achieve this. However, it is not known if increasing neurogenesis in this way adds functional new neurons. Here, 8 week-old wild-type mice underwent 1 h of EC electrical stimulation using clinically analogous parameters. Thymidine-analogue cell labeling demonstrated a nearly doubling of DG proliferation that peaked 3-5 days following stimulation, and resulted in additional new neurons for at least 10 weeks. DG apoptosis was unaffected. Survival and neuronal differentiation of recently born cells was modestly enhanced. GFP expressing retroviral-labeling revealed normal morphological development of newborn neurons following stimulation. Furthermore, immediate-early gene expression within labeled neurons, indicating neuronal activation, demonstrated that increasing new neuron availability led to a proportional rise in their contribution to hippocampal networks supporting Morris water maze memory. Finally, stimulation-induced increased neurogenesis was associated with improved water maze performance consistent with the maturational timeline of adult-generated granule cells. Collectively, these findings suggest that targeted electrical stimulation of DG inputs can augment adult hippocampal neurogenesis and the participation of adult-generated granule cells in memory networks at the cellular and behavioral level, evoking a potential utility for DBS as a neurogenerative therapy.

SATURDAY, NOVEMBER 6

SCIENTIFIC PROGRAM

American Academy of Neurological Surgery Annual Meeting The Inn at Spanish Bay, Pebble Beach, CA, November 3-6, 2010

PAPER PRESENTATIONS

8:30 – 8:42 AM **EXPERIMENTAL TRAUMATIC BRAIN INJURY ALTERS THE ORGANIZATION OF THE MOSSY FIBER PROJECTION IN THE IMMATURE RAT HIPPOCAMPUS**

P. David Adelson, MD, FACS, FAAP, Fan Zhang, MD, J. Patrick Card, PhD

INTRODUCTION/HYPOTHESIS: We have previously shown that aberrant pathways exist within the hippocampus (HC) in children following resection for medically intractable epilepsy, particularly in the Mossy Fiber (MF) projection, an area of the HC thought to be involved in memory and learning. Similarly, children are known to be more susceptible to developing post traumatic seizures, occuring in 10-20% who suffer a moderate or severe traumatic brain injury (TBI) and also frequently suffer from cognitive deficits post injury. As part of our ongoing investigation into the acute and chronic injury and reorganization following TBI, we previously demonstrated that following experimental TBI using controlled cortical impact (CCI), cognitive function is altered as well as hippocampal cytoarchitecture in immature rats (postnatal day (PND) 7 and 17) in an age dependent manner. Using viral transneuronal tracing, the basic organization of the "tri-synaptic circuit" though was preserved by post injury day (PID) 30 indicating that the neonatal HC has the capacity to reorganize following injury. The details of the synaptology within hippocampal circuits though have yet to be defined. We hypothesized that despite intact connectivity, following experimental TBI, the HC would be histopathologically altered, specifically in the MF projection system and might explain the cognitive functional deficits seen following this type of injury.

METHODS: To characterize the reorganization of the hippocampal MF projection system in immature animals at two post natal ages, Sprague Dawley PND 7 and 17 rats were injured using CCI (3 mm tip, 4m/ sec, deflection= 1.75 mm and 6 mm tip, 4m/ sec, deflection= 2.0 mm, respectively) (sham- operated but uninjured controls). At PID 30, coronal sections (200 um) were Timm's stained to visualize the MF pathway extending from dentate gyrus (DG) granule cells to stratum lucidum of CA3.

RESULTS: At PID 30, CCI induced a necrotic loss of cortex at the site of impact and variable damage to the underlying HC that was more extensive in PND 17 than PND 7, and not present in sham. The MF pathway ipsilateral to injury was truncated in its mediolateral extent in both age groups, consistent with the loss of hippocampal volume however, age dependent changes were observed in the cross-sectional width of stratum lucidum defined by MF afferents. In PND 7, stratum lucidum was increased ipsilateral to injury compared to the contralateral HC and to sham animals. A smaller increase was seen in PND 17 including MF sprouting into the molecular layer of the DG with the magnitude greatest at the core of the contusion. Analysis of the DG demonstrated that the thickness of the ipsilateral granule cell layer was comparable to the contralateral side in PND 7 in the area and cross-sectional widths of the dorsal and ventral blades. In contrast, the thickness of the ipsilateral granule cell layer in animals injured on PND 17 was reduced compared to that in the contralateral HC.

CONCLUSIONS: Our data indicates that in the developing (PND 7 and 17) rat, experimental TBI using CCI resulted in an alteration in the cytoarchitecture of the MF pathway of the HC 1 month after injury during an age where the MF are in various stages of extensive growth and development. These data provide insight into the trauma-induced reorganization of this HC projection system during its crucial developmental phases. Further electrophysiologic, histopathologic, and molecular study is necessary to correlate these findings with the clinical condition and potential correlates for post traumatic cognitive deficits and seizures/ epilepsy.

SATURDAY, NOVEMBER 6

8:44 – 8:56 AM **POLOXAMER-188 AND BUMETANIDE: POTENTIAL COMBINATORIAL NEUROPROTECTIVE THERAPY IN AN EXPERIMENTAL MODEL OF NEURAL INJURY**

David M. Frim, MD, PhD, David A. Wright, PhD

INTRODUCTION: The synthetic surfactant Poloxamer-188 (P-188) is a potent neuroprotectant after infusion of quinolinate, an NMDA-receptor agonist, into rat striatum. Consistent with observations that a late step in NMDA-receptor mediated neurotoxicity is lipid peroxidation and membrane degradation, P-188 can repair neuronal membranes after poration and prevent cell death. Bumetanide inhibits the Na-K-2Cl co-transporter (NKCC1) and in the setting of membrane degradation may help to maintain cellular ion gradients. Therefore, we hypothesize that Bumetanide, in and of itself, should prove to be a neuroprotectant after striatal quinolinate infusion. Beyond that, if our understanding of P-188 neuroprotection is correct, combining P-188 and Bumetanide in a single therapeutic intervention should provide additional combinatorial protection due to maintenance of cellular membrane integrity and maintenance of cellular ion gradients—two independent mechanisms.

