THE
AMERICAN ACADEMY
OF
NEUROLOGICAL SURGERY

74th Annual Meeting

The Chatham Bars Inn
Chatham/Cape Cod, Massachusetts

October 17-20, 2012

American
Association of
Neurological
Surgeons

Jointly Sponsored by AANS

FUTURE MEETINGS

2013

September 25-28, 2013
The Resort at Pelican Hill
Newport Beach, California

2014
TBD

Mark your calendars now!

GENERAL INFORMATION

HOTEL INFORMATION

Chatham Bars Inn
297 Shore Road
Chatham, Cape Cod, MA
1.800.527.4884 phone

REGISTRATION DESK LOCATION AND HOURS:

Wednesday, October 17	Main Inn Lobby	12:00 PM – 6:30 PM
Thursday, October 18	Main Inn Lobby	6:00 AM – 12:00 PM
Friday, October 19	Main Inn Lobby	6:00 AM – 12:00 PM
Saturday, October 18	Main Inn Lobby	6:00 AM – 12:00 PM

PROGRAM SUMMARY

WEDNESDAY, OCTOBER 17

EVENTS	TIME	LOCATION
Registration	12:00 PM-6:30 PM	Main Inn Lobby
Academy Executive Comm. Mtg	3:00 PM-5:00 PM	Executive Boardroom
Opening Reception Neurosurgery Jazz Band	6:00 PM -10:00 PM	Beach House Grill or South Lounge (rain)

THURSDAY, OCTOBER 18

EVENTS	TIME	LOCATION
Registration	6:00 AM-12:00 PM	Main Inn Lobby
Continental Breakfast (Members)	6:30 AM-7:30 AM	Monomoy Room
Continental Breakfast (Spouse/Guest)	6:30 AM-9:30 AM	Seaview Room
General Scientific Session	7:30 AM-12:30 PM	Monomoy Room
Breakfast at Puritan's/ Shopping in Chatham	9:00 AM	Meet Hotel Lobby for Chatham Bars Inn's Trolley
Film/Video Discussion (Spouse/Guest)	9:30 AM	Seaview Room
Lunch		At Leisure on Own
Golf Tournament-	1:06 PM	Brewster Captains Course Port Course
Deep Sea Fishing	1:00 PM-5:00 PM	Local Pier TBA
Beach Side Hike with a Naturalist	TBD	Meet Main Inn Lobby
Seaside Lobster Bake	6:30 PM-	Beach House Grill

FRIDAY, OCTOBER 19

Registration	**6:00 AM-12:00 PM**	**Main Inn Lobby**
Breakfast (Members)	**6:30 AM-7:30 AM**	**Monomoy Room**
Breakfast (Spouse and Guest)	**6:30 AM-9:30 AM**	**Seaview Room**
General Scientific Session	**7:30 AM-12:30 PM**	**Monomoy Room**
Book Discussion, Spouse/ Guest, led by-Mari Rutka	**10:00 AM**	**Seaview Room**
Presidential Address	**11:55 AM**	**Monomoy Room**
Lunch		**At Leisure On Own**
Golf	**1:06 PM**	**Captains Course Starboard Course**
Seal Watching/Monomoy Cruise	**TBD**	**Local Pier TBA**
Black Tie Optional Reception	**6:30 PM-7:30 PM**	**South Lounge**
Black Tie Optional Dinner	**8:00 PM-11:30 PM**	**Main Dining Room,**

SATURDAY, OCTOBER 20

Registration	**6:00 AM-12:00 PM**	**Main Inn Lobby**
Breakfast All together	**6:30 AM-9:30 AM**	**Seaview Room**
General Scientific Session	**7:30 AM-12:30 PM**	**Monomoy Room**

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74th Annual Meeting

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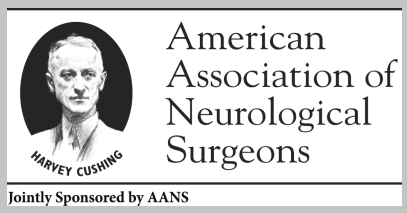
Mission Statement:

The purpose of the live Academy meeting shall be to promote scientific and social interaction 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



Learning Objectives

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

- Understand the most recent results of clinical trials comparing open vs. endovascular treatment
- Know which cases should be referred for endovascular treatment first vs. open craniotomy first
- Understand the principles of skull base endoscopy
- Understand the advantages/disadvantages of endoscopy vs. open skull base surgery
- Understand how prospective vs. retrospective trials are conducted
- Understand the disadvantages and advantages of both types of medical evidence
- Understand the basis of scientific projects progression
- Understand how to evaluate data from basic sciences

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 through the joint sponsorship of the AANS and the American Academy of Neurological Surgery. The AANS is accredited by the ACCME to provide continuing medical education for physicians.

Designation Statement

The AANS designates this live activity for a maximum of 13.75 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Intended Audience/Background Requirement

The scientific program presented is intended for neurosurgeons either in training or in active practice.

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Speakers with no potential conflict of interest to declare:

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Boockvar, John
Brem, Steven
Burchiel, Kim
Cahill, Daniel
Chalif, David
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Michael Tymianski	Other financial or material support	President and CEO of NoNo Inc., a biotech company founded to develop PSD95 inhibitors discovered in my academic lab.

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

SCIENTIFIC PROGRAM AGENDA 2012

THURSDAY OCTOBER 18

730- 800 Point-counterpoint session: Cerebrovascular
Robert Spetzler: When is endovascular therapy appropriate in aneurysm treatment?
Charles Prestigiacomo: When do I reserve standard craniotomy for aneurysm treatment?

Prospective Clinical Trials in Neurosurgery (800-951) (Moderator: Stephen Haines)

800-810	John Boockvar	Phase I trial of superselective intraarterial cerebral infusion of Cetuximab
812-822	Michael G. Fehlings	The AOSpine Multicenter, International Cervical Spondylotic Myelopathy Study: One Year Outcomes in 486 patients
824-834	James Markert	Phase I Clinical Trial of Intratumoral Reovirus Infusion for the Treatment of Recurrent Malignant Gliomas in Adults
836-846	Ali Rezai	A randomized sham-controlled trial of DBS of the Ventral Capsule/Ventral Striatum (VC/VS) for Treatment-Resistant Depression
848-858	Theodore H. Schwartz	Phase I/II study of cesium-131 brachytherapy following surgical resection for newly diagnosed brain metastases
900-910	Justin S. Smith	The Schwab-SRS Adult Spinal Deformity Classification: Assessment and Clinical Correlations Based On A Prospective Operative and Non-Operative Cohort
912-922	Gary K. Steinberg	A Novel Phase 1/2A Study (Safety and Efficacy) of Intraparenchymal Transplantation of Human Modified Bone Marrow Derived Cells in Patients with Stable Ischemic Stroke
924-934	Michael Tymianski	Evaluating Neuroprotection in Aneurysm Coiling Therapy (ENACT) trial: A test of neuroprotection for procedurally induced ischaemic stroke using NA-
936-946	Miguel A. Arraez-Sanchez	Current trends in the management of Normal Pressure Hydrocephalus. Results of the Spanish Cooperative Study

946-951 **Discussion (Stephen Haines To Lead)**

941-1010 **BREAK**

Brain metastases: survival and outcomes (10:10-10:49, Moderator, Jacques Morcos)

1010-1020	Douglas Kondziolka	How accurately can we predict the survival of patients with cancer?
1022-1032	Andrew E. Sloan	A Normogram for Individualized Estimates of Survival Outcomes for Patients with Brain Metastasis1022
1034-1044	Jason Sheehan	Impact of Triple negative phenotype on the prognosis of patients with breast cancer metastases undergoing SRS

1044-1049 Discussion (Jacques Morcos to lead)

New avenues in DBS surgery (1049- 1128) (moderator: Schulder)

1049-1059	Francisco A. Ponce	DBS under general anesthesia without neurophysiology: Initial experience and comparison to the standard technique
1101-1111	Kim J. Burchiel	Image-guided DBS electrode surgery without microelectrode recording: accuracy and costs of electrode placement using Nexframe and the Ceretom intraoperative CT scanner
1113-1123	Kendall H. Lee	Mechanism studies using fMRI, WINCS, and MINCS: towards neural engineering electrochemical feed back DBS

1123-1128 Discussion (Michael Schulder to lead)

The value of surgical volume and experience (1128-1207) (moderator: Peter Jannetta)

1128-1138	Paul Gardner	Endoscopic endonasal approach for resection of skull base chordomas: outcomes and learning curve
1140-1150	Michael Lawton	Current Management of MCA aneurysms; results with a "clip first" approach
1152-1202	Michael J Link	Use of Supramaximal Stimulation to Predict Facial Nerve Outcomes Following Vestibular Schwannoma Microsurgery: Results from a Decade of Experience

1202-1207 Discussion (Peter Jannetta to lead)

1207-1227 Academy Winners

Daniel A. Orringer –Honorable Mention: Label-Free Detection of Microscopic Tumor Boundaries Using Stimulated Raman Scattering Microscopy. (Dr. Orringer will not present)

Derek G. Southwell: Intrinsically Determined Cell Death Of Developing Cortical Interneurons

FRIDAY OCTOBER 19

730- 800 Point-counterpoint session: Skull base
William Couldwell: When is standard craniotomy appropriate for skull base lesions?
Anil Nanda: When do I prefer endoscopic endonasal approaches to skull base lesions?

Research in Neurosurgery I (800-1003) (Moderator: Robert Friedlander)

800-810	Issam A. Awad	Quantitative iron burden as a biomarker of cumulative hemorrhages in cerebral cavernous malformation: studies in mouse and man
812-822	Yutaka Mine	Grafted human neural stem cells enhance several steps of endogenous neurogenesis and improve behavioral recovery after middle cerebral artery occlusion in T cell-deficient rats.
824-834	Shenandoah Robinson,	Erythropoietin promotes restoration of inhibitory circuit development after transient prenatal global hypoxia-ischemia
836-846	J. Marc Simard	Update on Glyburide in Stroke and Malignant Cerebral Edema
848-858	Joerg-Christian Tonn	Hot-spots in dynamic 18FET-PET are associated with unfavorable outcome in patients with suspected WHO grade II glioma
900-910	Franco DeMonte	Meningioma gene expression profiling as a potential guide to postoperative patient management
912-922	Raj Narayan	The next generation intracranial monitor
924-934	Daniel W. Fults	Functional Genomics Identifies Drivers of Medulloblastoma Dissemination
936-946	Robert E. Gross	Electrical and optogenetic neuromodulation of septohippocampal oscillations for the treatment of epilepsy
948-958	Adel M. Malek	Thin-Walled Dome Regions Co-Localize with Low Hemodynamic Wall Shear Stress in Unruptured Cerebral Aneurysms

958-1003 DISCUSSION (Robert Friedlander to lead)

1003-1030- BREAK

The socioeconomics of NS (1030- 1109) (moderator: Gene Barnett)

1030-1040	Anthony Asher	The national neurosurgery quality and outcomes database (N^2QOD): review of founding site performance, regional challenges to implementation and strategies for streamlining national data collection requirements
1042-1052	Christopher S. Ogilvy	Integration of Three Separate Departments into a Combined Neuroendovascular Unit, Facilitated by a Collaborative Financial Model
1054-1104	Charles Prestigiacomo	Improving medical student recruitment into neurosurgery: a multi-tiered strategy

1104-1109 Discussion (Gene Barnett to lead)

Special Lecture
1109-1145 Richard Delaney (to be introduced by Ralph Dacey)

1145-1150 Presentation of President (James Drake)

1150-1220 Presidential Address
President James Rutka: William S. Keith and the Founder Effect

SATURDAY OCTOBER 20

Research in neurosurgery 2 (730-952) (moderator: Loch McDonald)

730-740	Amgad Hanna	Peripheral Nerve Grafts and Chondroitinase ABC Application Improves Functional Recovery after Complete Spinal Cord Transection
742-752	Amy Heimberger	MicroRNA as a novel immunotherapeutic strategy to reverse glioma-mediated immune suppression and enhance anti-tumor clearance
754-804	Corey Raffel	Treatment of Medulloblastoma with Measles Virus Encoding the Thyroidal Sodium-Iodine Symporter Plus Radio-Iodine

806-816	Fredric Meyer	Preoperative assessment of meningioma stiffness by magnetic resonance Elastography
818-828	Roger Härtl	Tissue Engineered Intervertebral Discs: An *in vivo* study
830-840	Rajiv Midha	Skin-derived precursor Schwann cell therapy improves behavioural outcome for both immediate and delayed nerve repair
842-852	David S. Baskin	Novel Nanovector Nanosyringe Delivered Drug Pump Inhibitors (CERBERUS) Potentiate the Action of Nanovector-Delivered Chemotherapy (HADES) in Cultured Primary Human Glioblastoma
854-904	Peter Nakaji	Rapid and specific diagnosis of astrocytic tumors using immediate ex vivo SRS101 confocal microscopy
906-916	Robert J. Dempsey	Cytokines at the cross roads of brain injury and repair: Galectin-3, a potential target for enhancing injury repair and recovery following ischemic stroke.
918-928	Donald M. O'Rourke	Advanced MRI Imaging of the Epidermal Growth Factor Receptor (EGFR): From Noninvasive Detection to Prediction of Glioblastoma Recurrence
930-940	Matt Howard	Preliminary studies of a human spinal cord stimulator
942-952	Mitch Berger	The adult bay area GLIOMA study: a followup

954- 1015 BREAK

<u>Retrospective trials and surgical experience 2 (1017- 1115) (moderator: E. Antonio Chiocca))</u>

1017-1027	James Drake	Patient-specific modeling for pediatric craniofacial reconstruction
1029-1039	Graham Fieggen	Long-term outcome of Selective Dorsal Rhizotomy for Cerebral Palsy spasticity
1041-1051	Michael W. McDermott	Volume-Staged Gamma Knife Radiosurgery for Large Arteriovenous Malformations

Research in Neurosurgery (1117-1225) (Bermans Iskandar)

1105-1115	Fred Lang	Exosomes from Glioma-Associated Mesenchymal Stem Cells Modulate the Proliferation of Glioma Stem Cells
1117-1127	Steven Brem	Senescence-Associated-Gene Signature Identifies Genes Linked to Age, Prognosis, and Progression of Human Gliomas
1129-1139	Daniel Cahill	Prolonged survival in patients undergoing aggressive surgery for IDH1-mutant malignant astrocytoma
1141-1151	Rafael J. Tamargo	Enhanced aneurysm formation in pro-inflammatory, transgenic haptoglobin 2-2 mice
1153-1203	Aaron Cohen-Gadol	A New Method for Intraoperative Fluorescence-Guided Resection of High-Grade Gliomas
1205-1215	Howard Riina	The eCLIPS Self Expanding Endoluminal Device For the Treatment of Bifurcation Aneurysms: Preliminary Animal Model Study Results.

SCIENTIFIC PROGRAM

THURSDAY, OCTOBER 18

8:00 – 8:10 PHASE I TRIAL OF SUPERSELECTIVE INTRAARTERIAL CEREBRAL INFUSION OF CETUXIMAB

***John Boockvar, MD**, Weill Cornell Medical College, New York, New York*

INTRODUCTION: High-grade malignant brain tumors are the most common and most aggressive adult brain tumors with median overall survival durations of only 9-12 months for glioblastoma multiforme (GBM), and 3-4 years for anaplastic astrocytoma (AA). All patients experience a recurrence after first-line therapy, so improvements in both first-line and salvage therapy are critical to enhancing quality-of-life and prolonging survival. A significant number of gliomas overexpress Epidermal Growth Factor Receptor (EGFR), so this is thought to be an important therapeutic target. A phase I clinical research trial was designed to test the hypothesis that Cetuximab, a chimeric monoclonal antibody that inhibits EGFR, can be safely used by direct intracranial superselective intraarterial cerebral infusion (SIACI) to ultimately enhance survival of patients with relapsed/refractory GBM/AA. By achieving the aims of this study we will determine the toxicity profile and maximum tolerated dose (MTD) of SIACI Cetuximab.

METHODS: EGFR overexpression or amplification was analyzed via fluorescence in situ hybridization (FISH) or immunohistochemistry. Subjects with recurrent or relapsing high grade glioma with EGFR expression or amplification were treated with mannitol followed by a single SIACI of Cetuximab. Dose was started at 100mg/m^2 with intent to undergo dose escalation to 500 mg/m^2 to determine maximal tolerated dose.

RESULTS: Twelve patients were treated at dose of 100 mg/m^2 or 200 mg/m^2 and maximal tolerated dose was determined to be 200 mg/m^2. Tolerable rash was seen in 3 patients, anaphylaxis in 1 patient, seizure in 2 patients, and cerebral edema in 1 patient.

CONCLUSION: Cetuximab is safely tolerated through intraarterial delivery up to a dose of 200 mg/m^2. A Phase II trial is currently underway to determine the efficacy of SIACI of cetuximab.