METHODOLOGY: Quinolinate was infused into rat striata in a standard model of lesioning. Animals were subsequently treated with P-188, Bumetanide, or a combination of both. Drugs, and an artificial CSF control, were delivered intrathecally by an intracisternal route. Animals were sacrificed 7 days post lesioning, brains were removed, sectioned, and analyzed for volume of neuronal loss by NeuN immunostaining.

RESULTS: P-188 treatment resulted in a 27% decrease in volumes of neuronal loss. As theorized, Bumetanide treatment was also associated with an approximately 30% decrease in volumes of neuronal loss. Surprisingly, the combination of P-188 and Bumetanide was not associated with any decrease in lesion volume when compared to the artificial CSF control.

CONCLUSIONS: P-188, as previously shown, is a potent neuroprotectant after quinolinate lesioning. Bumetanide, hypothesized to be a neuroprotectant through stabilization of ionic gradients, is also found to reduce neuronal lesion volumes after quinolinate lesioning. However, when given in combination, these two neuroprotectants are not synergistic, and may in fact be antagonistic. Though issues of dosing and drug delivery in this model have yet to be elucidated in confirming this finding; if true, current understanding of the mechanisms of surfactant-mediated and ionic gradient-mediated neuroprotection after quinolinate lesioning will need to be revised.

SATURDAY, NOVEMBER 7

8:58 – 9:10 AM **A PROSPECTIVE RANDOMIZED TRIAL COMPARING EXPANSILE CERVICAL LAMINOPLASTY VERSUS CERVICAL LAMINECTOMY AND FUSION FOR MULTI-LEVEL CERVICAL MYELOPATHY**

Allan D. Levi, MD, PhD

INTRODUCTION/HYPOTHESIS: Cervical laminoplasty is commonly used in the Orient particularly for patients with ossification of the posterior longitudinal ligament and cervical myelopathy with excellent long-term results. Cervical laminectomy and fusion with instrumentation is commonly used in North America for multi-level stenosis and has become increasingly popular with the advent of the user-friendly posterior screw-rod systems. We sought to determine clinical, radiological and patient satisfaction outcomes between these two surgical procedures.

METHODS: We performed a prospective, randomized study of Expansile Cervical Laminoplasty (ECL) vs. Cervical Laminectomy and Fusion (CLF) in patients suffering from cervical spondylotic myelopathy. Consent and randomization occurred prior to surgery. End-points included the SF-36, the neck disability index (NDI), visual analog scales for neck, interscapular and arm pain, modified Japanese Orthopedic Association score, Nurick score and radiographic measures of cervical alignment, motion and spinal canal area pre-operatively and one year post-operative.

RESULTS: A survey of academic North American spine surgeons (n=30) demonstrated that CLF is the most commonly used (70%) posterior procedure to treat multi-level spondylotic cervical myelopathy, while ECL was used only by 23% of surgeons. A total of 16 patients consented and were randomized: CLF (n=7) / ECL (n=9). There were no operative complications in either group, but trends toward an increase in operative time, blood loss and length of stay was seen in the CLF group. Both groups (ECL and CLF) showed improvements in their Nurick grade and JOA score postoperatively, but only the improvement in the Nurick grade for the ECL group was statistically significant ($p<0.05$). Improvements in neck pain, interscapular, arm pain, SF-36 and NDI were seen in both groups but significant improvements ($p<0.05$) between pre- and post-op within patients were only seen in the ECL group. There was an increase in cervical kyphosis between C2 and C7 as measured by the neutral curvature index (CI) at 1 year in the ECL (-4.45°) group and to an equal extent in the CLF (-4.41°) group. The cervical ROM between C2 and C7 was reduced by 75% in the CLF group and by only 20% in the ECL group when comparing pre- and post-op range of motion. The overall increase in canal area was significantly ($p<0.001$) greater in the CLF when measured at the 3 most stenotic levels, but there was a suggestion that the adjacent level was more narrowed in the CLF group in as little as 1 year post-operative.

CONCLUSION: ECL compares favorably in many respects to CLF. While patient numbers are small, there were significant improvements in pain measures in the ECL group while still maintaining range of motion. Restoration of spinal canal area was superior in the CLF group.

SATURDAY, NOVEMBER 7

9:12 – 9:24 AM **TEMPORAL LOBECTOMY FOR MEDICALLY INTRACTABLE EPILEPSY: EFFECT OF THE EXTENT OF HIPPOCAMPAL RESECTION IN PATIENTS WITH HIPPOCAMPAL SCLEROSIS AND WITH NORMAL PATHOLOGY**

Richard Byrne, MD

INTRODUCTION: The technique of tailored temporal lobectomy allows for a choice of the extent of resection of the lateral temporal lobe and the hippocampus as dictated by the patient's pre-operative and intra-operative findings. This technique thus allows for an evaluation of the importance of the extent of hippocampal resection.

METHODS: A retrospective analysis was performed on 222 consecutive non-selected patients with at least 1 year follow-up. All data relevant to post-operative surgical outcome was evaluated using univariate (chi-squared, Fisher's exact test, T-test, and wilcoxon rank sum test) and multivariate analysis (logistic regression analysis) with attention to the effect of the extent of resection of the hippocampus as it relates to epilepsy outcome as measured by Engel's classification. A sub-analysis was performed in patients with hippocampal sclerosis and with normal pathology. A complete hippocampal resection was considered a resection to the level of the tectum. A partial resection was taken to the level of the choroidal point.

RESULTS: With a mean follow-up of 5.4 years, 70% of patients achieved Engel's class 1 outcome. Patients with lesional pathology were significantly more likely to achieve Engel's class 1 outcome on both univariate ($p=0.0008$) and multivariate analysis ($p=0.04$, OR=2.1, CI=[1.03,4.31]. There was no difference in likelihood of Engel's class 1 outcome between patients who had a complete hippocampectomy($n=113$) and a partial hippocampectomy($n=82$) ($p=0.47$). However, on analysis of Engel's class subgroups a-d, patients who has a complete hippocampectomy were more likely to achieve an Engel's class 1a outcome ($p=0.05$ univariate and $p=0.04$ multivariate). This was also true among patients with hippocampal sclerosis ($p=0.02$ multivariate). Among patients with normal pathology there was no difference in outcome among patients who underwent partial or complete hippocampectomy. There was no difference in the extent of lateral neocortex resection between the two groups (mean 3.49cm vs 3.59cm), but there was a difference in length of follow-up (3.7yrs vs 7.4yrs $p<0.0001$).

CONCLUSIONS: Using the technique of tailored temporal lobectomy, patients who undergo a partial hippocampectomy are as likely to achieve an Engel's class 1 outcome as those who undergo a complete hippocampectomy, but may be less likely to maintain class 1a outcome on long term follow-up. The decision to perform a partial hippocampectomy must take this difference into consideration.

SATURDAY, NOVEMBER 6

9:26 – 9:38 AM **HUMAN WIRELESS ELECTROCHEMICAL RECORDINGS DURING DEEP BRAIN STIMULATION NEUROSURGERY USING THE WINCS SYSTEM IN PARKINSON'S AND ESSENTIAL TREMOR PATIENTS**

Kendall H. Lee, M.D., Ph.D., Su-youne Chang, Ph.D., Inyoung Kim, B.A., Kevin E. Bennet, .S.Ch.E., M.B.A, Paul Garris, Ph.D., Charles Blaha, Ph.D.

INTRODUCTION: Deep brain stimulation (DBS) has been demonstrated to be an effective neurosurgical treatment for several pathologies including Parkinson's disease, tremor, epilepsy, depression, and chronic pain. We have developed a novel intraoperative neurochemical monitoring system, using wireless instantaneous neurotransmitter concentration sensor (WINCS) system, which combines digital telemetry with amperometry and fast-scan cyclic voltammetry (FSCV) for real-time, chemically resolved measurements at an implanted microelectrode of neurotransmitters including dopamine, adenosine, serotonin, glutamate, and histamine. Here we describe our first application of the WINCS system in human Parkinson's Disease (PD) and Essential Tremor (ET) patients during DBS neurosurgery. Our protocol was approved by the Mayo Clinic IRB for human studies.

METHODOLOGY: WINCS hardware is a micro-processor controlled, MRI-compatible, battery-powered instrument that combines Bluetooth digital telemetry with FSCV and constant potential amperometry. The WINCS base-station software (WINCSware) controls the wireless patient module, filters and processes the received data stream, and displays the results in nearly real time. The WINCS Electrode (WINCStrode) is based on an approved human extracellular tungsten electrophysiology electrode that was modified by the addition of a short section of carbon-fiber to enable FSCV recordings. To date, the WINCS hardware, WINCSware, and WINCStrode were used in 10 human PD or ET Patients during clinical subthalamic nucleus (STN) or ventralis intermedius (VIM) thalamic DBS neurosurgery, respectively. Leksell Model G Stereotactic headframe, MRI guided neuronavigation for surgical targeting, and Alpha-Omega computer driven electrode delivery system were also utilized.

RESULTS: Successful real-time analysis of wireless FSCV recordings from WINCS using the WINCStrode were performed both in a flow cell *in vitro* and in human PD and ET patients undergoing DBS neurosurgery. For FSCV, neurochemical data were transmitted by the WINCS hardware to the computer base station for presentation of a continuous three-dimensional color plot of sequential background-subtracted voltammograms. Analysis of the voltammograms revealed signals consistent with measurements of dopamine efflux when the WINCStrode was placed in the caudate and adenosine efflux when placed in the VIM thalamus. Following WINCS recordings, the patients had good clinical response from the bilateral STN or VIM DBS electrodes, without complication.

CONCLUSIONS: This study represents the first successful feasibility and safety study using WINCS in humans. We believe the combination of these sophisticated *in vivo* techniques will provide important new insights into the neurobiological mechanisms of DBS action. Further, our results suggest that next generation DBS systems that couples digital telemetry with FSCV may be useful as the sensing component of a "smart" DBS device providing enhanced utility to human patients.

SATURDAY, NOVEMBER 6

9:40 – 9:52 AM **STEREOTACTIC RADIOSURGERY FOR GLOSSOPHARYNGEAL NEURALGIA: PRELIMINARY REPORT OF 4 CASES**

Bruce E. Pollock, M.D., Christopher J. Boes, M.D.

INTRODUCTION: Glossopharyngeal neuralgia (GPN) is a rare disorder characterized by severe, stabbing pain of the ear, posterior tongue, and throat. The treatment of GPN is generally medical therapy initially with surgery reserved for patients who continue to have pain or who experience significant side effects. Sectioning of the glossopharyngeal nerve and upper rootlets of the vagus nerve or microvascular decompression (MVD) are associated with high rates of pain relief (80-90%), but persistent lower cranial nerve damage has been reported in 8 to 19 percent of patients. We report our early experience of using stereotactic radiosurgery as an alternative to posterior fossa surgery for patients with medically resistant GPN.

METHODOLOGY: Four patients (3 men, 1 woman) with medically resistant GPN underwent Gamma Knife ® radiosurgery. The median patient age was 67 years (range, 60-83) and the median pain duration was 5 years (range, 1 month-6 years). Three patients had persistent pain despite medical therapy, whereas one patient was pain-free on carbamazepine but developed thrombocytopenia. One patient had documented asystole in conjunction with his painful attacks. Dose planning was performed using a combination of stereotactic three-dimensional MRI and CT. The radiosurgical target was the distal portion of the glossopharyngeal nerve at the level of the glossopharyngeal meatus. The maximum radiation dose in all cases was 80 Gy. The median follow-up after radiosurgery was 13.5 months (range, 5-18).

RESULTS: Three patients became pain-free at 2 days, 3 days, and 2 weeks, respectively, and were able to discontinue the medications taken pre-operatively for their pain. None of these patients have suffered any recurrent pain since becoming pain-free. One patient had no benefit from the procedure and underwent a MVD five months after radiosurgery. No patient developed hoarseness or dysphagia after radiosurgery.

CONCLUSIONS: This preliminary experience demonstrates that stereotactic radiosurgery is possible as an option for patients with medically resistant GPN. Additional follow-up and a larger number of patients are needed to demonstrate the long-term safety and optimal radiation dosimetry for this indication.