THURSDAY, OCTOBER 18

8:12 – 8:22 THE AOSPINE MULTICENTER, INTERNATIONAL CERVICAL SPONDYLOTIC MYELOPATHY STUDY: ONE YEAR OUTCOMES IN 486 PATIENTS

Michael Fehlings, MD PhD[1], *Branko Kopjar, MD PhD[2], Shashank Kale, MD[3], Helton Delfino, MD[4], Giuseppe Barbagallo, MD[5], Ronald Bartels, MD[6], Qiang Zhou, MD[7], Paul Arnold, MD[8], Mehmet Zileli, MD[9], Gamaliel Tan, MD[10], Osmar Moraes, MD[11], Yasutsugu Yukawa, MD[12], Manuel Alvarado, MD[13], Massimo Scerrati, MD[14], Tomoaki Toyone. MD[15], Masato Tanaka, MD[16], Ciaran Bolger, MD[17]*

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INTRODUCTION: Although cervical spondylotic myelopathy (CSM) is the commonest cause of spinal cord impairment globally, little objective prospective data exists on the outcomes of surgical intervention and the international variations in clinical presentation and management.

METHODS: A total of 486 patients with clinically symptomatic CSM were enrolled in a prospective multicenter, international study which was undertaken at 16 sites in Europe, Asia, North and South America. Outcome measures included the modified Japanese Orthopaedic Assessment scale (mJOA), Nurick Score, Neck Disability Index (NDI), short form 36v2, and complications. Data were analyzed using multivariate techniques (SAS 9.2 PROC MIXED) adjusting for baseline differences in patient populations (age, gender, surgical approach, number of spinal levels and baseline outcome parameter value).

RESULTS: A total of 389 patients have completed one year follow-up to date. There were 35% females with an average age of 56.16 yrs (SD 12.44). Patients underwent anterior (58%), posterior (40%) or circumferential (2%) surgery. There were significant differences in the age at presentation and baseline neurological status among the regions, with Asian and Latin American patients being noticeably younger. There has been a significant ($P < 0.001$) improvement from baseline values to 12 months in all outcome parameters. The mJOA improved from 12.5±2.8 at baseline to 14.9±2.6 at 12 months. The NDI improved from 38.0±20.2 at baseline to 24.7±18.7 at 12 months. The Nurick improved from 3.3±1.2 at baseline to 1.9±1.5 at 12 months. The SF36 PCS improved from 35.2±8.5 at baseline to 43.5±10.2 at 12 months. The SF36 MCS improved from 38.8±9.9at baseline to 46.5±10.7 at 12 months. Of note, the amount of improvement varied across the regions with patients from Asia-Pacific and Latin America having generally better outcomes than those from North America and Europe.

CONCLUSION: This large prospective international clinical study shows that surgical treatment for CSM is associated with significant improvements in generic and patient-specific outcome measures at one year. Interestingly, there are significant variations in clinical presentation and in patient perceptions of improvement that are currently being examined in detail.

THURSDAY, OCTOBER 18

8:24 – 8:34 PHASE I CLINICAL TRIAL OF INTRATUMORAL REOVIRUS INFUSION FOR THE TREATMENT OF RECURRENT MALIGNANT GLIOMAS IN ADULTS

*Kimberly P. Kicielinski, MD—UAB, **James M. Markert, MD MPH**--UAB, E. Antonio Chiocca, MD PhD, FAANS—Ohio State University→BWH, John S. Yu, MD—Cedars Sinai Medical Center, George M. Gill, MD—Oncolytics, Matt Coffey, PhD--Oncolytics*

INTRODUCTION: Reovirus is an RNA virus shown to have in vivo activity in malignant glioma (MG) in preclinical studies. A single Phase I trial of one-time intratumoral reovirus inoculation in patients with MG showed the virus to be well tolerated, without dose-limiting toxicity (DLT). The goal of this multicenter Phase I study was to determine the DLT and maximum tolerated dose (MTD), as well as the effects of intratumoral reovirus infusion in patients with recurrent MG. The response rate of the targeted lesions was also evaluated as a secondary endpoint.

METHODS: Patients were adults with a first, second, or third recurrence of a histologically confirmed supratentorial MG with a Karnofsky Performance score (KPS) of ≥60, and who had undergone. prior surgery and radiation. A total of 15 patients were enrolled in a classic 3x3 dose escalation scheme with three patients treated at each of the following tissue culture infectious dose 50 (TCID50) doses: 1 x 108, 3 x 108, 1 x 109, 3 x 109, and 1 x 1010. Each patient received a 72-hour infusion via one to four catheters implanted intraoperatively at the enhancing border of target lesions. Patients underwent examinations of neurological and functional performance as well as MRI scans at baseline, time of discharge from infusion, and at 4, 8, 12, 16, and 24 weeks post infusion.

RESULTS: The patients treated had a median age of 51.5 years, a median enrollment KPS of 90, and there were 10 males, and 14 Caucasians. There was one grade III adverse event (AE, convulsions), felt to be possibly related to treatment, but no grade IV AEs graded probably or definitely related to treatment. Twelve patients had tumor progression, two had STABLE disease, and one had a partial response. Median survival was 140 days (range, 97 – 989), and one patient was still alive more than 16 months post treatment. Median time to progression (TTP) was 61 days (range, 29 – 150 days). DLTs were not identified and a MTD was not reached.

CONCLUSIONS: A 72-hour intratumoral infusion of genetically unmodified reovirus was well tolerated at the above doses in patients with recurrent MG.

THURSDAY, OCTOBER 18

8:36 – 8:46 A RANDOMIZED SHAM-CONTROLLED TRIAL OF DBS OF THE VENTRAL CAPSULE/VENTRAL STRIATUM (VC/VS) FOR TREATMENT-RESISTANT DEPRESSION

Ali Rezai, *Gordon Baltuch, Douglas Kondziolka, Andre Machado, and Emad Eskandar*

INTRODUCTION: Deep Brain Stimulation (DBS) of the ventral capsule/ventral striatum (VC/VS) for Treatment-Resistant Depression (TRD) has been investigated in previous open-label studies with promising results of 60% responders. We now report the outcomes of a randomized, prospective, double-blind controlled multi-center feasibility trial of VC/VS DBS for TRD.

METHODS: Thirty subjects across five centers with severe, chronic and intractable TRD underwent stereotactic bilateral implantation of Medtronic 3391 leads in the VC/VS. Nominal target coordinates were 5-10 mm from midline, 0-5 mm anterior to AC, and 1-4 mm ventral to AC. The leads were connected to bilateral Kinetra® pulse generators. Active or sham stimulation was delivered during a 4- month blinded phase, followed by an open stimulation continuation phase. The primary outcome measure was proportion of responders ($\geq$50% improvement on Montgomery-Asberg Depression Rating Scale (MADRS)) at the 4-month endpoint.

RESULTS: Of 30 subjects randomized (mean current depressive episode 11.4 years; mean baseline MADRS 36.7 $\pm$ 4.3), 29 completed the blinded phase. 3/15 subjects (20%) responded to active and 2/14 (14.3%) responded to sham stimulation. Mean MADRS reduction was 19.6% for active and 24.6% for sham stimulation ($p=0.34$). Complications included 4 infections, 3 lead revision, 1 asymptomatic hemorrhage, and reversible stimulation related events. Active contact location did not appear to correlate with clinical outcomes.

DISCUSSION: Study variables of patient selection, blinding, surgical targeting, and programming will be discussed in the context of the blinded and longer-term outcomes.

CONCLUSIONS: DBS of the VC/VS for TRD was not superior to sham stimulation in a 4-month randomized, controlled trial. However, improvements in some subjects were noted in the continuation open label phase.

THURSDAY, OCTOBER 18

8:48 – 8:58 PHASE I/II STUDY OF CESIUM-131 BRACHYTHERAPY FOLLOWING SURGICAL RESECTION FOR NEWLY DIAGNOSED BRAIN METASTASES

A.Gabriella Wernicke, MD MSc; Luke Peng, MD; Menachem Yondorf, BA; Dattatreyudu Nori, MD; KS Clifford Chao, MD; Susan Pannullo, MD; Philip E. Steig PhD, MD, John A. Boockvar, MD; Theodore ***H. Schwartz, MD***

INTRODUCTION: Resected brain metastases have a high rate of local recurrence without adjuvant therapy. Adjuvant whole brain radiotherapy (WBRT) provides local and distant control >90% but is associated with acute and long-term toxicities. Stereotactic radiosurgical (SRS) targeting of an irregularly shaped cavity can be challenging and requires a delayed second therapeutic session, which permits interval tumor proliferation. Intra-operative permanent Cs-131brachytherapy (BT) implants can be performed at the time of surgery, thereby avoiding any additional therapy providing cost savings.

METHODS: Patients with a newly diagnosed metastasis to the brain were prospectively enrolled in an IRB-approved study between 2010 and 2012. After maximal surgical resection, the cavity was lined with permanent Cs-131 stranded seeds. Prescription dose was 80Gy at 5mm depth from the resection cavity. A post-implant CT scan was performed within 48 hours to determine dose distribution. End points were local freedom from progression (FFP), distant metastases FFP, median survival, overall survival (OS), and toxicity. A case control study of cost comparing surgery(S)+BT, SRS, surgery +WBRT and WBRT alone was performed.

RESULTS: 24 patients were enrolled. Median follow-up was 9.5 months (range, 1.1 – 17.8 months). Median age was 65 years (range, 45-84 years). Median volume of resected tumor was 10.3 cc (range, 1.8 – 87.1 cc). Histology included lung (16), breast (2), kidney (2), melanoma (2), colon (1), and cervix (1). Median number of seeds employed was 12 (range, 4-35) with median activity per seed of 3.8 mCi (range, 3.3-4.8 mCi) and total activity of 46.9 mCi (range, 15.3-130.6 mCi). The 1-year resection cavity FFP was 100%. Exposure to the surgeon was < 0.2mRem/hr. There were 9 distant recurrences, resulting in 1-year distant metastases FFP = 36.5% (95% CI = 7.8-67.1%). There was a total of 10 deaths rendering a median OS = 12.4 months and 1-year OS = 59.1% (95% CI = 27.3-80.8%). Complications included CSF leak (1) and seizure (1). There were no infections and no radiation necrosis. The direct hospital costs of treatments per patient with surgery+BT ($19,271) was significantly lower than, S+WBRT (10fx; $30,46), SRS ($44,219), WBRT (15fx; $24,283;P<0.0001).

CONCLUSIONS: Post-resection intracavitary Cs-131 BT is a safe, well tolerated, technique for achieving local control for newly diagnosed brain metastases during a single therapeutic session. Dosage is delivered maximally and uniquely to the residual microscopic disease and not to an empty cavity or surrounding normal brain. High local control and low radiation necrosis rates make this an attractive therapy. Further trials in larger patient groups are warranted.

THURSDAY, OCTOBER 18

9:00-9:10 THE SCHWAB-SRS ADULT SPINAL DEFORMITY CLASSIFICATION: ASSESSMENT AND CLINICAL CORRELATIONS BASED ON A PROSPECTIVE OPERATIVE AND NON-OPERATIVE COHORT

Justin S. Smith, MD, PhD *(Neurosurgery, University of Virginia), Christopher I. Shaffrey, MD (Neurosurgery, University of Virginia), Christopher P. Ames, MD (Neurosurgery, University of California, San Francisco), Kai-Ming G. Fu, MD, PhD (Neurosurgery, Weill Cornell Medical Center), Praveen V. Mummaneni, MD (Neurosurgery, University of California, San Francisco), Frank J. Schwab, MD (Orthopaedic Surgery, NYU Hospital for Joint Diseases), Virginie Lafage, PhD (Orthopaedic Surgery, NYU Hospital for Joint Diseases), Shay Bess, MD (Orthopaedic Surgery, Rocky Mountain Hospital for Children), International Spine Study Group Foundation (ISSGF; Denver, CO)*

INTRODUCTION: A recent study of elderly volunteers reported a prevalence of adult spinal deformity (ASD) of 68%. As medical advances continue to extend life expectancy and population demographics of the population expand the numbers of elderly to unprecedented levels, the impact of ASD will continue to increase. ASD has traditionally been described using pediatric classification systems that neglect to account for sagittal spino-pelvic alignment parameters that are known to strongly correlate with health-related quality of life (HRQOL) in ASD. The Schwab-SRS Classification of ASD is a recently developed system that provides a common language for the complex pathology of ASD. The inter- and intra-observer reliability of this classification has been reported; however, the clinical relevance, including correlation with treatment approach has not been demonstrated. Our objective was to assess whether the Schwab-SRS classification correlates with disability and the decision of whether to pursue operative (OP) or nonoperative (NONOP) treatment.
METHODOLOGY: This study was based on a multicenter, prospective analysis of consecutive ASD patients. Inclusion criteria included: age ≥18 yrs and spinal deformity (scoliosis ≥20°, sagittal vertical axis ≥5cm, pelvic tilt ≥25° or thoracic kyphosis >60°). Patients were classified based on the Schwab-SRS classification, which includes curve type (thoracic only, thoracolumbar/lumbar only, double curve, or primary sagittal deformity) and 3 sagittal modifiers, each with 3 grades (normal, moderately poor and poor). These modifiers are sagittal vertical axis (<4, 4-9 or >9cm), pelvic tilt (<20, 20-30 or >30□), and pelvic incidence/lumbar lordosis mismatch (<10, 10-20 or >20□). Differences in demographics, HRQOL, and classification curve type/modifier distribution between OP and NONOP patients were evaluated. HRQOL was assessed based on the Oswestry Disability Index (ODI), Scoliosis Research Society-22 (SRS-22), and the Short Form-36 (SF-36).
RESULTS: 757 patients (mean age 53 yrs, range 18-85) met inclusion criteria. OP patients (n=311) were older (mean age 56 vs 51 yrs), had greater body mass index (27.7 vs 25.7), had more previous surgery (45% vs 19%), and had greater Charlson comorbidity index (1.1 vs 0.85) compared with NONOP (n=446) patients, respectively ($p<0.05$). OP patients had worse baseline HRQOL scores on all surveys compared with NONOP patients ($p<0.05$). OP and NONOP patients had similar coronal alignment ($p<0.05$), but OP patients had worse sagittal spino-pelvic alignment for all measures compared with NONOP patients, except for cervical lordosis, thoracic kyphosis and pelvic incidence. OP patients had a greater percentage of pure sagittal deformity classification (23% vs 14%; $p<0.05$) and had worse grades for all modifier categories: pelvic tilt (26% vs 16%), pelvic incidence-lumbar lordosis mismatch (37% vs 21%) and global sagittal alignment (29% vs 9%), OP vs NONOP, respectively ($p<0.05$).
CONCLUSION: Prospective analysis of OP vs NONOP treated ASD patients demonstrated that OP patients were older, had more co-morbidities, greater disability and worse sagittal spino-pelvic alignment as defined by the Schwab-SRS Classification curve type and sagittal modifiers. This study demonstrates that the Schwab-SRS Classification is descriptive, correlates with HRQOL scores, and corresponds to treatment preference for ASD.

THURSDAY, OCTOBER 18

9:12-9:22 A NOVEL PHASE 1/2A STUDY (SAFETY AND EFFICACY) OF INTRAPARENCHYMAL TRANSPLANTATION OF HUMAN MODIFIED BONE MARROW DERIVED CELLS IN PATIENTS WITH STABLE ISCHEMIC STROKE

Gary K. Steinberg, MD, PhD*, Douglas Kondziolka, MD†, Neil E. Schwartz, MD, PhD*, Lawrence Wechsler, MD†, Maria L. Coburn, BA*, Julia B. Billigen, RN†, Michael McGrogan, PhD#, Keita Mori, MBA#, Ernest W. Yankee, PhD#*

**Departments of Neurosurgery and Neurology and Stanford Stroke Center, Stanford University, Stanford CA, †Departments of Neurological Surgery and Neurology, University of Pittsburgh, Pittsburgh, PA, #SanBio, Inc, Mountain View, CA*

INTRODUCTION: No treatment currently exists to restore lost brain function after stroke. Animal studies demonstrate that intraparenchymal brain transplantation of SB623, a human bone marrow derived stromal cell with transient transfection of Notch-1 gene, one month after experimental stroke can improve neurologic outcome. The likely mechanisms are secretion of trophic factors and immunomodulation that enhance endogenous plasticity and recovery. This clinical study is the first North American trial of intraparenchymal bone marrow-derived cell therapy for chronic stroke patients.

METHODS: This is a two center open label safety and efficacy study. Eighteen patients will be treated at Stanford University and the University of Pittsburgh with a dose escalation paradigm of 2.5M, 5.0M, and 10.0 M cells. Stereotactic intraparenchymal transplantation is targeted to the subcortical peri-infarct area using three tracks with five cell deposits/track through a burr hole and utilizing a specially designed cell transplantation needle. Inclusion criteria include age 18-75 years, subcortical middle cerebral artery (MCA) ischemic stroke (with or without cortical involvement), 6-36 months post-stroke, modified Rankin Scale (mRS) three or four and NIHSS >7. Safety parameters are World Health Organization (WHO) toxicity scales and magnetic resonance imaging (MRI) brain scans. Primary efficacy is European Stroke Scale (ESS) at six months and secondary efficacy measures include ESS, National Institutes of Health Stroke Scale (NIHSS), Fugl-Meyer, mRS, cognitive scores, and fluorodeoxyglucose-positron emission tomography (FDG-PET) at multiple time points up to two years.

RESULTS: As of 6/15/12, eight patients have been treated (six with 2.5M cells; two with 5M cells). Transplantation was performed under local anesthesia and mild sedation. Patients were discharged home on the first post-operative day. Follow-up is currently 1-9 months; we will be 4-13 months at the time of this presentation. No adverse events related to the cells have occurred. Clinical results will be discussed.

CONCLUSIONS: Intraparenchymal transplantation of human modified bone marrow-derived stromal cells in chronic stroke patients is safe, feasible, and may have therapeutic potential. Larger studies will be initiated to further assess clinical efficacy.