SATURDAY, NOVEMBER 6

9:54 – 10:06 AM **DEEP BRAIN STIMULAITON (DBS) OF THE VENTRAL CAPSULE/VENTRAL STRIATUM (VC/VS) FOR THE TREATMENT OF NERUOBEHAVIORAL DISORDERS**

Ali R. Rezai MD

INTRODUCTION: Neurobehavioral disorders such as Obsessive compulsive disorder (OCD), Major Depression and addictions are common conditions, which can become severe, disabling and intractable despite best attempts with medication and behavioral therapy. A number of brain structures have been the targets for surgical interventions using RF and gamma knife lesioning and deep brain stimulation (DBS) in neurobehavioral disorders. The ventral anterior limb of the internal capsule and the ventral striatum (VC/VS) is a target of growing interest. This presentation will review the anatomical, physiological and circuit/network based rationale for VC/VS surgery for neurobehavioral disorders. The surgical procedure, outcome, and the mechanisms of DBS for refractory OCD and major depression will be discussed.

PATIENTS AND METHODS: Severe and treatment refractory OCD (n=26) and major depression (n=15) patients were enrolled in a prospective multi-center study open label study. All patients underwent a standardized battery of assessments at baseline and various intervals after DBS. Patients underwent stereotactic implantation of bilateral DBS in the ventral anterior limb of the internal capsule and ventral striatum (VC/VS) using image-guidance and physiological testing and verification.

OUTCOME: The long term (>12 months) outcome of VC/VS DBS for severe and intractable OCD depression patients demonstrates significant improvements in standardized scales (Yale Brown Obsessive Compulsive Scale, Hamilton Depression and Anxiety, Montgomery Asberg Depression) and quality of life measures. Overall, more than 50% of these severe and incapacitated patients became treatment responders with DBS. There were no permanent and significant complications.

SUMMARY AND DISCUSSION: VC/VS DBS appears to be a safe and effective surgical approach and option for patients with severe and intractable OCD and depression. Phase III randomized controlled studies are currently in progress. The anatomy, circuitry, and role of the VC/VS region will be reviewed in the context of mood, anxiety and addictive disorders. The rationale for VC/VS DBS and the mechanisms of VC/VS DBS action will be examined. The emerging use of VC/VS surgery for addiction and severe anorexia nervosa will be also be discussed.

SATURDAY, NOVEMBER 6

10:28 – 10:40 AM **DYNAMICS OF THE DEEP-BRAIN STIMULATOR TISSUE-ELECTRODE INTERFACE**

Karl Sillay MD, Joseph Hippensteel MS

INTRODUCTION: The dynamic nature of the electrochemical interface between deep brain stimulation (DBS) devices and the brain is intricately linked to the efficacy and safety of DBS therapy. Extensive literature exists describing representative interfaces using model systems and computer simulations, but there is inadequate empirical knowledge of these characteristics in humans. The Medtronic (Minneapolis, MN) Soletra DBS device provides clinicians with significant information about this interface by providing an option to record therapeutic electrode impedances. These therapeutic impedances are collected to ensure that the interface remains intact, and is typically interpreted as a binary indicator of functionality. It is posited that these measurements provide a unique opportunity to investigate and potentially characterize this incompletely understood aspect of DBS therapy.

METHODOLOGY: A retrospective review of 89 patients implanted with Soletra DBS devices between January 2001 and May 2009 at the University of Wisconsin Hospitals and Clinics was conducted. Impedance measurements and stimulation settings were extracted from each patient's electronic chart. Final analysis was restricted to patient data with consistent programming settings for a period of three or more days without stimulator setting changes. Additionally, sequences with an impedance recording registered as >2000 Ohms were not included in final analysis. A total of 71 patients, 127 electrodes, and 661 data points fit these criteria. Variability in impedance with time and electrode settings was explored. Data was partitioned into monopolar electrode settings (MP; DBS case as cathode) and bipolar (BP; DBS case not used as cathode) and analyzed in the Matlab (Natick, MA) programming environment.

RESULTS: It was found that the mean MP impedance of 979 Ohms across all times was significantly different than the mean BP impedance of 1224 Ohms ($p << .001$). Correlations between impedance and time from stimulator setting change were analyzed for all data, data from the first 100 days after a stimulator setting change and data from within 18 days of a stimulator setting change. Patients with MP settings were found to have a significant decrease in impedance of .25 Ohms/Day over the first 18 days post-setting change ($P < .0021$). No significant difference in rate of impedance variation was found between MP and BP settings.

CONCLUSIONS: The first significant finding indicates that impedance measurements using the built-in therapeutic impedance function of the Soletra DBS device can differentiate between the higher impedance of BP electrode orientation when compared to MP. This is consistent with the larger surface area of charge transfer available during MP stimulation compared to BP. The immediate decrease in MP impedance seen following a setting change may be the result of a transient disruption of a previously stable electrode-tissue interface. Similar results have been reported following brief polarization of stimulating microelectrodes in model systems. The results of this study suggest the efficacy of therapeutic impedance measurements for characterizing and monitoring the electrode-tissue interface during DBS therapy and is a major step in further understanding this dynamic system.

SATURDAY, NOVEMBER 6

10:42 – 10:54 AM **DEEP BRAIN STIMULATION OF THE SUBTHALAMIC NUCLEUS IN PRIMARY CERVICAL DYSTONIA: RESULTS OF A PILOT TRIAL**

Philip A. Starr, MD, PhD, Caroline A. Racine, PhD, Graham A. Glass, MD, Andrea Crowell, MD, Shoichi Shimamoto, BS, Jamie Grace, BA, Jill L. Ostrem, MD

INTRODUCTION: The standard brain target for deep brain stimulation (DBS) to treat primary dystonia has been the globus pallidus internus (GPi). However, in patients with predominantly cervical dystonia, GPi-DBS may produce reversible bradykinesia in previously normal limbs, limiting its therapeutic efficacy. The subthalamic nucleus (STN), commonly implanted in patients with Parkinson's disease (PD), represents an alternative target for the modulation of basal ganglia function. We therefore performed a pilot clinical trial of STN DBS in patients with primary cervical dystonia. In order to better understand the pathophysiology of primary dystonia, we also evaluated STN single unit discharge, and STN and cortical local field potentials (LFPs).