THURSDAY, OCTOBER 18

9:24 – 9:34 THE EVALUATING NEUROPROTECTION IN ANEURYSM COILING THERAPY (ENACT) TRIAL: A TEST OF NEUROPROTECTION FOR PROCEDURALLY INDUCED ISCHEMIC STROKE USING NA-1

Michael Tymianski, ***for the ENACT trial Investigators****

**The following Investigators participated in the ENACT trial: Steering Committee: Roberta Anderson, Ottawa, Canada (Chair), Michael D. Hill, Calgary, Canada (Principal Investigator), Michael Tymianski, Toronto, Canada (Sponsor representative), Peter S. Lu, Sunnyvale, CA (Cosponsor representative), Renee Martin, Charleston, SC (Lead Statistician); Data and safety monitoring board. Gary Redekop, Vancouver, Canada (Chair), Gord Gubitz, Halifax, Canada, Dean Johnston, Halifax, Canada, Randomization: Wenle Zhao, Charleston, SC; Plasma Concentration Analysis: Charles River, Senneville, Canada; Histamine Analysis: Gamma Dynacare, Brampton, Canada; Clinical Monitoring: NoNO Inc., Ottawa, Canada, PRC, Inc, Calgary, Canada. and Study Hall Inc., Hudson, MA; Drug Manufacturing: The University of Iowa Pharmaceuticals, Iowa City, Iowa; Data Management: BioClinica, Audubon, PA and Hotchkiss Brain Institute – Clinical Research Unit, Calgary, Canada. Medical Monitors: Michael D. Hill (all sites other than Calgary), Michael Tymianski (Calgary Site). MRI Assessment: David Mikulis, Toronto, ON. Julien Poublanc, Toronto, ON. Timo Krings, Toronto, ON. Mayank Goyal, Calgary, AB. Andrew M. Demchuck, Calgary, AB. Clinical Sites: Calgary, AB – John H. Wong. Edmonton, AB – Mike Chow. Saskatoon, SK – Michael E. Kelly. Toronto, ON (St Michael Hospital) – R. Loch MacDonald. Toronto, ON (Toronto Western Hospital) – Frank L. Silver, Karel terBrugge. London, ON – Melford Boulton. Ottawa, ON – Cheemun Lum. Hamilton, ON – Thorsteinn Gunnarsson. Quebec, QC – Genevieve Milot. Halifax, NS – Ian Fleetwood. Phoenix, AZ – Cameron McDougall. Palo Alto, CA – Robert Dodd. Portland, OR – Wayne Clark.*

BACKGROUND: Despite many previous attempts, the salvage of ischemic human brain tissue by a neuroprotectant has never been demonstrated. We used a novel approach to explore whether NA-1 [Tat-NR2B9c], a PSD95 inhibitor, could reduce ischemic brain damage in humans. NA-1 is a promising agent that we have shown to reduce ischemic brain damage in old-world primates in several clinically relevant scenarios including middle cerebral artery occlusion and in embolic strokes (Cook et al., Nature, 2012; Cook et al., Sci Trans Med 2012 [Accepted]). The ENACT trial (Evaluating Neuroprotection in Aneurysm Coiling Therapy trial; ClinicalTrials.gov number, NCT00728182) was designed to test whether or not it is feasible to achieve neuroprotection in the human brain.

METHODS: Subjects undergoing endovascular repair of ruptured or unruptured intracranial aneurysms have a high incidence of small, procedurally-induced, ischemic strokes that are detectable by MR imaging. We enrolled 185 such subjects in a multi-center randomized, double blinded trial conducted in 14 sites across Canada and the USA, to receive a single intravenous infusion of NA-1 or Saline control at the termination of the endovascular procedure. Ischemic damage was assessed using DWI and FLAIR MR imaging, and clinical outcome was assessed using neurological evaluations and a cognitive battery throughout a 30-day study period.

RESULTS: Subject demographics, medical risks, adverse events and procedure details were balanced between the groups. There were no safety concerns attributable to NA-1. Subjects who received NA-1 (n = 92) sustained fewer ischemic infarcts as gauged by DW (adjusted IRR = 0.53 CI95 0.38-0.74) and FLAIR (adjusted IRR = 0.59 CI95 0.42-0.83) MR imaging. Those with uncomplicated procedures (strokes < 10ml) also exhibited reduced infarct volumes by DWI and FLAIR.

Among subjects with ruptured aneurysms (n = 37), NA-1 treatment reduced the number and volume of strokes by all MRI criteria and resulted in improved neurological outcome (NIHSS 0-1 risk difference 31.6%, p = 0.020), suggesting that NA-1 improves the brain injury suffered following a SAH.

CONCLUSIONS: In subjects with small, peri-procedural ischemic strokes, treatment with a single intravenous infusion of NA-1 post-procedure reduces structural ischemic damage. Tissue neuroprotection in aged humans is feasible. These data support further clinical trials of the neuroprotectant NA-1.

THURSDAY, OCTOBER 18

9:36-9:46 CURRENT TRENDS IN THE MANAGEMENT OF NORMAL PRESSURE HYDROCEPHALUS. RESULTS OF THE SPANISH COOPERATIVE STUDY

Miguel A. Arraez, MD, PhD. *Chairman, Dept of Neurosurgery. Carlos Haya University Hospital. Malaga. Spain*

BACKGROUND

The management of Normal Pressure Hydrocephalus is still under debate. Several issues as prognostic factors, role of hydrodynamic tests and the best shunt option have not been still clarified. This presentation deals with the current aspects of the NPH and also presents the preliminary results of the Spanish Cooperative Study about the efficacy and safety of the gravity-assisted shunt systems for the treatment of this condition.

MATERIAL AND METHODS

Prospective study on 136 patients belonging to 12 neurosurgical Spanish centers with diagnosis of Normal Pressure Hydrocephalus with one or several symptoms of the Hakim triad, Evans index over 0,3 and positivity of the tap test and / or lumbar infusion test and / or abnormal ICP recording. Follow-up: 12 months in 33 patients; 6 months in 62 patients. End-points: Clinical outcome according the NPH scale, daily life activities (modified Rankin scale), radiological changes (Evans index among other parameters), shunt related complications and establishment of clinical (neurological and systemic), radiological and hydrodynamic prognostic factors.

RESULTS

There was a clear improvement in the clinical scales at three and six months after shunt insertion (gait: 3.2□1.0 vs 2.5□1.1 vs 2.2□1.1; cognitive function: 2.1□0.9 vs 1.8□0.9 vs 1.7□0.9; sphincter disturbance: 2.9□1.2 vs 2.5□1.3 vs 2.4□1.3; $p<0.05$) and also in the global scale (8.2□2.2 vs 6.8□2.7 vs 6.3□2.7; $p<0.05$). There was no difference after comparison of improvement at six and 12 months (gait: 2.1□1.0; cognitive function: 1.6□0.9; sphincter disturbance: 2.3□1.2; global scale: 6.0□2.6; $p>0.05$). The only prognostic parameter found was the rout value (lumbar infusion test). There was a clear reduction in the ventricular size (Evans index) at three months (basal, 0.49□0.54 vs 0.35□0.042; $p<0.05$). There were three recorded shunt-related postoperative complications (subdural hematoma, intraparenchymal hemorrhage and catheter malfunction).

CONCLUSIONS

Gravity-assisted shunts seems to be effective and safe for the treatment of patients with Normal Pressure Hydrocephalus with high rate of improvement and low shunt-related morbidity. The improvement seems to be maintained at least in the first

THURSDAY, OCTOBER 180

10:10-10:20 HOW ACCURATELY CAN WE PREDICT THE SURVIVAL OF PATIENTS WITH CANCER?

Douglas Kondziolka, MD[1]*, Phillip V. Parry, MD*[1]*, L. Dade Lunsford, MD*[1]*, John C. Flickinger, MD*[2]*, Susan Rakfal, MD*[2]*, Yoshio Arai, MD*[2]*, Jay S. Loeffler, MD*[5]*, Stephen Rush, MD*[6]*, Jonathan Knisely, MD*[7]*, Jason Sheehan, MD*[8]*, William Friedman, MD*[9]*, Ahmad Tarhini MD*[3]*, Lanie Francis MD*[3]*, Manmeet Ahluwalia, MD*[10]*, Mark E. Linskey, MD*[4]*, Paul Sperduto, MD*[11]*, and Roger Stupp, MD*[12]

INTRODUCTION: For cancer patients estimated survival time is crucial for clinicians, patients, families, and payors. To provide appropriate and cost effective care, various data sources are tapped in order to provide rational, reliable, and reproducible estimates. Using specific data we asked 14 medical, radiation, or surgical oncologists to predict the survival of patients with cancer metastatic to the brain.

METHODS: During a 2 year interval we prospectively estimated survival in 150 consecutive cancer patients (median age =62 ,range, 33-84) with brain metastases undergoing radiosurgery. We recorded the cancer type (e.g. lung, renal, melanoma, breast), the number of brain metastases, and activity of extracranial disease status, which was graded as either a) none; b) minimal; c) symptomatic; d) diffuse; or e) cachectic. We also recorded the Karnofsky Performance Score (KPS), exposure to prior whole brain radiation therapy (WBRT), and whether the patient had synchronous or metachronous presentation of their brain disease. The brain disease was rated as a) asymptomatic; b) causing seizures only; c) causing headache only, or d) associated with a neurological deficit. We provided the Recursive Partitioning Analysis (RPA) score (grades 1-3). Finally, we asked each physician to provide a prediction of expected survival (in months) beginning at the time the patient underwent radiosurgery.

RESULTS: The actual median patient survival was 10.3 months (95%; 6.4-14). The median physician predicted survival was 9.7 months (neurosurgeons [NS]=11.6, radiation oncologist [RO]=11.6, medical oncologist [MO], 7.8 months). For patients who died before 10 months, both NS and RO clinicians were more optimistic. No group could accurately predict survivors alive at 14 months. For specific tumor types, predictions were accurate within the following ranges: a) Breast: prediction was accurate within 0-3 months = 31%, 3-6 = 22%, 6-9 = 21%, 9-12 = 9%, 12-18 = 12%, >18 = 6%; b) NSC Lung: 0-3 = 34%, 3-6 = 31%, 6-9 = 19%, 9-12 =7%, 12-18 = 7%, >18 = 1.4%; c) Melanoma: 0-3 = 57%, 3-6 = 19%, 6-9 = 10%, 9-12 = 9%, 12-18 = 35, > 18 = 2%. All physicians failed to predict patients who actually had extended survivals. In general medical oncologists had better predictive abilities. Only the one neurosurgeon who actually examined each patient had better predictive abilities. All physicians had survival predictions that were incorrect by as much as 12-18 months, and 7 of 11 had individual predictions that were in error by >18 months.

CONCLUSIONS: Predicting the survival of cancer patients is difficult despite the importance of such estimates when making educated treatment recommendations. For patients with actual brief survivals, physician predictions had greater accuracy. Survival predictions proved least accurate for patients who had extended survivals. In this study all physicians were unable to accurately predict long term survivors. Despite valuable clinical data and predictive scoring techniques, advanced brain and systemic management often led to patient survivals well beyond estimated survivals. Such survival predictions should not be used to ration care.

THURSDAY, OCTOBER 18

10:22-10:32 A NOMOGRAM FOR INDIVIDUALIZED ESTIMATES OF SURVIVAL OUTCOMES FOR PATIENTS WITH BRAIN METASTASIS

Andrew E. Sloan, MD, FAANS, FACS*; Jill Barnholtz-Sloan, Ph.D; Changhong Yu; Jaime Venoechea; Michael Vogelbaum, MD; Minesh Mehta, MD; MD;Mitchell Machtay, MD; & Michael W. Kattan, PhD.*

INTRODUCTION: Brain metastases are the most common intracranial mass lesions, with an incidence of 98,000-170,000 cases each year in the US. Its estimated that 24-45% of cancer patients develop brain metastasis which account for 20% of cancer deaths annually. Therapeutic options include hospice, chemotherapy, immunotherapy, whole brain radiotherapy, radiosurgery, and surgery, but the selection of the optimal treatment for individual patients remains controversial. Several risk classification schemes such as recursive partitioning analysis (RPA), and diagnosis-specific graded prognostic assessment (DS-GPA) have been developed and validated, but these provide group, rather than individualized estimates of outcome. Individualized estimations of survival could be useful for counseling patients and optimizing therapeutic approaches. The purpose of this study was to develop and validate a nomogram for individualized patient prognosis based on data from randomized controlled trials.

METHODS: De-identified data from 7 randomized controlled trials of brain metastasis in 2367 patients was obtained from the RTOG database and data on several variables of interest was obtained. Overall survival was estimated with adjustment for variables of interest using the Cox proportional hazards regression, RPA, and random survival forests (RSF) methods. The models were internally validated via 10-fold cross validation and the predictive accuracy for overall survival of the three approaches was calculated using a concordance index (CI). The significance levels for the three approaches was calculated from bootstrap and a final nomogram was built using the model with the highest predictive accuracy for individualized estimation of survival based on CI.

RESULTS: The majority of patients were classified as RPA class II and had DS-GPA scores of 1.24-2.5 and within each RPA and DS-GPA class, there was a wide range of individualized survival probabilities. The Cox analysis outperformed the RPA and RSF methods thus the nomogram was built to estimate the 6 and 12 month survival probabilities and median survival based on the Cox model. The predicted value approximated the observed value within a 95% confidence interval. When the accuracy for estimating survival of RPA and DS-GPA classes was compared with the nomogram, we found that the nomogram-estimated 12 month survival probabilities were heterogeneously distributed within each RPA and DS-GPA class. Variables which contributed significantly to risk assessment included: primary site; histology; status of primary disease; extent of metastatic spread; age; KPS; and number of brain lesions.

CONCLUSIONS: The nomogram based on the Cox model enabled better and more refined survival predictions than those based on RPA, DS-GPA or RSF models. This was due in part to consideration of primary site and histology compared to the RPA and DS-RPA classifications, as well as the number of brain lesions which was not considered in either of the earlier schemes. This predictor of outcome could be readily applicable to clinical practice in enabling patients and their physicians to make informed decisions regarding treatment options and will soon be provided as free software application. Future directions include external validation in a prospective dataset. The benefits and limitations of this approach will be illustrated using specific case vignettes.

THURSDAY, OCTOBER 18

10:34-10:44 IMPACT OF TRIPLE NEGATIVE PHENOTYPE ON THE PROGNOSIS OF BREAST CANCER BRAIN METASTASES UNDERGOING STEREOTACTIC RADIOSURGERY

Jason Sheehan, MD, PhD*, Zhiyuan Xu, MD, David Schlesinger, PhD, Tyvin Rich, MD, University of Virginia*

INTRODUCTION:
Hypothesis: The impact of triple-negative (TN) phenotype on survival of patients who harbored brain metastases arising from breast cancer and underwent stereotactic radiosurgery (SRS) is controversial. This study aims to elucidate survival times and identify potential prognostic factors.

METHODOLOGY: A total of 103 breast cancer patients with brain metastases were treated with SRS and then studied retrospectively. Twenty-four patients (23.3%) were TN. Survival times were estimated using Kaplan-Meier method with a log-rank test computing the survival time difference between groups. Univariate and multivariate analyses to predict potential prognostic factors were performed using a Cox proportional hazard regression model.

RESULTS: The presence of TN phenotype was associated with worsened survival times, including overall survival following the diagnosis of primary breast cancer (43 months vs. 82 months), neurological survival after the diagnosis of intracranial metastases and radiosurgical survival following SRS with median survival times being 13 months vs. 25 months, and 6 months vs. 16 months, respectively ($p<0.0002$ in all three comparisons). On multivariate analysis, radiosurgical survival benefit was associated with non-TN status and lower RPA class at the initial SRS. Conclusions: TN phenotype represents a significant adverse prognostic factor with respect to OS, NS, and RS in breast cancer women with intracranial metastasis. RPA also served as an important and independent prognostic factor.

THURSDAY, OCTOBER 18

10:49-10:59 DBS UNDER GENERAL ANESTHESIA WITHOUT NEUROPHYSIOLOGY: INITIAL EXPERIENCE AND COMPARISON TO THE STANDARD TECHNIQUES

Francisco A. Ponce, MD, *Barrow Neurological Inst.*

INTRODUCTION. There is a growing trend in functional neurosurgery toward direct anatomical targeting for deep brain stimulation (DBS). This study reports the results of an initial experience using a portable head CT scanner intraoperatively to place DBS electrodes under general anesthesia without the use of microelectrode recordings (MER).

METHODOLOGY. Direct anatomical targeting was performed preoperatively using a 3T-MRI study developed for visualization of DBS targets. Surgeries took place in a standard neurosurgical operating room. Following induction of general anesthesia and placement of bone-implanted fiducials, a CT was obtained. CT/MRI fusion was performed using a surgical navigation system. A frameless navigation-based skull-mounted DBS trajectory guide was used for placement of DBS leads. Following lead placement and prior to closing, a second CT was performed to verify accuracy. Accuracy was assessed using 2 types of measurements: "off plan" defined as the shortest distance from the line of the surgical plan to the targeted electrode contact, and the "tip error," defined as the vector distance between the intended target and the actual electrode contact. The results are compared with those obtained using traditional frame-based stereotaxy.

RESULTS. In a consecutive series of the 72 surgically implanted DBS electrodes, the described method was used for 19 electrodes placed in 10 patients. Targets included GPi (n = 11), STN (n = 4), and VIM (n = 4). Indications included Parkinson's disease (n = 7), essential tremor (n = 2), and dystonia (n = 1). The mean (± SD) distance off plan was 1.1 □□0.4 mm, and the mean tip error was 1.5 □□0.5 mm. Both the mean tip error and mean distance off plan were significantly smaller ($p < 0.05$, unpaired t-test) than the means for DBS electrodes implanted using traditional frame-based stereotaxy (1.8 □ 0.9 mm and 2.3 □□1.2 mm, respectively). Eighty-nine percent of the leads were placed with a single brain penetration, compared to 65% of leads using frame-based stereotaxy. No hematomas were visible on CT. In 7 patients in whom STN or GPi electrodes were implanted bilaterally, the mean operative time was 3.4 □ 0.8 hours (Range 2.1-4.7; mean room time = 4 hours). This was significantly shorter than for bilateral STN or GPi electrodes placed using MER-guided frame-based stereotaxy (n = 10, mean operative time 6.0 □ 1.0 hours, range 4.9-7.6; mean room time = 6.2 hours). Eight patients noticed favorable microlesion effects postoperatively, all patients had improvement of symptoms with stimulation, and no patient experienced unacceptably low thresholds for stimulation-related side effects.

CONCLUSIONS. The described method combines direct DBS targeting techniques, a frameless navigation-guided DBS system, and a portable CT to perform DBS under general anesthesia. The procedure offers the advantages of reduced operative times, improved accuracy that can be verified intraoperatively, and improved patient comfort.

THURSDAY, OCTOBER 18

11:01-11:11 IMAGE-GUIDED DBS ELECTRODE SURGERY WITHOUT MICROELECTRODE RECORDING: ACCURACY AND COSTS OF ELECTRODE PLACEMENT USING NEXFRAME AND THE CERETOM INTRAOPERATIVE CT SCANNER.

***Kim J. Burchiel, M.D., F.A.C.S.**, Ahmed Raslan, M.D., Stephen E. Griffith, M.D., Claire Glasgow, B.S., and Valerie C. Anderson, Ph.D. Dept. of Neurological Surgery, Oregon Health and Science University, Portland, OR*

INTRODUCTION: Image-guided DBS electrode placement using an intraoperative CT scanner was performed in patients with Parkinson Disease and Essential Tremor, under general anesthesia without the use of microelectrode recording (MER).

METHODS: Data were collected on patients who underwent DBS electrode placement without MER, during the first year that this new method of implantation was employed (February 2011 – March 2012). Electrodes were placed using the Medtronic NexFrame utilizing the Stealth Station running Framelink software. Intraoperative CT images were obtained using the Ceretom scanner, and merged on the Stealth Station with pre-operative MRI images, or with pre-operative CT images if MRI was not feasible. All procedures were performed with the patient under general anesthesia. Accuracy of electrode placement was the calculated as the linear distance between the target and the actual electrode location. A financial analysis of the procedure was also conducted to compare the costs to the hospital of this new procedure and the more traditional MER-based approach in a matched sample.

RESULTS: 51 patients participated in the study (64 $\pm$ 9.5 years old). There were 31 patients with Parkinson Disease, 19 with Essential Tremor, and 1 patient with Dystonia. All patients had bilateral DBS electrodes placed in one stage as follows: 19 ventralis intermedius (Vim), 10 subthalamic nucleus (STN), and 22 globus pallidus internus (GPi). There were no intraoperative complications. The mean accuracy of final DBS electrode placement was 1.524 $\pm$ 1.04 mm. There was a moderate negative correlation between the distance of the closest approach of the electrode to the surface of the lateral ventricle, and the accuracy of placement (Pearson correlation coefficient = - 0.40). Electrode trajectories passing > 4.0 mm from the wall of the ventricle were significantly more accurate ($p < .05$) than those whose approach was < 4.0 mm. Any deviation of the electrode trajectory was in every case medial to the anticipated target. There was no statistically significant difference between the closest approach of the electrode to the ventricle comparing the right and left sided targets. There were no intraoperative complications. The financial analysis indicated that there was no difference in costs to the hospital when comparing this image-guided approach to the MER-based approach.

CONCLUSIONS: The accuracy of DBS electrode placement under general anesthesia, without MER, is comparable to reported series using MER mapping. We have made a novel discovery that electrode trajectories that pass within 4 mm of the ventricle wall have a significantly higher risk of medial deviation of the electrode. This CT-based method avoids the additional risks of hemorrhage associated with MER, and attendant neurologic deficits. This method also allows immediate intraoperative feedback to the surgeon such that any unanticipated deviation of the electrode trajectory can be corrected immediately. In our experience, patient satisfaction with this method is high, accuracy of placement is high, and complications are probably less than 2%. Although MER continues to have a role in intraoperative research, its routine use is not required for accurate DBS electrode placement. There is no financial incentive, or disincentive, to the hospital to perform DBS procedures using image guidance.

THURSDAY, OCTOBER 18

11:13-11:23 MECHANISM STUDIES USING fMRI, WINCS, AND MINCS: TOWARDS NEURAL ENGINEERING ELECTROCHEMICAL FEED BACK DBS

Kendall H. Lee, MD, PhD, *Mayo Clinic, Rochester, MN*

INTRODUCTION: Deep brain stimulation (DBS) is an established treatment for movement disorders, and it is a rapidly emerging therapy for numerous psychiatric disorders. Thus, elucidating DBS mechanisms for improving outcomes has become a critical clinical goal in stereotactic and functional neurosurgery. We have addressed this issue by combining for the first time two powerful technologies, functional Magnetic Resonance Imaging (fMRI) and in vivo neurochemical monitoring to investigate DBS-mediated modulation of neural activity. We have previously described a wireless neurochemical monitoring device called Wireless Instantaneous Neurotransmitter Concentration Sensing (WINCS) system that combines fast scan cyclic voltammetry (FSCV) with wireless telemetry. For these studies, we have also developed a novel wirelessly controlled stimulation device called Mayo Investigational Neuromodulation Control System (MINCS), which provides electrical stimulation interleaved with rapid scan voltammetry obtained using WINCS. Here, we utilize fMRI, WINCS, and MINCS in the pig model to test the hypothesis that subthalamic nucleus (STN) DBS results in distal fMRI BOLD activation that correlates with specific neurotransmitter release.

METHODOLOGY: MINCS was designed to integrate operator-controlled neurostimulation with FSCV electrochemical recordings made by our previously described Wireless Instantaneous Neurochemical Concentration Sensing system (WINCS). MINCS incorporates analog circuitry for current- and voltage-regulated electrical stimulation, a 32-bit ARM microcontroller, a Bluetooth® transceiver, a multilayered printed circuit board, and, through an optical connection, is coupled to WINCS. To determine the feasibility and functionality of the integrated stimulation and recording system we conducted in vivo tests in the pig model of DBS, where the STN was targeted. Further, we employed 3Testla fMRI to determine the major sites of activation during application DBS (1-7V, 10- 300Hz 100-500 μs pulse width) in the isoflurane (1%) anesthetized porcine model.

RESULTS: With STN DBS, consistent areas of fMRI BOLD activation was seen, including the ipsilateral head of the caudate, premotor cortex, primary motor cortex, peduncular pontine nucleus, and the contralateral cerebellum. MINCS was capable of producing diverse waveform patterns under wireless control. Importantly, MINCS successfully demonstrated the capability to interleave stimulation pulses with FSCV scans, thereby avoiding stimulation artifacts. When various stimulation intensities and frequencies were applied, stimulation dependent dopamine release was seen in the area of fMRI BOLD activation.

CONCLUSIONS: These results suggest that the combination of fMRI, MINCS and WINCS may be useful for investigating the mechanisms of therapeutic neural stimulation. Because fMRI allows for global assessment of neural networks and electrochemistry allows for targeted analysis of the neurochemicals released in these same areas, this combination offers a new and exciting approach to understanding the anatomical and neurochemical correlates of the therapeutic action of DBS. Importantly, continuous monitoring of neurochemical changes following DBS surgery could pave the way toward a future implantable closed-loop “smart” device. Such a device would incorporate stimulation based on FSCV feedback from an implanted electrochemical micro-sensor so as to maintain neurochemicals at desired levels.

THURSDAY, OCTOBER 18

11:28-11:38 ENDOSCOPIC ENDONASAL APPROACH FOR RESECTION OF SKULL BASE CHORDOMAS: OUTCOMES AND LEARNING CURVE

***Paul Gardner, MD**, Maria Koutourousiou, MD, Matthew Tormenti, MD, Stephanie L. Henry, RN, BSN, Susan T. Stefko, MD, Juan Carlos Fernandez-Miranda, MD, Carl H. Snyderman, MD, MBA*
University of Pittsburgh School of Medicine, Pittsburgh, PA

BACKGROUND:
Present the endoscopic experience in the treatment of skull base chordomas

OBJECTIVE:
Gross total resection (GTR) of skull base chordomas represents a surgical challenge because of the location, invasiveness and tumor extension. In the last decade, the endoscopic endonasal approach (EEA) has been employed with notable outcomes.

METHODS:
From April 2003 to March 2011, 60 patients underwent an EEA for primary (n=35) or previously treated (n=25) skull base chordomas. We evaluated the degree of GTR and our complications. We studied the factors that influenced outcomes and compared our surgical results in the early and late years of our experience.

RESULTS:
The overall rate of GTR of skull base chordomas was 66.7% (82.9% in primary and 44% in previously treated cases). The most important limitations for GTR were tumor volume >20 cm^3 (p=0.042), tumor location in the lower clivus with lateral extension (p-0.022) and previously treated disease (p=0.0002). the learning curve had a significant impact on GTR, increasing the success rate to 88.9% (92.6% in primary cases, 63.6% in previously treated) during recent years (p<0.0001). The most frequent complication was cerebrospinal fluid leak (20%) resulting in meningitis in 3/3%. Carotid injuries occurred in 2 cases without any resulting deficit. Neurological complications included new cranial neuropathies (6.7%) and long tract deficits (1.7%). There was no operative mortality in our series.

CONCLUSION:
For the treatment of skull base chordomas, EEA represents a competitive alternative to transcranial approaches with minimal morbidity and high rates of GTR when performed by experienced skull base surgeons.

THURSDAY, OCTOBER 18

11:40-11:50 CURRENT MANAGEMENT OF MCA ANEURYSMS, RESULTS WITH A "CLIP FIRST" APPROACH

***Michael T. Lawton, MD**, University of California, San Francisco*

OBJECTIVE:
To review an experience with MCA aneurysms managed with microsurgery as the treatment of first choice.

METHODS:
During a 13-year period, 543 patients with 631 MCA aneurysms were managed with a "clip first" policy, with 115 patients (21.2%) referred from the Neurointerventional Radiology service and none referred from the Neurosurgical service for endovascular management.

RESULTS:
282 patients (51.9%) had ruptured aneurysms and 261 (48.1%) had unruptured aneurysms. MCA aneurysms were treated with clipping (88.6%), thrombectomy/clip reconstruction (6.2%), and bypass/aneurysm occlusion (3.3%). Complete angiographic aneurysm obliteration was achieved with 620 MCA aneurysms (98.3%). Relative to preoperative neurological baseline, 487 patients (89.7%) were improved or unchanged after therapy, with a perioperative mortality rate of 5.3% and a permanent neurological morbidity rate of 4.6%. Good outcomes were observed in 92.0% of patients with unruptured aneurysms and 70.2% with ruptured aneurysms. Worse outcomes were associated with rupture ($p=0.04$), poor-grade ($p=0.0004$), giant size ($p=0.03$), and hemicraniectomy ($p<0.0001$).

CONCLUSIONS:
The MCA aneurysm is managed better surgically than endovascularly, and surgery should remain the treatment of choice. Surgical morbidity was low and poor outcomes were due to an inclusive surgical policy which aggressively managed poor-grade patients with hemicraniectomy and hematoma evacuation, and complex aneurysms with thrombectomy and bypass. Surgical results from our experience set a benchmark that endovascular results should match before considering endovascular therapy as an alternative for MCA aneurysms.

THURSDAY, OCTOBER 18

11:52-12:02 USE OF SUPRAMAXIMAL STIMULATION TO PREDICT FACIAL NERVE OUTCOMES FOLLOWING VESTIBULAR SCHWANNOMA MICROSURGERY: RESULTS FROM A DECADE OF EXPERIENCE

***Michael J Link MD*[1,2]*, Colin L Driscoll MD*[2,1]*, William R Schmitt MD*[2]*, Matthew L Carlson MD*[2]*, Brian A Neff MD*[2]*, Jasper R Daube MD*[3]

Departments of Neurologic Surgery[1]*, Otorhinolaryngology*[2] *and Neurology*[3]*. Mayo Clinic and Mayo Foundation, Rochester, Minnesota*

INTRODUCTION/HYPOTHESIS: The goal of vestibular schwannoma (VS) surgery is tumor removal and preservation of neural function. Intraoperative facial nerve monitoring has emerged as the standard of care, however its role in predicting long-term facial function remains a matter of debate and techniques vary greatly between institutions. We describe, and critically assess the value of intraoperatively applying current at supramaximal stimulation (SMS) levels in an effort to identify patients destined for permanent facial paralysis following VS removal.

METHODS: Over more than a decade, the protocol for stimulating and assessing the facial nerve during VS surgery at our institution has consisted of applying pulsed constant-current stimulation at SMS levels proximally and distally following tumor resection in order to generate an amplitude ratio, which subtracted from 100% yields the degree to which the functional integrity of the facial nerve "dropped off" intraoperatively. This data was prospectively collected and additional variables that might impact postoperative facial nerve function were retrospectively reviewed from the medical record. Only patients with anatomically intact facial nerves and >12 months of follow-up data were analyzed.

RESULTS: There were 267 patients available for review. The average posterior fossa tumor diameter was 24 mm and the rate of long-term good [House Brackmann (HB) grades 1 – 2] facial nerve function was 84%. Univariate logistic regression analysis revealed prior treatment, NF2 status, tumor size, cerebellopontine angle extension, subjectively thinned facial nerve at the time of operation, minimal stimulation threshold of the proximal facial nerve, percent drop off by SMS, and postoperative facial nerve function were all found to correlate statistically ($p<0.05$) with long-term facial function. When evaluating patients with significant facial weakness at the time of hospital discharge, only the percent drop off by SMS remained a statistically significant predictor of long-term facial function. For all patients, regardless of facial function in the immediate perioperative period, who had a proximal-to-distal drop off >69%, the rate of poor long-term function (HB grades 3 – 6) was 44% (15/34). Conversely, those patients with a proximal-to-distal drop off of ≤69% experienced poor facial function only 6% (8/138) of the time. The positive predictive value of SMS for long-term severe weakness (HB grades 5 – 6) however, is low at 46%.

CONCLUSIONS: In a large cohort of patients, we found that interrogating intraoperative facial nerve function with SMS is safe and technically simple. This is the first report of using this technique. It is very useful, and we believe superior to other reported techniques, for predicting which patients will ultimately have good long-term facial function, but is very limited in identifying patients destined for long-term severe facial weakness that might benefit from early facial reanimation surgery. This technique may prove helpful in the future in tailoring less than gross total tumor removal to limit postoperative facial weakness but maximize tumor resection

THURSDAY, OCTOBER 18
INTRODUCTION OF ACADEMY AWARD WINNER AND HONORABLE MENTION

Daniel A. Orringer, MD, Honorable Mention: Label-Free Detection of Microscopic Tumor Boundaries Using Stimulated Raman Scattering Microscopy. (Dr. Orringer will not present)

Derek G. Southwell, MD, PhD:
INTRINSICALLY DETERMINED CELL DEATH OF DEVELOPING CORTICAL INTERNEURONS.

Derek G. Southwell[1, 2, 3, 9], Mercedes F. Paredes[2, 4],Rui P. Galvao[2, 10], Daniel L. Jones[1, 2], Robert C. Froemke[5, 11], Joy Y. Sebe[2], Clara Alfaro-Cervello[6, 12], Yunshuo Tang[2, 3, 7], Jose M. Garcia-Verdugo[6], John L. Rubenstein[8], Scott C. Baraban[1, 2] and Arturo Alvarez-Buylla[1, 2]

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Cortical inhibitory circuits are formed by GABAergic interneurons, a cell population that originates far from the cerebral cortex in the embryonic ventral forebrain. Given their distant developmental origins, it is intriguing how the number of cortical interneurons is ultimately determined. One possibility, suggested by the neurotrophic hypothesis, is that cortical interneurons are overproduced, and then following their migration into cortex, excess interneurons are eliminated through a competition for extrinsically derived trophic signals. Here we have characterized the developmental cell death of mouse cortical interneurons in vivo, in vitro, and following transplantation. We found that 40% of developing cortical interneurons were eliminated through *Bax- (Bcl-2 associated X-)* dependent apoptosis during postnatal life. When cultured in vitro or transplanted into the cortex, interneuron precursors died at a cellular age similar to that at which endogenous interneurons died during normal development. Remarkably, over transplant sizes that varied 200-fold, a constant fraction of the transplanted population underwent cell death. The death of transplanted neurons was not affected by the cell-autonomous disruption of TrkB (tropomyosin kinase receptor B), the main neurotrophin receptor expressed by central nervous system (CNS) neurons. Transplantation expanded the cortical interneuron population by up to 35%, but the frequency of inhibitory synaptic events did not scale with the number of transplanted interneurons.