METHODS: Eight patients ((5M, 3F; mean age 46 yrs (range 24-71)) with medically refractory primary, predominately cervical dystonia were enrolled in a prospective open-label clinical trial of bilateral STN DBS implantation. Severity of dystonia was rated by a blinded neurologist using the videotaped Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS) severity subscale preoperatively, and at 3, 6, and 12 months post-surgery. Neuropsychological testing was performed at baseline and 12 months postoperatively. Subthalamic single neuron discharge, and subthalamic and primary motor cortex local field potentials (LFPs), were recorded intraoperatively and analyzed off-line for firing rate and pattern and for frequency domain characteristics of the LFPs.

RESULTS: STN DBS was well tolerated with no serious adverse effects. Twelve months after STN DBS, the total TWSTRS score improved by 59.8 % from a mean ($\pm$SEM) baseline score of 54.8 ($\pm$2.2) to 21.4 ($\pm$5.9), ($p=0.012$, Wilcoxon signed-rank test). STN DBS induced no deficits in verbal fluency or other neuropsychological measures. No patients developed bradykinetic side effects, but all patients developed transient dyskinetic movements with DBS stimulation, that was relieved by programming adjustments. Mean ($\pm$ SD) STN neuronal firing rate in dystonia was 26.3 $\pm$ 13.6 Hz (N=62 neurons), significantly lower ($p<.001$, Wilcoxon test) than STN firing route in a comparison group of idiopathic PD (35.6 $\pm$ 15.2 Hz, N=143 neurons). Analysis of cortical and STN LFPs revealed an impairment of movement-related beta desychronization in dystonia.

CONCLUSIONS: This single-blind, prospective study showed that patients treated with bilateral STN DBS had significant improvements in dystonia after surgery and suggests that STN DBS may be an important alternative to GPi DBS, with fewer persistent stimulation-induced adverse effects. Intraoperative physiological studies suggest a novel hypothesis for the pathophysiology of primary dystonia: Overflow of muscle activation during movement is the result of excessive beta band oscillatory activity in the basal ganglia-thalamocortical circuit.

SATURDAY, NOVEMBER 6

10:56 – 11:08 AM **THE EFFECTS OF DISEASE AND AGING ON NEURAL GRAFTS: IMPLICATIONS FOR FUTURE STEM CELL THERAPIES**

Thomas B. Freeman, M.D., Francesca Cicchetti, Ph.D., Q. C. Sante-Foy, Robert A. Hauser, M.D., Yaping Chu, Ph.D, Sam Saporta, M.D., Elliott J. Mufson, Ph.D., Warren Olanow, M.D., Jeffrey H. Kordower, Ph.D.

INTRODUCTION: Autopsy evaluations of neural transplants in patients with Parkinson's and Huntington's diseases have demonstrated transmission of the diseases to the grafts. There is additional evidence that aging may also adversely affect embryonic transplants *in vivo*. Here, we address the possible mechanisms that may be responsible for these observations, and delineate opportunities as well as obstacles for the future utilization of stem cell therapies in the treatment of neurologic disorders.

METHODS: Autopsy studies of 2 patients with Parkinson's disease (PD) and 3 with Huntington's disease (HD) following neural transplantation were evaluated at 10-14 years after transplantation. Grafts were evaluated immunohistochemically and with electron microscopy.

RESULTS: Neural transplants in patients with PD developed Lewy body-like inclusions that resembled the actual disease in the patient's substantia nigra. This occurred in spite of the fact that the neural transplants were unrelated genetically or immunologically to the recipient. Clinical efficacy in these two patients lasted approximately 12 years. Only a small minority of grafted neurons developed these inclusion bodies. Metabolic changes (decreased dopamine transporter expression) was also observed in the grafts, similar to PD.

Similarly, neural transplants in the 3 HD patients underwent disease-like neuronal degeneration, but in these cases there were no inclusions seen within the transplanted cells. Instead, the grafts underwent disease-like neural degeneration via different mechanisms, most likely related to cortical-striatal glutamatergic neurotoxicity as well as aberrant microglial inflammatory changes. Clinical efficacy was quite limited and short term, at best. Poor trophic support in the caudate in HD and aging in both diseases may have contributed to these findings as well.

CONCLUSIONS: These findings speak to the vulnerability of transplanted neurons to the underlying disease processes, and preliminary evidence suggests that these types of observations will generalize to stem cell therapies as well. The mechanisms and severity of these disease-like changes in the transplants are unique to each specific disease. Factors such as age, loss of trophic support in the brain and technical aspects of graft preparation may also contribute to these findings as well. Finally, clinical benefits, in spite of evidence of disease within the grafts, may still be meaningful and quite long lasting, particularly in the case of Parkinson's disease.

SATURDAY, NOVEMBER 6

11:10 – 11:22 AM **DOSE AND DURATION OF NERVE GROWTH FACTOR (NGF) ADMINISTRATION DETERMINE BEHAVIORAL RECOVERY FOLLOWING PERIPHERAL NERVE INJURY**

Stephen WP Kemp, PhD, Aubrey A Webb, DVM, PhD and ***Rajiv Midha, MD, MSc, FRCS.***

Nerve Growth Factor (NGF) has been previously shown to support neuron survival and neurite outgrowth *in vitro*, and to enhance sprouting as well as regeneration of sympathetic and sensory neurons *in vivo*. Due to its lack of effect on motor neuron regeneration and mixed results for functional outcomes, investigators have questioned the efficacy of NGF on improving behavioral recovery after nerve injury. We undertook a systematic analysis of NGF dose and dose duration on nerve regeneration and behavioral recovery following peripheral nerve injury in rodents with the ***hypothesis*** that optimal NGF dosing would influence functional outcomes and behavioral recovery. Using a 5 mm sciatic nerve injury gap model in rats, repaired with a nerve tube, the entubalation gap site was exposed to NGF administered daily over 7 days via a microinjection port. We found that NGF promoted a bell shaped dose-response on nerve regeneration, with an optimal effect occurring at a concentration of 800 pg/ul (80 ng/day). High dose (160 or 240 ng/day) NGF significantly retarded regeneration, an effect which could be reversed through functional blockade of the low affinity p75NGF receptors, thus implicating these receptors as mediators of the inhibitory response. In separate groups of rats, longer term (up to 12 weeks) evaluation showed that animals administered NGF at 80 ng/day for a 3 week period recovered behavioral function to a greater extent than all other treatment groups, including those animals which were administered NGF for 1 week duration. Animals given NGF over 3 weeks committed significantly less errors in skilled locomotor (horizontal ladder rung and tapered beam) assessments, and on video gait analysis displayed both increased vertical and fore-aft ground reaction forces during free flat surface locomotion than the other nerve injury gap groups. Furthermore, histomorphometric indices of the regenerated nerve population and terminal functional assessments (CMAP amplitudes and wet gastrocnemius muscle weights) were significantly superior, corroborating the behavioral data. Remarkably, rats in the 3 week NGF group had significantly improved behavioral and terminal outcomes than rats receiving a direct nerve repair (the current gold standard of treatment). All treatment groups had similar levels of hip, knee and ankle joint angle abnormalities on kinematic gait assessment. Tests for mechanical (von Frey hair) allodynia and thermal nociception demonstrated that all sciatic nerve injured rats exhibited similar levels of hypersensitivity, with no discernable differences in the rat groups receiving either 1 or 3 weeks NGF, as compared to saline controls and ones receiving nerve autografts. Collectively, these data support the hypothesis that exogenous NGF therapy at both appropriate dose and duration can profoundly influence several facets of behavioral recovery and functional outcome following nerve injury in the rat.

SATURDAY, NOVEMBER 6

11:24 – 11:36 AM

A NEW TREATMENT PARADIGM: NEO-ADJUVANT RADIOSURGERY PRIOR TO SURGICAL RESECTION OF BRAIN METASTASES WITH ANALYSIS OF LOCAL TUMOR RECURRENCE

Anthony L. Asher, MD, FACS, Stuart H. Burri, MD, Renee P. Kelly, RN, BSN, Margaret Boltes RN, BSN, OCN, CCRP, Melissa Mehrlich, RN, BSN, H. James Norton, PhD., Robert W. Fraser. MD, FACR

INTRODUCTION: Surgical resection alone of brain metastases (BM) results in an unacceptably high risk of recurrence. Whole brain radiation therapy (WBRT) following surgical resection reduces recurrence, but with near-term and potential long-term toxicity. There is little published data utilizing post-operative SRS and target delineation can be problematic. SRS delivered in the pre-operative setting (neo-adjuvant SRS or NaSRS) allows clearer target definition with the theoretic benefit of reducing intra-operative dissemination of viable tumor cells.

METHODS: In 2005 our institution adopted treatment of resectable BM with NaSRS. Subsequently, a prospective trial of NaSRS was undertaken. A total of 47 consecutively treated patients (23 database, and 24 prospective trial) from 2005-2008 with a total of 51 lesions are reviewed. No statistical difference was observed between the two cohorts in regards to outcome, thus the data were combined for analysis. Median follow-up was 12 months (range 1-58) with median age of 57 and KPS=90 at time of treatment. A median of one day elapsed between NaSRS and resection. The median size of the lesions (largest cross sectional diameter) was 3.04cm (range 1.34-5.21cm) with a median volume of 8.49cc (range 0.89-46.7cc). An approximately 20% dose reduction from standard protocol (for size) was utilized based on the theory that NaSRS was only required to control microscopic residual tumor at the resection margin rather than gross disease. The median dose was 14 Gy (range 11.6-18Gy) prescribed to 80% isodose line. 37.25, 23.5 and 19.6% of patients had lung, breast and melanoma primaries, respectively.

RESULTS: Actuarial overall survival was 77.8%, 60.0% and 26.9% at six, twelve, and twenty-four months respectively. Actuarial local control was 97.8%, 85.6% and 71.8% at six, twelve, and twenty-four months respectively. Five of the eight failures were proven pathologically without evidence of radiation necrosis (RN). There were no peri-operative adverse events. 16.7% of the patients were ultimately treated with WBRT. Local failure was statistically more likely with lesions >10cc (p=.01), >3.4cm (p=.014), with a trend observed in surface lesions (p=.066) and eloquent areas (p=.052). Failure also trended to statistical significance in NSCLC patients. Upon further analysis, six of the eight failures had either a broad dural base or proximity to draining veins that complicated total surgical resection.

CONCLUSIONS: NaSRS prior to surgical resection can be performed safely and effectively with excellent results without documented RN. Local control was excellent even in the setting of large (> 3 cm) lesions. The strong majority of patients were able to avoid WBRT. We conclude that NaSRS merits consideration for a multi-institution trial, with reduced dosing (as employed here) and standard dosing paradigms to determine if the excellent local control results can be further improved.

SATURDAY, NOVEMBER 6

11:38 – 11:50 AM **BONE MARROW MESENCHYMAL STEM CELLS LOADED WITH AN ONCOLYTIC ADENOVIRUS SUPPRESS THE ANTI-ADENOVIRAL IMMUNE RESPONSE IN AN IMMUNOCOMPETENT MODEL.**

Atique U. Ahmed, PhD, Cleo E. Rolle, PhD, Matthew A. Tyler, B.A., Yu Han, B.A., Sadhak Sengupta, PhD, Derek Wainwright, PhD, Irina V. Balyasnikova, PhD, Ilya I. Ulasov, PhD and ***Maciej S. Lesniak, MD***

INTRODUCTION: Oncolytic adenoviral virotherapy is an attractive treatment modality for cancer. However, following intra-tumoral injections, oncolytic viruses fail to efficiently migrate away from the injection site and are rapidly cleared by the immune system. We have previously demonstrated enhanced viral delivery and replicative persistence *in vivo* using human bone marrow derived mesenchymal stem cells (MSC) as delivery vehicles. In the present study, we evaluated the immune response to adenovirus-loaded MSCs using the immunocompetent cotton rat (CR) model.

METHODS/RESULTS: First, we isolated MSCs from CR bone marrow aspirates. Real-time quantitative PCR analysis revealed that CR MSCs supported the replication of adenoviruses *in vitro*. Moreover, we observed similar levels of suppression of T cell proliferation in response to mitogenic stimulation, by MSCs alone and virus-loaded MSCs. Additionally, we found that MSCs suppressed the production of IFNγ by activated T cells. Most importantly, in our *in vivo* tumor model, CR MSCs enhanced the dissemination and persistence of adenovirus, compared to virus injection alone, effectively down-regulating the anti-adenoviral immune response.