Together, our findings indicate that interneuron cell death is intrinsically determined, either cell-autonomously, or through a population-autonomous competition for survival signals derived from other interneurons.

FRIDAY, OCTOBER 19

8:00-8:10 QUANTITATIVE IRON BURDEN AS A BIOMARKER OF CUMULATIVE HEMORRHAGES IN CEREBRAL CAVERNOUS MAFORMATION: STUDIES IN MOUSE AND MAN

Issam A. Awad, MD, MSc, FACS*, Univ of Chicago Medicine and Biological Sciences,*

INTRODUCTION/HYPOTHESIS: Cerebral cavernous malformations (CCM), which affect more than 0.5% of the population, are characterized by dilated "leaky" brain capillaries. These lesions expose patients to a lifetime risk of epilepsy and focal neurologic deficits related to repetitive hemorrhages. The CCM lesions contain iron-rich hemoglobin breakdown products, which can be depicted by susceptibility weighted imaging (SWI) using magnetic resonance imaging (MRI). However, SWI only provides lesion count and volume information, but is unable to provide information regarding the density of iron within individual lesions. Furthermore, SWI cannot distinguish hemorrhage from calcification, which can be a common occurrence in CCM patients. We hypothesize that iron burden in lesions can be quantified, and therapeutically modified in experimental CCM lesions in mice. We further hypothesize that a novel MRI technique, Quantitative Susceptibility Mapping (QSM), can distinguish iron from calcification, and can quantify iron burden in human CCM lesions.

METHODOLOGY: For the murine studies, we assessed iron burden in genetically engineered heterozygous Ccm1+/- models, sensitized to enhance spontaneous somatic mutations in Msh null background, which we demonstrated to form spontaneous CCM lesions recapitulating all histologic features of the human disease. Iron deposit was visualized by Perls blue staining, and quantified using NIH Image-J software by integrated density measurement of color thresholded blue signal, controlling for light intensity and magnification. Iron staining intensity of CCM lesions was compared in animals treated with placebo, and RhoA kinase inhibitor fasudil, previously shown to rescue the CCM phenotype in vitro and in vivo. The logarithm of integrated density of each lesion was used for statistical analysis. For the human studies, 5 patients with CCM (3 sporadic and 2 familial; the latter harboring 5 and 6 lesions respectively) were imaged with conventional, SWI and the new QSM technique.

RESULTS: Fourteen of 18 mature multicavernous CCM lesions identified in Ccm1+/-Msh2-/- mice (16 placebo and 14 fasudil animals) exhibited iron staining, but none of 82 solitary cavern capillary ectasia (p= 0.0002, two-tailed Fisher's exact). There was a significantly lower prevalence of mature CCM lesions in fasudil treated mice compared to placebo (p=0.02, Fisher's exact), and also a lower iron staining intensity in the CCM lesions of fasudil treated animals (Figure 5; P = 0.007, Student's t test). In the human studies, all CCM lesions (3 sporadic and 11 familial) were identified on SWI and QSM images. The mean susceptibility of the lesion ROIs across all patients was 0.49 ± 0.16 ppm. The lesion volume varied from 5 – 1370 mm3. The averaged total susceptibility per volume was 0.66 ± 0.34 ppm/mm3. Larger lesions had higher susceptibility values/ mm3, indicating higher iron concentration. Calcifications were clearly differentiated from iron in lesions (inverted signal on QSM), and this was verified in choroid plexus calcifications and on computed tomographic scans in the same patients (control).

CONCLUSIONS: Our results demonstrate a range of iron deposits in experimental CCM lesions, which can be altered by pharmacologic intervention. Preliminary studies in man demonstrate QSM's unambiguous ability to separate calcification from iron-rich CCM lesions, and to provide quantitative evaluation of the iron burden in lesions. Together, SWI and QSM may serve as novel imaging biomarkers to provide accurate lesion count (SWI) and quantitative changes in iron content in individual lesions (QSM). These will allow the longitudinal monitoring of CCM disease progression, and potential response to therapeutic intervention.

FRIDAY, OCTOBER 19

8:12-8:22 GRAFTED HUMAN NEURAL STEM CELLS ENHANCE SEVERAL STEPS OF ENDOGENOUS NEUROGENESIS AND IMPROVE BEHAVIORAL RECOVERY AFTER MIDDLE CEREBRAL ARTERY OCCLUSION IN T CELL-DEFICIENT RATS.

Yutaka Mine, MD, PhD [1, 2, 3], *Jemal Tatarishvili, MD, PhD* [1, 2], *Koichi Oki, MD, PhD* [1, 2], *Emanuela Monni, MSc* [1, 2], *Zaal Kokaia PhD* [1, 2], *Olle Lindvall, MD, PhD* [1, 2]

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OBJECTIVE: Neurogenesis from endogenous neural stem cells (NSCs) and NSC transplantation have been suggested as novel approaches to reconstruct stroke-damaged neural circuitry. In rats, endogenous NSCs in subventricular zone (SVZ) produce new neurons migrating into striatum during several months after stroke but their long-term survival is poor. For maximum recovery, enhancement of endogenous neurogenesis should probably be combined with NSC transplantation. NSCs have beneficial effects not only through cell replacement but also by trophic actions, neuroprotection, and modulation of inflammation. Our preliminary data suggested that transplanted human NSCs might enhance endogenous neurogenesis in intact brain. The aim of this study was to analyze the interaction between endogenous neurogenesis and human-derived NSC transplantation in stroke-damaged brain.

MATERIAL AND METHODS: T cell-deficient rats were subjected to unilateral 1-hour middle cerebral artery occlusion and human foetal NSCs or vehicles were transplanted into ipsilateral striatum 48 hours thereafter. Behavioural function was assessed using stepping and cylinder tests every 3 weeks. Animals received BrdU injections for 2 weeks at 4 weeks before termination, and were perfused 6 or 14 weeks after transplantation. Specimens were immunohistochemically assessed for endogenous neurogenesis, graft survival and inflammation after stroke.

RESULTS: We observed higher numbers of Ki67+ proliferating cells in the SVZ of the human NSC grafted group than in the vehicle group both at 6 and 14 weeks after transplantation. Also the numbers of Dcx+ migrating neuroblasts and BrdU+/Fox3 (NeuN) + newly formed mature neurons in the ischemic striatum were significantly higher in the human NSC transplantation group compared to the vehicle group at both time points. Human NSC transplantation reduced the number of Iba1+/ED1+ macrophages in the ischemic striatum. Grafted human NSCs were observed in all rats, but the number and the morphology of the grafts varied in the group. Animals with human NSCs grafts showed functional recovery in stepping test at 6 weeks and thereafter, whereas vehicle-injected animals did not. In cylinder test, the NSC transplanted animals showed improved recovery of impaired forelimb use at 12 weeks.

CONCLUSION: Intrastriatal human NSC transplantation enhances the proliferation, migration and maturation of endogenous NSCs after stroke, and this effect is long-lasting. Transplantation of human NSCs also reduces striatal inflammation and ameliorates neurological deficits after stroke. Our findings indicate that human NSC transplantation promotes endogenous neurogenesis from SVZ and modulates inflammation and support the idea that combination of NSC transplantation and stimulation of endogenous neurogenesis may become of value for functional restoration after stroke.

FRIDAY, OCTOBER 19

8:24-8:34 ERYTHROPOIETIN PROMOTES RESTORATION OF INHIBITORY CIRCUIT DEVELOPMENT AFTER TRANSIENT PRENATAL GLOBAL HYPOXIA-ISCHEMIA

***Shenandoah Robinson, MD**, Children's Hospital, Harvard Univ*

INTRODUCTION: Children born very preterm are prone to cognitive delay, and behavioral abnormalities such as autism, and epilepsy. Similar deficits are observed in infants who suffer injury brain injury during critical periods of cerebral circuit formation. The primary inhibitory neurotransmitter in the mature brain is □-amino butyric acid (GABA). As the cerebral cortex matures and responds to environmental stimuli, GABA responses switch from excitatory to inhibitory coincident with the upregulation of the cation-chloride co-transporter KCC2. In addition to regulating inhibitory responses, KCC2 regulates interneuron migration, and maturation of dendritic spines and synapses. Previously we found diminished cortical KCC2 expression in preterm infants with white matter injury. We hypothesized that perinatal brain injury delays or decreases KCC2 expression at critical periods in development, thus impairing formation of cerebral inhibitory circuits, and that the neuroprotective agent erythropoietin (EPO) could restore KCC2 expression.

METHODS: An established rat model of transient systemic hypoxia-ischemia (TSHI) on embryonic day 18 was used to mimic early third trimester placental insufficiency. Pups were born at term and EPO or vehicle (saline) was given on postnatal day 1 (P1)-P5. mRNA and protein expression were quantified using qPCR and Western blots, and whole cell voltage clamp of CA3 pyramidal neurons was used to measure miniature inhibitory postsynaptic currents (mIPSCs). We have previously shown reduced seizure threshold in adult rats after.

RESULTS: We observed a developmental upregulation of KCC2 from P7 to P21, and a concomitant decrease in the NKCC1/KCC2 ratio, as previously reported. Following TSHI (n=3), KCC2 membrane expression in CA3 was decreased by 62% compared to sham (n=4). EPO treatment attenuated the reduction in KCC2 expression (TSHI+veh: 39% control vs. TSHI+EPO 155% control). Functional analysis showed TSHI (12 cells/7 rats) decreased the mean amplitude and frequency of CA3 mIPSCs at P10-11, compared to shams (11 cells/6 rats). EPO treatment normalized the mIPSCs. qPCR analysis confirms loss of KCC2 protein concomitant with decreased transcription. Because brain-derived neurotrophic factor (BDNF) signaling through TrkB receptors regulates KCC2 transcription, we studied TrkB expression and found TSHI induces a loss of TrkB protein expression, which was restored by EPO treatment.

CONCLUSIONS: Perinatal brain injury exacts not only a tremendous toll on children and their families, but also society, by limiting their potential to become independent productive adults. Here we propose a mechanism for impaired formation of inhibitory circuits during development via loss of KCC2 membrane expression. The restoration of KCC2 expression and corresponding functional improvement after neonatal EPO treatment suggests a novel mechanism of EPO neuroprotection via upregulation of KCC2 expression.

FRIDAY, OCTOBER 19

8:36-8:46 UPDATE ON GLYBURIDE IN STROKE AND MALIGNANT CEREBRAL EDEMA

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Baltimore, MD

Extensive preclinical work over the last decade has established that the sulfonylurea receptor 1 (Sur1)-regulated $NC_{Ca\text{-}ATP}$ channel is upregulated *de novo* following cerebral ischemia/reperfusion. In animal models of stroke, block of Sur1 using a constant infusion of low-dose (non-hypoglycemogenic) glibenclamide (US adopted name, Glyburide) has been shown to exert robust salutary effects. In non-lethal models, glibenclamide reduces infarct volume and improves neurological outcomes. In lethal models with malignant cerebral edema, glibenclamide reduces edema, brain swelling and death, improves neurological outcomes, and has been found to be superior to decompressive craniectomy. The treatment window for glibenclamide exceeds 10 hours following onset of ischemia. Retrospective studies of humans with diabetes presenting with stroke have shown that patients who are on and stay on a sulfonylurea drug (glibenclamide, glimepiride, or gliquidon) fare better than matched controls. Patients with non-lacunar strokes who are on a sulfonylurea drug are far more likely to enjoy significant neurological improvement during hospitalization (a decrease in NIHSS score ≥4, or reaching 0) (42% vs. 0% in controls), they are far less likely to suffer symptomatic hemorrhagic transformation (0% vs. 11% in controls) and they are far less likely to die (0% vs. 10% in controls). A 10-patient Phase IIa open label prospective study of RP-1127 (Glyburide for injection, Remedy Pharmaceuticals) in non-diabetics was recently completed. This study, named "GAMES" (Glyburide Advantage in Malignant Edema and Stroke) pilot, examined the effect of a 72-hour infusion of RP-1127 in patients with large strokes (102±23 ml) at risk for malignant cerebral edema. In treated patients, the incidence of malignant cerebral edema and clinically significant hemorrhagic transformation were 20% and 0%, respectively, compared to 88% and 30% in large observational cohorts with similarly large strokes. Moreover, in treated patients, the incidence of modified Rankin scores (mRS) ≤4 at 1 month was 90%, compared to ~25% at 3 months in large observational cohorts. A large clinical trial studying the effect or RP-1127 in patients with large ischemic strokes is anticipated.

FRIDAY, OCTOBER 19

8:48-8:58 HOT-SPOTS IN DYNAMIC 18FET-PET ARE ASSOCIATED WITH UNFAVOURABLE OUTCOME IN PATIENTS WITH SUSPECTED WHO GRADE II GLIOMA

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INTRODUCTION: Three different uptake patterns of O-(2-[(18)F]fluoroethyl)-l-tyrosine (18FET) have been shown to occur in patients with suspected WHO II glioma after dynamic PET evaluation: 1; a constantly increasing uptake throughout the entire tumor volume indicative for a grade II glioma. 2; an early peak of uptake with following decline throughout the entire tumor volume indicative for a malignant glioma. 3; a heterogeneous uptake exhibiting both low- and high-grade characteristics (HOT-SPOT) at different sites of the tumor. The prognostic impact of these findings remains unclear so far. For clarification the following prospective study (2006-2010) was conducted.

METHODS: Adult patients with a magnetic resonance imaging based suspicion of a so far untreated WHO grade II glioma were considered eligible. Date of last follow-up was October 2011. Informed consent was available for all patients. Dynamic FET-PET evaluation was performed according to the protocol of Poepperl et al (2007). All patients underwent PET-guided stereotactic biopsy. Molecular genetics (MGMT, LOH 1p/19q, IDH-1) were evaluated. Progression free survival (PFS) was estimated with the Kaplan Meier method.

RESULTS: Ninety-eight patients (f/m 56/42, median age 45yrs) were included. Median follow-up was 16 months. Histological evaluation revealed 54 grade II and 44 high-grade gliomas. Tumor progression was noted in 31 patients. The diagnostic sensitivity and specificity of FET-PET was 89% and 87%. Homogeneous low-grade, homogeneous high-grade, and heterogeneous kinetics were seen in 52, 27, and 19 patients, respectively. The size of the HOT-SPOT in the heterogeneous group ranged from 5 to 66% of the entire tumor volume. Heterogeneous tumours showed significantly higher numbers of LOH1p/19q (56% vs. 16%) and IDH1 mutations (74% vs. 15%) than homogeneous high-grade gliomas. One/two-year PFS for patients exhibiting homogeneous low-grade, homogeneous high-grade, and heterogeneous kinetics was 86% / 78%, 63% / 35%, and 87% / 26%, respectively (p=0.002). Patients exhibiting heterogeneous kinetics did not better than those with homogeneous malignant kinetics. The size of the HOT-SPOT did not gain prognostic relevance.

CONCLUSIONS: Consideration of HOT-SPOT volumes within suspected grade II glioma is essential for histological and prognostic evaluation. Failure to detect these sometimes small malignant foci either by microsurgery or biopsy could easily lead to both undergrading and undertreatment.

FRIDAY, OCTOBER 19

8:36-8:44 MENINGIOMA GENE EXPRESSION PROFILING AS A POTENTIAL GUIDE TO POSTOPERATIVE PATIENT MANAGEMENT

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INTRODUCTION/HYPOTHESIS: The current WHO grading system for meningiomas does not adequately predict meningioma recurrence following surgical excision. From a practical patient management perspective one would like to identify the WHO grade 2 tumors that are unlikely to recur and the WHO grade 1 tumors that have an increased risk of recurrence. We hypothesized that gene expression profiling would identify a group of tumors that have either a greater or lesser risk of recurrence and thus aid in postoperative patient management.

METHODOLOGY: Tumor tissue was obtained from patients with a diagnosis of meningioma who underwent surgical excision between 1991 and 2007. The obtained tissue was from the first diagnosis of meningioma. Total RNA was prepared from snap-frozen tumor specimens and analyzed on Affymetrix GeneChip HG-U133 Plus 2.0. Clinical data was collected retrospectively from a prospectively documented patient database. Survival associates were analyzed by the Kaplan Meier/log rank method. Filtered Heat map was constructed using the top 250 genes based on median average deviation score.

RESULTS: There were 43 tumors identified. These tumors were excised from 13 male and 30 female patients. The median patient age at the time of surgery was 57years. The mean length of clinical follow-up was 3.9years. Recurrence was identified in 9 patients. Thirty-six of 43 patients were alive at the time of the study. A WHO grade 1 meningioma was identified in 26, grade 2 in 16 and grade 3 in 1. The median Ki-67 labeling index was 8. A Simpson grade 1 or 2 resection was accomplished in 37 of 43 patients (86%). Only 4 patients received postoperative radiation treatments. Of 9 recurrences 2 were WHO grade 1 (8% of all grade 1 tumors) and 7 were WHO grade 2 (44% of all grade 2 tumors). Hierarchical clustering revealed 2 dominant gene expression profiles. (Groups 1 and 2). The 5-year PFS for patients in profile group 1 was 100% while that for group 2 was 60% (p=0.05). There was no difference in overall survival. Correlation between recurrence and gene expression group using the Fischer's exact test was P=0.0196. All 9 patients with recurrence were in group 2. 14/16 WHO grade 2 tumors and the sole WHO grade 3 tumor were in gene expression group 2 (85%) but this did not reach significance. Molecular grade and WHO grade were independently significant predictors of recurrence based on multivariate analysis.

CONCLUSIONS: Gene expression analysis of a group of 43 meningiomas identified a distinct gene expression profile which was associated with a decreased risk of recurrence independent of WHO tumor grade. Identification of tumors with a decreased risk of recurrence may, at the very least, increase the interval of recommended follow-up and thus decrease costs. Identification of a subgroup of higher WHO grade tumors with a lower recurrence risk may influence the need and timing of postoperative radiation therapy.

FRIDAY, OCTOBER 19

9:12-9:22 THE NEXT GENERATION INTRACRANIAL MONITOR

***Raj K. Narayan, MD**, Chunyan Li, PhD, North Shore Univ Hospital, Manhasset, NY*

INTRODUCTION: Current monitoring of the injured brain generally consists of intracranial pressure (ICP) monitoring with a ventricular catheter, sometimes supplemented with the additional monitoring of brain temperature, brain oxygen and perhaps cerebral blood flow (CBF). Several additional indices are believed to affect outcome in these patients, and ideally these physiological and biochemical parameters should also be tracked. However, currently available monitoring technology is limited in this regard and treatment decisions are made empirically and opportunities to avoid or reverse secondary insults due to avoidable pathophysiology may be missed. Our research over the past few years has focused on developing a novel multimodality "smart catheter" that could accurately and simultaneously track multiple parameters in the injured brain. IN addition, the smart catheter should allow for drainage of excess cerebrospinal fluid (CSF) when needed to reduce intracranial pressure.

METHOD: Seven microsensors to measure brain pressure, temperature, flow, oxygen tension, glucose, lactate and electrophysiology were fabricated on a 7.5µm thick polyimide substrate using micro electro mechanical systems (MEMS) technology and rolled spirally to form a catheter structure (inner diameter =1.3mm; outer diameter =1.4mm). t he mechanical design and electrical operation of the sensors were carefully chosen such that potential electronic, thermal and chemical crosstalk among the sensors was negligible. A polysilicon-diaphragm-based pressure sensor was embedded on a flexible substrate. Temperature and flow sensors were based on micromachined gold resistance temperature detectors with a 4-wire configuration. The temperature sensor operated with AC excitation current without causing self-heating and the flow sensor employed a periodic heating and cooling technique with a constant-temperature mode. An oxygen sensor with three-electrode configuration was designed to achieve zero net oxygen consumption. Glucose and lactate sensors were based on amperometric enzyme-based electrochemical detection. Heterostructured electroencephalograph (EEG) electrode array was developed to achieve a superior signal-to-noise ratio.

RESULTS: The performances of the microsensors were compared to commercial probes for each of the different measures. We evaluated the sensors for accuracy, crosstalk and long-term drift in human blood-stained cerebrospinal fluid. The pressure sensor was found to have an accuracy of 1 mmHg in the linear range of 0 to 50 mmHg. The temperature and flow sensors had resolutions of 0.013ºC and 0.18ml/100g/min and achieved accuracies of 0.1ºC and 5ml/100g/min, respectively. The oxygen, glucose and lactate sensors had an accuracy of 1mmHg, 0.02mM, 0.05mM in the linear range from 0 to 60mmHg, 0.1 to 10mM, 0.05 to 8mM, respectively. EEG electrodes resulted in a more than 17-fold improvement in the electrode-electrolyte impedance at 1KHz than gold electrodes.

CONCLUSION: We have been able to simultaneously and accurately measure intracranial pressure, brain temperature, local cerebral blood flow, oxygen tension, glucose and lactate concentrations with a single smart catheter over the course of 5 days. This device has the potential to advance the field of neuromonitoring into a completely new era, in which medical decisions will be based on real-time continuous measures of brain biochemistry and physiology during the critical period immediately following a brain injury.

FRIDAY, OCTOBER 19

9:24-9:34 FUNCTIONAL GENOMICS IDENTIFIES DRIVERS OF MEDULLOBLASTOMA DISSEMINATION

Daniel W. Fults, MD, Univ of Utah School of Medicine, Salt Lake City, Utah, USA,

Medulloblastomas are malignant brain tumors that arise in the cerebellum in children and disseminate via the cerebrospinal fluid to the leptomeningeal spaces of the brain and spinal cord. Challenged by the poor prognosis for patients with metastatic dissemination, pediatric oncologists have developed aggressive treatment protocols, combining surgery, craniospinal radiation, and high-dose chemotherapy, which often cause disabling neurotoxic effects in long-term survivors. Insights into the genetic control of medulloblastoma dissemination have come from transposon insertion mutagenesis studies (Wu *et al*, *Nature* 482: 529-33, 2012). Mobilizing the Sleeping Beauty transposon in cerebellar neural progenitor cells caused widespread dissemination of typically nonmetastatic medulloblastomas in *Patched*$^{+/-}$ mice, in which Sonic Hedgehog (Shh) signaling is hyperactive. Candidate metastasis genes were identified by sequencing the insertion sites and then mapping these sequences back to the mouse genome. To determine whether genes located at transposon insertion sites directly caused medulloblastomas to disseminate, we overexpressed candidate genes in Nestin^{+} neural progenitors in the cerebella of mice by retroviral transfer in combination with Shh. We show here that ectopic expression of *Eras* (embryonic stem cell–expressed Ras), *Lhx1* (LIM homeobox protein–1), and *Ccrk* (cell cycle–related kinase) shifted the *in vivo* growth characteristics of Shh-induced medulloblastomas from a localized pattern to a disseminated pattern in which tumor cells seeded the leptomeningeal spaces of the brain and spinal cord. Expression of *Eras*, *Lhx1*, and *Ccrk* was elevated in tumor subgroups that show a high rate of metastasis and short patient survival times, indicating that these genes promote aggressive growth in human medulloblastomas, as they do in experimentally induced tumors in mice.

FRIDAY, OCTOBER 19

9:36-9:46 ELECTRICAL AND OPTOGENETIC NEUROMODULATION OF SEPTO-HIPPOCAMPAL OSCILLATIONS FOR THE TREATMENT OF EPILEPSY

Robert E. Gross, MD, PhD [1,2,3], *Nealen G. Laxpati, BS* [1,2], *Sharanya A. Desai, BS* [2], *Jack Tung, BS* [2,4], *Claire-Anne Gutekunst, PhD* [1], *and Steve M. Potter, PhD* [2]

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RATIONALE: Hippocampal oscillations in the theta range are associated with decreased epileptogenicity. We hypothesize, therefore, that induction of hippocampal theta with neuromodulatory techniques will decrease seizures. First, we are exploring induction of theta by direct hippocampal distributed electrical microstimulation and single-point macrostimulation in the tetanus toxin rat model of mesial temporal lobe seizures. Second, we are utilizing cell-specific optogenetic techniques within the medial septum (MS) which, with the hippocampus, comprises the septohippocampal system. Glutamatergic neurons of the MS have been proposed as a theta pacemaker due to their direct projections to hippocampal pyramidal neurons and tendency to fire at theta frequencies. To functionally investigate these connections in vivo, we explored cell-type specific activation of the MS and the resulting oscillatory local field potential (LFP) activity in the dorsal hippocampus, in control and epileptic rats.

METHODS: Various patterns of electrical stimulation were studied in control and epileptic (tetanus-toxin hippocampal injections) rats implanted with either a 16-channel multimicroelectrode array (MEA) (8 electrodes targeted to both CA3 and CA1 regions) or a single macroelectrode in CA3. In optogenetic experiments, rats were infected in the MS with 1) AAV2-CaMKIIα-ChR2, specifically targeting glutamatergic neurons; 2) AAV5-hSynapsin-ChR2, non-specifically expressing in all neurons; or 3) a control virus. After two weeks, each rat was implanted with an optical fiber targeting the MS and a 16-channel MEA in CA3/CA1. Animals underwent 473 nm blue laser stimulation across numerous parameters, including frequencies from 7-42 Hz and pulse widths of 1-10 ms. Electrode and optical fiber location was confirmed histologically and cell-specific transgene expression was immunohistochemically determined. Recorded electrophysiological data was spectrographically analyzed using custom-written and Chronux Matlab scripts.

RESULTS: Compared to controls, epileptic rats exhibited decreased hippocampal theta power ($p<0.0001$), and asynchronous theta square pulse stimulation reduced seizures by 58% ($p<0.05$). Optogenetic control of cell-type specific neurons of the MS in awake and behaving rats drove activity locally and in the dorsal hippocampus. At beta (15-35Hz) and gamma (40+Hz) frequencies, increase in stimulus frequency-specific power in the hippocampal LFP was observed with either neuronal target, but not in ChR2-negative controls. Notably, frequency-specific power generated in CaMKIIα-ChR2 rats was less than that generated in hSyn-ChR2 rats, and in anesthetized animals (reduced theta state), theta stimulation generated corresponding hippocampal LFP oscillations only in hSyn-ChR2 rats. In contrast, in awake behaving non-epileptic animals, neither hSyn-ChR2 rats nor CaMKIIα-ChR2 stimulation could increase theta power, but hSyn-ChR2 rats could phase-lock extant hippocampal theta oscillations to the optical stimulus.

CONCLUSION: Certain patterns of theta electrical stimulation decreased epileptic activity in rats. Further, we have developed a system for optogenetic stimulation and multielectrode recording, and used it to begin dissecting the neural circuitry of the septohippocampal axis. Our results show that the medial septum functionally modulates hippocampal activity, but that MS glutamatergic neurons are not the drivers of theta activity, due to their inability to modulate the hippocampal theta rhythm in awake, behaving animals. Conversely, non-glutamatergic neurons are involved in phase-locking hippocampal theta oscillations. Optogenetic experiments in epileptic animals are underway.

FRIDAY, OCTOBER 19

9:48-9:58 THIN-WALLED DOME REGIONS CO-LOCALIZE WITH LOW HEMODYNAMIC WALL SHEAR STRESS IN UNRUPTURED CEREBRAL ANEURYSMS

Laith M. Kadasi BS, Walter Dent, MS, and ***Adel M. Malek, MD, PhD***

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INTRODUCTION/HYPOTHESIS: Wall shear stress (WSS) plays a critical role in regulating endothelial function with low WSS being associated with a pro-inflammatory deleterious phenotype. The aim of this study was to evaluate the spatial relationship between localized thin regions of the aneurysm dome and estimated hemodynamic factors, hypothesizing that low WSS would correlate with aneurysm wall degeneration.

METHODOLOGY: Steady-state computational fluid dynamic (CFD) analysis was performed on sixteen aneurysms based on rotational angiographic volumes in order to derive maps of WSS, its spatial gradient (WSSG), and pressure (P). Local dome thickness was estimated categorically based on tissue translucency from high-resolution intraoperative microscopy during clipping. Each computational model was oriented to match the corresponding intraoperative view, and numerically sampled in thin and normal adjacent dome regions, with controls at the neck and parent vessel. Pressure differential (PD) was computed as the difference between aneurysm dome points and mean neck pressure. Pulsatile time-dependent analysis was carried out in a subset of seven patients to confirm the steady-state findings.

RESULTS: Matched-pair analysis revealed significantly lower levels of WSS (0.381 Pa vs. 0.816 Pa; $P<.0001$) in thin-walled dome areas compared to adjacent baseline thickness regions. Similarly, log WSSG and log WSS*WSSG were both lower in thin regions (both $P<.0001$); multivariate logistic regression analysis identified lower WSS and higher PD as independent correlates of lower wall thickness with an area under the curve of 0.80. This relationship was observed in both steady-state and time-dependent pulsatile analyses.

CONCLUSIONS: Thin-walled regions of unruptured cerebral aneurysms co-localize with low wall shear stress, suggesting a cellular mechanotransduction link between areas of flow stasis and aneurysm wall thinning. These findings suggest a possible supplementary role for pre-treatment computational hemodynamic analysis in aneurysm treatment decision analysis.

FRIDAY, OCTOBER 19

10:30-10.40 THE NATIONAL NEUROSURGERY QUALITY AND OUTCOMES DATABASE (N^2QOD): REVIEW OF FOUNDING SITE PERFORMANCE, REGIONAL CHALLENGES TO IMPLEMENTATION AND STRATEGIES FOR STREAMLINING NATIONAL DATA COLLECTION REQUIREMENTS

Anthony Asher, MD, FACS (*Carolina Neurosurgery and Spine Associates, Carolinas Medical Center), Mathew McGirt, MD (Vanderbilt Univ Department of Neurosurgery), Paul McCormick, MD, FACS (Columbia Univ Department of Neurosurgery)*

INTRODUCTION: The National Neurosurgery Quality and Outcomes Database (N2QOD) is a clinical registry designed to address the need for high-quality clinical outcomes data related to care of patients with neurosurgical and spine disorders. Emerging practice data requirements will impact all neurosurgeons and include PQRS, clinical "distinction" programs promoted by private insurers, MOC/MOL/hospital credentialing programs, value based purchasing and mandatory public reporting. Independent of these external requirements, collection and analysis of this data is essential to the development of risk-adjusted benchmarks for care and targeted local quality improvement efforts.

METHODS: The American Association of Neurological Surgeons has partnered with the Congress of Neurological Surgeons, American Board of Neurological Surgery and Society of Neurological Surgeons to create the NeuroPoint Alliance (NPA), a not-for profit organization dedicated to facilitating the collection of clinical and economic data from practice for a variety of purposes. The NPA's largest effort is the National Neurosurgery Quality and Outcomes Database (N2QOD), which was developed in conjunction with the Vanderbilt Institute of Medicine and Public Health (VIMPH), along with other national stakeholders. The information technology infrastructure for this project is based on the highly versatile and scalable REDCap data collection platform. NPA has recently completed development of a website and intra-net system to facilitate communication among participating sites and provide a repository for relevant reference information. The NPA has been engaged in an extended stakeholder outreach effort in order to intelligently inform all aspects of registry design and development. Our recent federal government outreach program has been particularly productive.

RESULTS: The N2QOD Lumbar "standard" Module was activated in 6 clinical centers on February 22, 2012 after 2 years of development. Presently, 24 large national centers are submitting data to the N2QOD; another 13 are in the process of activating contracts with NPA and several others are in various stages of registry activation. Verbal and written federal regulatory guidance from HHS (OCR and OHRP, respectively) has greatly facilitated local review and implementation of the program. As of June 8, 2012, over 51,000 independent data variables have been collected on 819 patients; 123 of those patients have completed their 3 month follow-up. Initial data capture rate in all sites is approximately 96%. Initial patient accrual at certain sites has not correlated with initial volume projections, and factors contributing to this variability will be reviewed. Preliminary (6 month) aggregate patient-reported outcomes data, data collection efficiencies, preliminary statistical analyses for required patient volumes per site and cumulative data collection rates will be presented. The authors will also review discussions with ABMS, ABNS, NQF, CMS and AHRQ relevant to the development of a national data collection system that will meet the requirements for PQRS, MOC and local quality improvement, along with business models that could support such an effort.

CONCLUSIONS: Practice data collection is being required on many levels-and this activity will redefine modern medical practice. Neurosurgeons have the opportunity to define quality and value in our specialty. It is essential that we commit to individual and collective investments in sustainable systems for data collection and analysis. The business and scientific models that sustain these systems will necessarily evolve as our efforts mature.

FRIDAY, OCTOBER 19

9:42-9:52 INTEGRATION OF THREE SEPARATE DEPARTMENTS INTO A COMBINED NEUROENDOVASCULAR UNIT, FACILITATED BY A COLLABORATIVE FINANCIAL MODEL

Christopher S. Ogilvy, MD,* *Thomas Moore,*[+] *Joshua A. Hirsch, MD**
*Albert J. Yoo, MD,** *James D. Rabinov, MD,** *Thabele M. Leslie-Mazwi, MD,** *Michael Jaff, DO***

**Department of Neurosurgery, Massachusetts General Hospital*
[+]*Human Resources, Massachusetts General Hospital*
***Cardiology, Massachusetts General Hospital*

Neuroendovascular techniques are currently practiced by neurosurgeons, neuroradiologists, and neurologists. The compensation model utilized for these various practitioners has created challenges at various institutions in terms of the compensation and service delivery models utilized for the practitioners. In many centers, each department is responsible for their practitioners' professional fees. At our institution, the Combined Neuroendovascular Unit is managed under the auspices of the Vascular Center. We have worked with our Vascular Center to develop a vascular model designed to enhance collaboration in management of patients pre-, intra-, and postprocedurally. In addition, financial incentive is provided for referral of patients within the group of practitioners.

For each physician within the group there is a 60/30/10 incentive compensation model. Making up the 60% is a stipend for call coverage, which is shared equally by the various members, a stipend for academic rank, and individual clinical productivity is compensated based on wRVUs. The 10% incentive pool is a group incentive for overall growth of the program.

Unique to this system is an incentive of up to 30% of the physician's compensation based on collaboration and referrals within the group. For collaboration, a three-tiered system of an interaction regarding a specific case has been established. Collaboration has been defined to range from periprocedural general consultation to intraprocedural consultation or extensive intraprocedural collaboration.

Institutions nationwide grapple with the challenge of equitable distribution of workforce resources and expertise, particularly where members of each department perform similar procedures. The plan presented here is an attempt to enhance collaboration as a solution to some of these concerns.

FRIDAY, OCTOBER 19

10:54-11:54 IMPROVING MEDICAL STUDENT RECRUITMENT INTO NEUROSURGERY: A MULTI-TIERED STRATEGY

Gandhi, Chirag D; Tomei, Krystal; Agarwal, Nitin; ***Prestigiacomo, Charles J***

INTRODUCTION: Attracting the brightest medical students into neurosurgery remains a challenge. Recent AANS data suggests that improving the opportunity for early exposure and mentorship for medical students can potentially improve recruitment into the subspecialty. We present our institutional experience with this approach.