CONCLUSIONS: Collectively, our data suggest that the use of MSCs as a delivery strategy for oncolytic adenovirus potentially offers a myriad of benefits, including improved delivery, enhanced dissemination, and increased persistence of viruses via suppression of the anti-viral immune response. These findings open up a promising new delivery strategy in the field of human gene therapy.

SATURDAY, NOVEMBER 6

11:52 – 12:04 PM **CpG ISLAND DNA METHYLATION SITES ARE ASSOCIATED WITH MALIGNANT POTENTIAL IN MENINGIOMAS**

Andrew E. Sloan, M.D., F.A.C.S.; Mark D. Adams, Ph.D., Mark Cohen, M.D., Nicholas Bambakidis, M.D., Robert Miller, Ph.D., Warren R. Selman, M.D., FACS, & Jill Barnholtz-Sloan, Ph.D.

The role of methylation, and epigenetics in the genesis of meningiomas is poorly understood. Meningiomas comprise nearly one-third of all brain tumors. While most are benign, atypical or malignant meningiomas may progress rapidly and are a cause of significant morbidity. Recent advances in our knowledge of the human epigenome, now facilitates high-throughput whole-genome methylation testing.

We used DNA extracted from snap-frozen tumor samples from 34 patients with newly diagnosed meningiomas (30 WHO Grade I, 2 WHO Grade II and 2 WHO Grade III) and performed genome-wide methylation analysis using the Illumina Infinium Human Methylation 27 BeadChip Assay which measures methylation status for 27,578 CpG sites in 14,000 genes. Methylation intensity is calculated as a normalized continuous measure that ranges from 0 for completely unmethylated to 1 for completely methylated. Two-sided t-tests were used to identify genes whose methylation intensity differed by benign and atypical/malignant groups using the autosomal CpG island sites only.

The correlation between replicate samples was >99%. In general, we find no significant differences in genome-wide average methylation intensity between the two groups. However, we found 465 CpG island markers in ~400 genes that differ between the 30 benign samples and the 3 higher grade samples using a stringent FDR corrected p-value. Further analysis showed that one of the “benign” samples clustered with the atypical group. Interestingly, this patient had early recurrence at which time the histopathology was atypical. When this patient was then grouped with the atypical samples, we found 231 CpG island markers in ~203 genes that differed between the 29 benign samples and the 4 atypical samples using a stringent FDR corrected p-value.

This analysis demonstrates that we can discover significant differences in gene-specific methylation patterns in meningioma associated with malignant potential and that these may potentially serve as molecular markers with prognostic implications. Integration of epigenetic, gene expression, and chromosomal patterns associated with meningioma grade will be discussed.

SATURDAY, NOVEMBER 6

12:06 – 12:18 PM **MP-MUS (I): A NOVEL MAO-B ACTIVATED PRO-DRUG WHICH SPECIFICALLY TARGETS AND DESTROYS GLIOMAL MITOCHONDRIA**

David S. Baskin, M.D., Marsha A. Widmayer, and Martyn A. Sharpe, Ph.D.

INTRODUCTION: Gliomas: Prognosis and Treatment. 10,000 Americans are diagnosed with malignant gliomas annually. The typical treatment, surgery, radiotherapy, and chemotherapy, is unfruitful with only 50% of patients surviving one year, 20% at two years and <3% alive at 5 years.

Monoamine oxidase B is upregulated in Gliomas. Monoamine oxidase B (MAO-B) catalyzes deamination of a wide variety of amines through a two-electron reduction of oxygen to hydrogen peroxide. MAO-B activity is four fold greater in glioblastoma multiforme, low-grade astrocytomas and in anaplastic astrocytomas than in postmortem control brains or meningiomas. This 4 fold difference will limit toxicity of any MAO-B bio-actived products in other cell types, while providing a highly specific toxin to glioma cells.

Exogenous MAO-B specific substrates. The best known exogenous substrate of MAO-B is MPTP, which is converted into the cation MPP^+ by glial cells. MPP^+ is a lipophilic cation and is concentrated inside mitochondria, with the accumulation driven by the membrane potential, **ΔΨ**.

Use of Gliomal MAO-B to bioactivate a pro-drug. We postulated that an anti-glioma drug could be synthesized by combining the MAO-B substrate characteristics of MPTP/MPP^+ with a conventional DNA acylating agent. A blood brain barrier permeable (neutrally charged) pro-drug could be converted into a (cationic) mitochondrially targeting, acylating agent, by gliomal MAO-B. Mitochondria **ΔΨ** driven accumulation of the mature drug would damage mtDNA/mtRNA. Such damage would affect both energy generation and pyrimidine synthesis, resulting in loss of mitochondria and eventual cell death.

MP-MUS (I) is a novel drug that specifically targets mitochondria for cancer treatment. We have designed, synthesized and tested a gliomal specific pro-drug, MP-MUS (I), is activated by MAO-B to form P^+-MUS (I), a mitochondrially targeted acylation agent.

RESULTS: MP-MUS (I): Cell growth and mitochondrial respiration. MP-MUS (I) is a potent toxin in both immortalized human gliomas (U87) and in primary gliomas. The LD_{50}, measured 24 hours after incubation, is ≈9μM, however a dosage of only≈2μM halves mitochondrial respiration.

MP-MUS (I): Mitochondrial membrane potential. We examined the accumulation of the mitochondrial **ΔΨ** reporting dye, Mitotracker Red, in primary glioma. The signal, per living cell, fell to only 30% of the control, in the presence of 9 μM MP-MUS (I). However, this collapse in mitochondrial function is almost completely arrested by co-administration of the MAO-B inhibitor, Selegiline, indicating that bio-activation is a key step in drug toxicity.

MP-MUS (I): Mitochondrial DNA. We examined the levels of DNA breaks and nicks in MP-MUS (I), treated cells. We find a, Selegiline sensitive, increase in the levels of DNA damage in treated cells. DNA damage is non-nuclear and is co-localized (>90%) with both mitochondrial **ΔΨ** dye and with mitochondrial proteins, including TWINKLE, mtRibosomal L11 and cytochrome *c*.

MP-MUS (I): Mitochondrial turnover. 24 hours after treatment with MP-MUS (I) a Selegiline sensitive, increase in mitochondrial proteins is evident. Elevated levels of TWINKLE, L11 and cytochrome *c* suggest that replacement of damaged mitochondria is occurring.