METHODS: Summarize the medical student experience at The New Jersey Medical School (NJMS) between 1195-2012. Review the staged improvements made in both the clinical and pre-clinical experience. Include strategies for involving faculty, residents/fellows; the integration of multi-disciplinary didactics; creating consistent research opportunities with supervision; the development of a neurosurgery-specific interest group.

RESULTS: Prior to 2000, four applicants successfully matched into ACGME-approved neurosurgery residency over the previous half-decade. Increasing numbers from 2001-2010 with improving opportunities for students. From 2007-2009 an average of 4.67 applicants annually matched from NJMS (AANS Data). From 2007-2012, notable annual increase in both abstracts and publications with student involvement. Interest group received very well among student body with increasing number of students further exposed to neurosurgical education.

CONCLUSION: Developing a comprehensive approach to medical student education can be a very effective strategy to improve interest in neurosurgery and disperse neurosurgical education. Key areas of concentration should include mentoring programs with faculty and house-staff, multi-disciplinary didactics, organized research opportunities, and the development of a neurosurgery-specific interest group.

SATURDAY, OCTOBER 20

7:30-7:40 PERIPHERAL NERVE GRAFTS AND CHONDROITINASE ABC APPLICATION IMPROVES FUNCTIONAL RECOVERY AFTER COMPLETE SPINAL CORD TRANSECTION

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INTRODUCTION: Spinal cord injury (SCI) is a devastating trauma. The incapacity for axonal regeneration at the injury site after a SCI is due to an accumulation of upregulated growth inhibitors such as chondroitin sulfate proteoglycans (CSPGs), which form a glial scar. Several studies showed that chondroitinase ABC (ChABC), an enzyme that digests the CSPGs, promotes axonal sprouting. At *in vivo* temperature ChABC loses its activity after approximately three days. Most current methods to apply ChABC involve either one time injection into the spinal cord or multiple intrathecal injections, which leads to periods of extreme high and low concentrations. This strongly implies there is a need for better ways to deliver ChABC. In the present study, we test the synergistic effect of using predegenerated peripheral nerve grafts (PNGs) as scaffolds, while delivering ChABC via oligo-(polyethylene glycol) fumarate (OPF) tubes, to promote functional recovery after complete spinal cord injury.

METHODS: Procedures were in accordance with the protocols of The Animal Use and Care Committee and followed National Institutes of Health guidelines for the use and care of laboratory animals. Female Sprague Dawley rats weighing 200-250 g were used. Rats were divided into four groups. Group 1, used as control, underwent complete spinal cord transection at T10. Group 2, after transection, received two 3 mm segments of PNGs. In Group 3, the PNGs were wrapped in OPF tubes containing ChABC in microspheres, before placement in the transection cavity. Group 4 is similar to Group 3 but ChABC was incorporated directly into the hydrogel tubes. Functional recovery of hind limb motor activity was assessed using the BBB rating scale. Rats were tested before surgery, then weekly post-surgery for 8 weeks. After 8 weeks, the sciatic nerves were exposed and 5 µl of 1% CTB conjugated to AlexaFluor 594 were injected into the sciatic nerves, and left for a week to allow for proximal transfer through the axons, to assess for regeneration. After one week, rats were perfused. Spinal cord segments were then frozen and sectioned horizontally (30 µm). Slides underwent different immunohistochemical procedures for detection of either CTB or neurofilaments (NF) under a fluorescence microscope. Myelin sheath staining on paraffin-embedded sections was performed with osmium tetroxide to check for myelinated axons and count them. All quantitative data are presented as means ± standard error of the mean (SEM). The Student's t test was used to compare specific groups to the control group. A value of $p < 0.05$ was considered statistically significant.

RESULTS: The rats treated with PNGs (Group 2) scored significantly higher than the controls on days 42, 49, and 56. ChABC treated rats did significantly better than the control group; Group 3 was significantly higher than Group 1 on all days except days 7 and 21 and Group 4 was significantly higher than Group 1 on all days except day 7. On all days after day 7, the ChABC groups did better than the rats that only received the PNGs (Group 2). Group 3 was significantly higher than Group 2 on days 14, 18, 35 and Group 4 was significantly higher than Group 2 on day 35. Although there is a trend showing that Group 3 did better than Group 4, there was no statistical significance. Numerous regenerated axons were observed with NF stain at 8 weeks in all treated groups. Axon counting using myelin staining revealed a mean number of axons of: 2640 in group 2, 2010 in group 3, and 2740 in Group 4. There was no significant difference between these groups. The CTB was successfully taken up by the sciatic nerve in 22 rats and seen caudal to the graft. In the control rats, the CTB was never seen cranial to the lesion. CTB was observed in the PNGs and in the spinal cord cranial to it in 5 out of 6 rats from Group 2, 4 out of 6 rats from Group 3, and 4 out of 5 rats from Group 4.

CONCLUSIONS: Our study shows that PNGs can establish a good anatomical bridge after SCI and set the stage for functional recovery. OPF tubes are a good vehicle for ChABC delivery and are easy to manipulate surgically. Future studies should test combining different modalities, to include use of additional molecules to promote axonal regeneration like NT3, retraining like biking, or even permissive hypoxia. We believe that future success in treating SCI will require a multimodality approach.

SATURDAY, OCTOBER 20

7:42-7:52 MicroRNA AS A NOVEL IMMUNOTHERAPEUTIC STRATEGY TO REVERSE GLIOMA-MEDIATED IMMUNE SUPPRESSION AND ENHANCE ANTI-TUMOR CLEARANCE

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INTRODUCTION: MicroRNAs (miRs) have been shown to modulate critical gene transcripts involved in tumorigenesis, but their role in tumor-mediated immune suppression is unknown. We have previously demonstrated that the signal transducer and activator of transcription 3 (STAT3) is a key molecular hub for gliomagenesis and tumor-mediated immune suppression. In this study, we evaluated miRNAs that are preferentially down-regulated in gliomas and that interact with immune suppressive pathways such as STAT3 as potential new therapeutics.

METHODOLOGY: Comparative glioblastoma to normal brain tissue microarrays were used to identify differentially expressed miRNAs. To determine if these miRNAs are interacting with and regulating the STAT3 pathway, target scan analysis, real-time quantitative PCR, mutational analysis and forced overexpression and inhibition were used in glioma cancer stem cells (gCSCs) and the immune cell population to dissect these interactions. Lead candidate miRNAs were administered in multiple immune competent murine models of glioma to ascertain therapeutic efficacy and modulation of the immune system including within the tumor microenvironment.

RESULTS: In a comprehensive glioma tissue microarray, miR-124 expression was significantly down modulated in all grades and types of gliomas relative to normal brain. Upon up regulating miR-124 in glioma cancer stem cells (gCSCs), STAT3 was inhibited; this inhibition reversed tumor-mediated immune suppression, as reflected by an increase in T cell proliferation, Foxp3+ regulatory T cell (Treg) inhibition, and pro-inflammatory immune response up regulation. Treatment of immune- suppressed glioblastoma patient T cells with miR-124 induced a marked effector response. Within the gCSC population – a direct inverse correlation is observed between miR-21 and miR-124 expression. Forced over expression of miR-124 in gCSCs inhibits the IL-6 receptor and STAT3 protein expression, inhibited miR-21, and decreases the gCSC immunosuppressive properties. In
contrast, the forced overexpression of miR-21 markedly enhances the immunosuppressive properties of the glioma cancer stem cells. The in vivo local or systemic administration of miR-124 in multiple murine models of glioma, including genetically engineered heterogeneous high-grade gliomas, exerted potent anti-glioma therapeutic effects secondary to STAT3 inhibition in the immune cell population and enhanced effector responses in the local tumor microenvironment.

CONCLUSION: In summary, miR-124 may be a novel immune-activating agent for glioma treatment (including all grades and types). Mechanistic studies demonstrate that miR-124 controls the STAT3 pathway proximally while STAT3 regulates miR-21, thus demonstrating a complex regulatory axis of miRNAs on this transcriptional pathway. By exploiting the immune system to mediate direct tumor cytotoxicity, the vexing problem of miR delivery to tumors has been overcome.

SATURDAY, OCTOBER 20

7:54-8:04 TREATMENT OF MEDULLOBLASTOMA WITH MEASLES VIRUS ENCODING THE THYROIDAL SODIUM-IODINE SYMPORTER PLUS RADIO-IODINE

Brian Hutzen, Adam Studebaker, ***Corey Raffel***

INTRODUCTION: Measles virus (MV) shows potential as an oncolytic treatment for a number of human tumors. We have investigated the use of measles virus for the treatment of medulloblastoma and have demonstrated increased survival by treatment with MV in murine xenograft models of both intracerebral and CSF-disseminated medulloblastoma. In order to increase the efficacy of measles virus treatment of medulloblastoma, we have investigating the use of a virus of armed with the sodium-iodine symporter (MV-NIS) from the thyroid gland and combining treatment with this virus with radioiodine treatment.

METHODS: Medulloblastoma cells lines were treated *in vitro* with MV-NIS and assessed for the ability to replicate in, kill, and concentrate radioiodine in infected cells. The ability of cells to incorporate radioiodine *in vivo* was assessed in the intracranial model. Survival of animals treated with MV-NIS plus radioiodine in both the localized intracerebral and CSF-disseminated xenograft models of medulloblastoma was determined by the Kaplan-Meyer method. Results were compared to survival with MV-NIS alone and with unarmed virus.

RESULTS: MV-NIS retained the ability to replicate in and to kill medulloblastoma cells in vitro. Efficacy was similar to the unarmed virus. In addition, infected cells concentrated radioiodine, and an inhibitor of NIS decreased the intracellular concentration of radioiodine by the infected cells. Intracranial tumors treated with MV-NIS incorporated radioiodine as demonstrated by radio-imaging. In the intracranial model, survival was significantly increased by a single does of MV-NIS and radioiodine compared to 5 doses of unarmed virus. Timing of the delivery of the radioiodine relative to the treatment with MV-NIS was critical, with mice receiving radioiodine at either 24 or 48 hours, but not 72 hours, after MV-NIS treatment exhibited a survival advantage over mice given MV-NIS alone ($p \leq 0.05$).

CONCLUSIONS: MV-NIS is a potentially useful agent in the treatment medulloblastoma. The ability of MV-NIS to induce medulloblastoma tumor cells to concentrate radioiodine may have clinical significance for radio-imaging and targeted radio-therapeutic applications. Further investigation of MV-NIS for eventual use in a medulloblastoma clinical trial is underway.

SATURDAY, OCTOBER 20

8:06-8:16 PREOPERATIVE ASSESSMENT OF MENINGIOMA STIFFNESS BY MAGNETIC RESONANCE ELASTOGRAPHY

Fredric Meyer, MD, Matthew Murphy, PhD, John Huston, MD, and Richard Ehman, MD

INTRODUCTION: Magnetic Resonance Elastography (MRE) is an MRI-based technology for quantitatively assessing the mechanical properties of tissue. MRE has emerged as a routine tool for diagnosing liver fibrosis. It is also now being used to evaluate patients with Alzheimer's Disease. Both the ease and risk of resection of some cranial tumors is in part dependent on the softness of the tumor. For example, the risk of vascular injury increases with paraclinoid meningiomas that are fibrous and encase the carotid artery. Likewise, ease of internal debulking of a large falcine meningioma is dependent on firmness of the tumor. Accordingly, it would be advantageous for the surgeon to have reliable preoperative information regarding tumor softness.

OBJECTIVE: To determine if MRE can be used preoperatively to determine meningioma firmness.

METHODS: In thirteen patients MRE data were collected with a spin-echo EPI pulse sequence on a 3T MR imager. Shear waves at 60 Hz were introduced with a soft pillow-like driver placed under the head. The curl of the wave images was calculated and stiffness was determined with a direction-inversion algorithm. Blinded to the MRE results, the neurosurgeons made a qualitative assessment of tumor stiffness at the time of resection using a 4-point scale. The ability of MRE to predict the surgical assessment of stiffness was tested with Spearman rank correlation.

RESULTS: One case was excluded due to small tumor size. In the remaining 12 cases, both tumor stiffness alone ($p=0.023$) and the ratio of tumor stiffness to the stiffness in the surrounding brain tissue ($p=0.0032$) significantly correlated with the surgeons' qualitative assessment of tumor stiffness. The results of the MRE exam provided a stronger correlation with the surgical assessment of stiffness compared to traditional T1 and T2 weighted imaging ($p=0.089$), particularly when considering meningiomas of intermediate stiffness.

CONCLUSIONS: In this cohort, MRE was able to correctly predict tumor consistency at the time of surgery. Tumor stiffness as measured by MRE outperformed conventional MRI assessment on T1 and T2 images. MRE may prove to be a useful preoperative surgical planning tool

SATURDAY, OCTOBER 20

8:18-8:28 TISSUE ENGINEERED INTERVERTEBRAL DISCS: AN *IN VIVO* STUDY

Peter Grunert MD, ***Roger Hartl MD,*** *Weill Cornell Neurosurgery Department*

INTRODUCTION: Degenerative diseases of the spine may lead to neurological symptoms which may require surgical intervention. As fusion procedures sacrifice motion and may lead to adjacent segment disease, disc prostheses were developed in order to maintain segmental mobility. Tissue engineered intervertebral discs (TE-IVDs) represent a potential alternative to conventional implants and are expected to reproduce the physiological properties of natural intervertebral discs (ND) as well as to fully integrate into the host disc space.
METHODS: In vitro studies by our group demonstrated the feasibility of synthesizing TE-IVDs constructed of bovine ND cells and type I collagen derived from rat tendons. Subsequently, we implanted the TE-IVDs into the rat caudal spine to evaluate *in vivo* matrix synthesis and mechanical properties. The TE-IVDs were composed of nucleus pulposus (NP) cells seeded in alginate (25 x 106 cells/ml) to form the NP and 1mg/ml type I collagen gel seeded with annulus fibrosus (AF) cells (1 xl06 cells/ml) to form the AF. Discs were implanted in a microsurgical procedure between the 3rd and 4th vertebrae of the rat caudal spine. The animals were sacrificed after 6 months (n=12) and the explanted segments were assessed for their biochemical and mechanical properties. MR imaging from another group of animals (n=8) was obtained at 1, 5, and 8 month time points. The images were used for disc height measurements as well as for qualitative and quantitative analysis of water and proteoglycan content. After 8 months segments were collected for histological analysis and stained with for proteoglycans (Alcian blue), and for collagen (Picrosirius red). Animals undergoing solely a discectomy without implantation served as controls.
RESULTS: After 8 months, qualitative MRI analysis on T2 sequences revealed morphology comparable to native discs, with a hyperintense NP located in the center of the disc space encompassed by a hypointense AF. The discectomy control group showed a black collapsed disc space. Quantitative analysis according to T2 relaxation time measurements revealed hydrated NP tissue with NP volume dropping between 1 and 5 months and remaining constant from 5 to 8 months. The discectomy control group showed no signs of hydrated NP tissue in the disc space. According to T1p relaxation time, the NP of the implanted discs showed properly distributed proteoglycan content with higher values in the NP region compared to the AF. ND height dropped initially but was maintained throughout the study duration, measuring 72% of normal disc height at 1 month, 66% at 5 months, and 69% at 8 months. Simple discectomy led to a rapid collapse to 51% of initial height. Histological sections after 8 months showed that the AF of the TE-IVDs consisted of spindle-shaped cells resembling fibroblasts and a dense parallel-aligned fiber structure encapsulating the nucleus. The NP cells were embedded in an amorphous matrix located predominantly at the AF/NP border and appeared chondrocyte-like. Polarized light micro-scopy revealed the AF fibers of the TE-IVDs had infiltrated the endplate of the vertebrae, demonstrating implant integration with the host tissue. Biochemical analysis after 6 months showed that the TE-IVDs contained collagen and proteoglycan distributions similar to those of the native AF and NP. The overall collagen and proteoglycan content of the TE-IVDs showed no significant difference compared to native NDs. Mechanical tests after 6 months revealed that motion segments containing the engineered discs had an average equilibrium modulus and hydraulic permeability similar to intact native segments.
CONCLUSIONS: This study demonstrates for the first time that TE-IVDs are viable for up to 8 months *in vivo* and integrate effectively with the surrounding vertebral bodies. TE-IVDs restore function to the rat spine as indicated by mechanical assessments and maintenance of disc height. Proteoglycan and collagen synthesis as well as the development of native disc-like morphology are promising parameters and indicate the functionality of the implants. Our current work aims at expanding these results by utilizing different cell sources such as mesenchymal stem cells for disc engineering. We also realize the limitations of the rat model and are currently studying TE-ND in a dog model.