CONCLUSION: We have demonstrated, *in vitro*, that up-regulated, MAO-B in gliomas converts a mildly toxic pro-drug, MP-MUS (I), into at a highly toxic, mitochondrial targeted, mtDNA acylating agent, P^+-MUS (I). As bio-activation is MAO-B dependent, an enzyme up-regulated in gliomal cells, MP-MUS (I) will have limited toxicity towards other cells types, especially neurons.

SATURDAY, NOVEMBER 6

12:20 – 12:32 PM **INTRATUMORAL HEMORRHAGE AND FIBROSIS IN VESTIBULAR SCHWANNOMA: A POSSIBLE MECHANISM FOR HEARING LOSS**

ME Sughrue, R Kaur, AJ Kane, MJ Rutkowski, I Yang, LH Pitts, T Tihan, ***Andrew T. Parsa***

OBJECTIVE: Vestibular schwannomas (VSs) are benign lesions with an unpredictable natural history. Perhaps the greatest barrier to predicting which patients need treatment is our poor understanding of how these tumors cause hearing loss in the first place. In this case-control study, the authors investigated the relationship between preoperative hearing loss and histological changes such as intratumoral microhemorrhage and extensive fibrosis.

METHODS: From a prospectively collected database, the authors selected all patients with VS who had undergone microsurgical resection as their initial treatment for histopathologically confirmed VS. Histological specimens obtained in 274 of these patients were systematically reviewed by a blinded neuropathologist who graded the extent of microhemorrhage and fibrosis in these tumors. The effect of these variables on preoperative hearing loss was studied using binary logistic regression.

RESULTS: On univariate analysis, patients with extensive intratumoral microhemorrhage or fibrosis ($p < 0.0001$), patients with larger tumors ($p < 0.05$), and patients 65 years of age or older ($p < 0.05$) were significantly more likely to have unserviceable hearing at the time of surgery. On multivariate analysis, only patients with extensive intratumoral microhemorrhage or fibrosis had an increased risk of having unserviceable hearing at the time of surgery (OR 3.72, 95% CI 1.3-10; $p = 0.01$). Older age and tumor size greater than 3 cm were not statistically significant risk factors for hearing loss, controlling for the effect of microhemorrhage and fibrosis.

CONCLUSIONS: In this study, the authors have demonstrated a correlation between the extent of nonneoplastic histological changes, such as microhemorrhage and fibrosis, and hearing loss. Our results potentially explain many of the exceptions to previously described mechanisms of hearing loss in patients with VS; particularly for patients with no evidence of tumor growth. The advent of high-resolution MR imaging technology to identify microhemorrhages may provide a method to screen for patients with VS at risk for hearing loss, independent of tumor growth rate.

SATURDAY, NOVEMBER 6

12:34 – 12:46 PM **QUANTITATIVE FLUORESCENCE IN INTRACRANIAL TUMOR: IMPLICATIONS FOR ALA-INDUCED PpIX AS AN INTRAOPERATIVE MARKER**

David W. Roberts MD FACS, Pablo A. Valdés BS, Frederic Leblond PhD, Anthony Kim PhD, Brent T. Harris MD PhD, Brian C. Wilson PhD, Xiaoyao Fan BE, Tor D. Tosteson ScD, Alex Hartov PhD, Songbai Ji DSc, and Keith D. Paulsen PhD

INTRODUCTION: Qualitative fluorescence of protoporphyrin IX (PpIX), synthesized endogenously following 5-aminolevulinic acid (ALA) administration, has been used to guide resection in high-grade glioma. We show that diagnostically significant but visually imperceptible concentrations of PpIX can be quantitatively measured in vivo and used to discriminate normal from neoplastic brain tissue across a range of tumor histologies.

METHODS: We studied fifteen patients with diagnoses of low- and high-grade glioma, meningioma, and metastasis under an IRB-approved protocol for fluorescence-guided resection. The primary aim of the study was to compare the diagnostic capabilities of a highly sensitive, spectrally-resolved quantitative fluorescence approach to conventional fluorescence imaging for detection of neoplastic tissue in vivo.

RESULTS: A significant difference in the quantitative measurements of PpIX concentration occurred in all tumor groups compared to normal brain tissue. Receiver-operating-characteristic (ROC) analysis of PpIX concentration as a diagnostic variable for detection of neoplastic tissue yielded a classification efficiency of 87% (area-under-the-ROC-curve=0.95, specificity=92%, sensitivity=84%) compared to 66% (area-under-the-ROC-curve=0.73, specificity=100%, sensitivity=47%) for conventional fluorescence imaging ($P<0.0001$). More than 81% (57/70) of the quantitative fluorescence measurements that were below the threshold of the surgeon's visual perception were classified correctly in an all-tumors analysis.

CONCLUSIONS: These findings are clinically profound because they demonstrate that ALA-induced PpIX is a targeting biomarker for a variety of intracranial tumors beyond high-grade glioma. This study is the first to measure quantitative ALA-induced PpIX concentrations in vivo and the results have broad implications for guidance during surgical resection of intracranial tumor.

NOTES

SPECIAL GUESTS

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Hunt Batjer

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Thomas Freeman
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Karl Sillay Madison, WI	Berman Iskandar
Michael Taylor Toronto, Ontario	Jim Rutka
Phil Tibbs Lexington, KT	Russell Travis

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
Scellig Stone……..2010

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
Enchantment Resort, Sedona Arizona …… September 10-13, 2008
The Breakers, Palm Beach, Florida …… November 4-7, 2009
The Inn at Spanish Bay, Pebble Beach, California…….. November 3-6, 2010

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
Steven Giannotta ……...2010

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
Warren Selman ……2010

PAST SECRETARY-TREASURERS

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

PAST SECRETARIES

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

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EBEN ALEXANDER, JR.	1950	2004
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(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		
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EDWIN B. BOLDREY	1941	1988
San Francisco, California		
(Senior)		
E. HARRY BOTTERELL	1938	1997
Kingston, Ontario, CANADA		
(Senior)		
ROBERT BOURKE	1983	1996
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SPENCER BRADEN	Founder	1969
Cleveland, Ohio		
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