SATURDAY, OCTOBER 20

8:30-8:40 SKIN-DERIVED PRECURSOR SCHWANN CELL THERAPY IMPROVES BEHAVIOURAL OUTCOME FOR BOTH IMMEDIATE AND DELAYED NERVE REPAIR

Helene Khoung, MD, FRCSC and ***Rajiv Midha, MD, FRCSC, FAANS,*** *Univ of Calgary*

BACKGROUND AND HYPOTHESIS: Previous work has shown that infusion of skin-derived precursors pre-differentiated into Schwann cell (SKP-SCs) within both nerve gap and chronically denervated models of nerve injury can improve indices of axonal regeneration and electrophysiological parameters. We hypothesized that SKP-SCs therapy would improve behavioural outcomes for nerve injury repair.
METHODS: A total of 61 adult male Lewis rats were trained prior to surgical intervention to perform a validated skilled locomotion task (horizontal ladder-rung). A right tibial nerve transection was performed just distal to the sciatic nerve trifurcation. The animals were divided in different groups within 2 separate arms of the study. One arm (ACUTE repair) involved an immediate direct repair model. One group (n=10) received an injection of SKP-SCs (500,000) 3 mm distally to the repair site at the time of the surgery, another group (n=10) received an injection of the same volume of carrier medium and a third group (n=5) received an injection of same number of dead SKP-SCs. 15 animals (5/group) were followed for 8 weeks, while 10 animals (5 with SKP-SCs and 5 with carrier medium) were followed for 17 weeks. Additional animals were included for histomorphometrical analysis at 4 weeks, and received bilateral surgeries (total of 16 surgeries) with injection of the same adjuvant therapy as above or no injection (4 nerves/group). The other arm of the study evaluated a DELAYED repair paradigm in 24 rats. After an initial right tibial nerve transection, both nerve ends were capped to undergo chronic denervation. Eleven weeks later, the caps were removed and the nerve was directly repaired. Group (n=8, each) received an injection of SKP-SCs, carrier medium or dead cells 3 mm distal to the repair site at delayed surgery and also 3 weeks later. All animals were followed for 9 more weeks after the delayed repair, for a total of 20 weeks. Common control groups (n=6, each) underwent sham surgery or chronic denervation (transaction injury and capping throughout without repair). All animals were serially tested for skilled locomotion on the horizontal ladder-rung at defined time-points over the duration of the study (8, 17 or 20 weeks) and video-recorded for frame by frame analysis. A slip ratio was calculated as the number of times the injured hindlimb slipped (between the rungs on the horizontal ladder) over the total number of steps. At study termination, a sample of tibial nerve was harvested distal to the repair site for histomorphometrical analysis for number of myelinated axons, fiber diameters, G-ratios, percent neural tissue, number of myelin debris complexes and percent myelin debris.
RESULTS: Baseline slip ratios were similar across all groups, at 2-5%. All animals that had undergone a nerve injury had a rise in slip ratio one week after, above 50%. In the ACUTE repair arm, the group with SKP-SCs showed marked improvement in performance as early as 5 weeks after surgery. The groups that received media and dead SKP-SCs both evolved with a much slower progression. In the DELAYED repair arm, all 3 groups showed an elevated slip ratio prior to their repair surgery. The SKP-SCs group progressively improved after surgery, becoming significantly better than the non-repair group 7 weeks after the repair, while the media and the dead SKP-SCs showed no significant improvement for the study duration. In the immediate repair arm, after 4 weeks, the group with SKP-SCs showed an increased axon count and percent neural tissue, coupled with significantly lower myelin complex count and percent myelin debris when compared with dead SKP-SCs injection. In the delayed repair arm, axon count and percent neural tissue were significantly higher in the SKP-SCs group when compared with the media and the dead SKP-SCs groups.
CONCLUSIONS: SKP-SCs therapy improves behavioural recovery in both acute and chronic nerve repair. It may do by enhancing (inhibitory) myelin clearance, thereby making the nerve more hospitable to accelerated axonal regeneration in the denervated nerve microenvironment.

SATURDAY, OCTOBER 20

8:42-8:52 NOVEL NANOVECTOR NANOSYRINGE DELIVERED DRUG PUMP INHIBITORS (CERBERUS) POTENTIATE THE ACTION OF NANOVECTOR-DELIVERED CCHEMOTHERAPY (HADES) IN CULTURED PRIMARY HUMAN GLIOBLASTOMA

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INTRODUCTION: Glioblastoma (GBM) remains a challenge to treat, as our best efforts with surgery, radiation therapy, and chemotherapy only provide 12-15 months of survival in most patients. One of the limitations of standard chemotherapy is the interaction with systemic organ systems and subsequent toxicity. The development of targeted delivery systems is a way to overcome this limitation. However, in addition, the tumor itself has its own defenses against chemotherapeutic drugs in the form of drug resistance pumps. These pumps are located on the plasma membrane of the glioma cells, and pump the chemotherapy molecules out of the cell before they strike a lethal blow to the cells. The heterogeneous upregulation of a variety of different drug pumps, such as MDR1 and others in GBM is well documented. We postulated that co-therapy with non-toxic drug pump inhibitors using nanovector nanosyringe based delivery should be able to sensitize drug resistant GBM to chemotherapeutic agents such as docetaxel (Doc) and vinblastine (Vin). Haloperidol (Halo) is a potent inhibitor of the MDR1 pump, which detoxifies both Doc and Vin. Indomethacin (Indo) is less specific, inhibiting the BCRP pump, as well as MRP1 and MRP2. These three pumps are typically found in high levels in GBM and the latter pair are very efficient at exporting Vin from GBM. Primary GBM's have heterogeneous expression of at least four xenobiotic pumps that provide drug resistance to Doc&Vin; one pump common to both Doc&Vin, the Halo sensitive MDR1 pump, and three Indo sensitive pumps, the BCRP (Doc) and MRP1 & 2 (Vin). We have studied the use of nanosyringe delivered pump inhibitors targeting these specific systems.
METHODOLOGY: Primary human GBM cell cultures were grown in 96 well plates and were treated using previously described drug carrying nanovector constructs using nano sized hydrophobic carbon clusters (HCC's) These can be targeted to the surface of primary human GBM cell cultures using mouse monoclonal antibodies that target specific cell surface epitopes, which we have called HADES (Hydrophobic Carbon Cluster Drug Enhancement Delivery System). Doc and Vin were used as HADES therapy as previously described; both at 100 nM. Doc/Vin-HADES resulting in ≈30%/50% cell death respectively. 2μM Halo/Indo-PEG-HCC was added to cells in the absence and presence of IgG. Continuing with the Greek mythological eponyms, we have called the targeted pump inhibitor delivery system, CEREBRUS, who was Hades' three headed dog.
RESULTS: We have previously demonstrated using the chemotherapeutic compounds Doc, Vin, and SN-38, that drug loaded HCCs show minimal transfer of their hydrophobic cargo into the bulk phase of cell culture media unless a cell surface targeting antibody is added to the construct. This slow rate of transfer of hydrophobic drug is confirmed herein using the drug pump inhibitors Halo and Indo. When pump inhibitor loaded PEG-HCC without the specific antibody against a cell surface epitope was added to cells dosed with Doc/Vin-HADES, there was no increase in the toxicity of these chemotherapeutic agents. However, when delivered as the targeted form, Halo/Indo- CERBERUS, in the presence of Doc/Vin-HADES, we recorded a large increase in cell death. Halo-CEREBRUS potentiated Doc/Vin-HADES equally, effectively doubling their toxicity, by eliminating the ability of the MDR1 pump to export these drugs. Indo-CEREBRUS increased the lethality of Vin-HADES by 70%, due to the blocking MRP1 and MRP2 Vin transport. Doc-HADES toxicity was also potentiated by Indo-CERBERUS by 30%, probably due to inhibition of the BCRP pump.
CONCLUSION: The ability to transport drug compounds to the surface of cancer cells is a way to widen the therapeutic window of chemotherapeutic agents. We show that in addition to specific targeting of cancer cells using HADES, we can widen the therapeutic window by the use of targeted drug pump inhibitors. These relatively nontoxic compounds have the ability to raise the steady state level of chemotherapeutics in targeted cells. We envisage a patient-centered personalized treatment regime based on the co-delivery of drugs with pump inhibitors to cancer cells. We would identify the up-regulated surface receptors and pumps present in a GBM biopsy and, based on specific pattern in a patient's tumor, chose which antigens and pumps to target using both HADES (the primary targeted nanosyringe chemotherapy delivery system) and CERBERUS (the targeted nanosyringe pump inhibitor delivery system). These constructs have potential widespread applications for treatment of any cancer.

SATURDAY, OCTOBER 20

8:54-9:04 RAPID AND SPECIFIC DIAGNOSIS OF ASTROCYTIC TUMORS USING IMMEDIATE *EX VIVO* SR101 CONFOCAL MICROSCOPY

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INTRODUCTION: Surgical resection of brain tumors is guided by intraoperative diagnosis. Unfortunately, current intraoperative diagnostics lack specificity for some common diagnoses that require conflicting treatment plans, and receiving the final diagnosis can take one or more days. We hypothesized that rapid fluorescence staining and imaging of human brain tumor biopsies with the live-cell fluorophore Sulforhodamine 101 (SR10l) could accurately provide specific identification of astrocytic tumors in a time frame that could support intraoperative decision-making.

METHODOLOGY: Fluorescence imaging of SR101-labeled cell cultures and acute slices prepared from human astrocytoma and lymphoma rodent xenografts was first performed. A variety of astrocytic and non-astrocytic human brain tumor specimens obtained at surgery were then immediately incubated with SR101 and analyzed by confocal microscopy. Immunohistochemical confirmation of staining patterns was confirmed by quantification and SR101 1abeling of human samples was compared to final pathological diagnosis. A rapid incubation and imaging protocol was instituted to test whether results could be obtained in under 20 minutes.

RESULT: SR101 showed specific marking of astrocytic tumors in cell culture, animal xenografts, and human specimens. In the human specimens, all grades of astrocytoma showed SR101 positivity. The rapid-staining protocol differentiated astrocytic tumors and their margins from other brain tumors and normal brain and allowed the distinction of tumor cells from reactive astrocytes. We confirmed specificity of SR101 by immunohistochemistry for both rodent xenografts and with final pathological diagnosis obtained for the human samples. In an improvement to current time-consuming diagnostic techniques, SR101 allowed rapid differentiation within 20 minutes of two central nervous system tumors that require polarized surgical interventions: glioma and central nervous system lymphoma. Furthermore, SR101 differentiated human astrocytomas from oligodendrogliomas, another rapid diagnosis not possible with currently available stains.

DISCUSSION: Coupled with fluorescence imaging, SR101 staining can provide a rapid and specific confirmation of astrocytic lineage in human brain tumors. To our knowledge, this is the first example of use of a clinical application of a f1uorophore for the diagnosis of human brain tumors. SR101 may be the first of many fluorophores that will show utility as clinical diagnostic agents.

SATURDAY, OCTOBER 20

9:06-9:16 CYTOKINES AT THE CROSSROADS OF BRAIN INJURY AND REPAIR: GALECTIN-3, A POTENTIAL TARGET FOR ENHANCING INJURY REPAIR AND RECOVERY FOLLOWING ISCHEMIC STROKE.

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Raghu Vemuganti, PhD, Associate Professor, Univ of WI, Dept. of Neurological Surgery
***Robert J. Dempsey, MD**, Professor & Chairman, Univ of WI, Dept. of Neurological Surgery*

Focal brain ischemia initiates transient and inefficient self-repair mechanisms including the production of neurotrophic factors and cytokines. Enhancing these self repair mechanisms requires an understanding of factors and molecular events that regulate these functions. Galectin-3 (Gal-3) is a cytokine with potential for protecting against brain ischemic injury. We have previously demonstrated that expression of cytokines including Gal-3, a carbohydrate binding protein is significantly upregulated in the ipsilateral hemisphere of ischemic brain of rats subjected to transient middle cerebral artery occlusion (MCAO). Furthermore, blocking of Gal-3 function through intra-cerebroventricular infusion of Gal-3 antibody decreased the microvessel density in ischemic brain. In the current study we show that Gal-3 in a concentration dependent manner significantly increases the viability/survival of microglia BV2 cells under *in vitro* ischemic conditions of oxygen glucose deprivation and re-oxygenation. Importantly, addition of exogenous Gal-3 promoted the formation of pro-angiogenic structures in an *in vitro* human umbilical vein endothelial and BV2 co-culture model. Gal-3 induced angiogenesis was associated with increased expression of vascular endothelial growth factor. Gal-3 also augmented the *in vitro* migratory potential of BV2 microglia. Gal-3 mediated functions were mediated through the increased levels of integrin-linked kinase (ILK) signaling as demonstrated by the impaired angiogenesis and migration of BV2 cells following targeted silencing of ILK expression by SiRNA. Furthermore, we show that phos-AKT and ERK1/2 are downstream effectors of Gal-3-ILK pathway. Finally, our initial studies in a clinically relevant animal model demonstrated that exogenous Gal-3 decreases the infarct size and promotes functional recovery as indicated by postural reflex test. Taken together, our studies indicate that cytokines including Gal-3 are potential targets for enhancing injury repair and functional recovery after ischemic stroke.

SATURDAY, OCTOBER 20

9:18-9:28 ADVANCED MRI IMAGING OF THE EPIDERMAL GROWTH FACTOR RECEPTOR (EGFR): FROM NONINVASIVE DETECTION TO PREDICTION OF GLIOBLASTOMA RECURRENCE.

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We have utilized advanced MRI imaging (aMRI) paradigms in the diagnosis and treatment of EGFR glioblastomas as part of an effort to noninvasively define glioblastoma subtypes. An example of this work has been in the noninvasive detection of the epidermal growth factor receptor variant III (*EGFRvIII*) mutation in glioblastoma. *EGFRvIII* is overrepresented in the "classical" GBM subtype and its identification has become increasingly relevant in the optimization of therapy. We assessed the accuracy of magnetic resonance perfusion-weighted imaging (MR-PWI) in discriminating the *EGFRvIII*-expressing glioblastoma subtype. In our extensive GBM database, *EGFRvIII*-expressing glioblastomas showed significantly higher rTBV, as determined by MR-PWI, compared to those tumors lacking *EGFRvIII* expression. By logistic regression analysis, rTBV has been shown to be a very strong predictor of *EGFRvIII* mutation (OR(rTBV)=2.70, p=0.000). Further, rTBV discriminated *EGFRvIII* with very high accuracy (A_z=0.81). A more recent extension of this work has been to use the temporal dynamics of MR-PWI, along with advanced statistical image analysis methods, and genomics, to predict peri-lesional edematous tissue that is likely to represent a focus for tumor recurrence. We have utilized MR perfusion images, and EGFRvIII mutational status, to predict regions that show a higher probability of recurrence. An early cohort of nineteen GBM patients was studied over time from initial resection to tumor recurrence. T1, T1CE, T2, FLAIR, and MR-PWI images were co-registered. Regions of interest (ROIs) were drawn for each subject on images before surgery in white matter, gray matter, CSF, edema, enhancing tumor, nonenhancing tumor and in regions of necrosis. Principal component analysis (PCA) was utilized to extract the uncorrelated variables that reflect the temporal dynamics of MR-PWI. Cross validations were utilized to build the PCA model from a training set and test it on new patients. The results show marked separation between edematous regions that recurred and edematous regions that did not recur, thereby indicating that predictive imaging biomarkers can be constructed using aMRI and analysis methods. Inclusion of *EGFRvIII* mutational status markedly enhanced the predictive accuracy of the model. In summary, MRI-PWI can be used to reliably predict mutational status of *EGFRvIII* in classical glioblastomas and *EGFRvIII* status can enhance a predictive model of glioblastoma recurrence derived from the dynamics of MRI-PWI imaging. These data suggest that radiogenomic efforts can improve diagnostic detection of glioblastoma mutations that may lead to more accurate and focused treatment planning.

SATURDAY, OCTOBER 20

9:30-9:40 PRELIMINARY STUDIES OF A HUMAN SPINAL CORD MODULATIOIN SYSTEM

***Matthew A. Howard III, MD**, Professor and Head, John C. Van Gilder Chair, Department of Neurosurgery, Univ of Iowa*

A wide range of neurological disorders could in theory be treated by selectively modulating neural structures within the human spinal cord. The therapeutic potential for spinal cord modulation strategies is reflected in the results of numerous experimental animal research studies. Because of the unique anatomy and biomechanical properties of the human spinal cord, however, there are major methodological barriers preventing translation of laboratory-based neuromodulation concepts to effective clinical treatments. A Human Spinal Cord Modulation System (HSCMS) is being developed to overcome these barriers and enable clinicians and researchers to selectively modulate targeted neural pathways within the human spinal cord.

Currently, there are no clinical devices that deliver electrical stimuli directly onto the spinal cord. Existing stimulators are all placed outside of the dural lining of the spinal canal. Because of the electrical shunting effect of the highly conductive cerebrospinal fluid within the spinal canal, these stimulators can only activate a small number of axons located within 300 µm of the dorsal surface of the spinal cord. This leaves >99% of the neural structures within the human spinal cord inaccessible to neuromodulation treatment strategies, including pathways postulated to be ideal targets for therapeutic interventions.

The HSCMS is designed to overcome this limitation by safely delivering electrical stimuli directly to the spinal cord. Prototype HSCMS devices were designed and fabricated in partnership with medical device industry technology partners. The first generation devices will be positioned on the pial surface of the spinal cord. The material and stimulus delivery characteristics of the portion of the device in direct contact with the spinal cord closely resembles the electrode bearing portion of the Auditory Brainstem Implant (ABI) currently used to restore hearing in deaf patients who cannot benefit from a cochlear implant. Other HSCMS design features include device elements that maintain the patency of the dural sac, insure safe and stable positioning of the electrodes without disrupting normal movements of the spinal cord, and a mechanism for surgical closure that addresses the risk of CSF leaks.

A range of pre-clinical studies have been performed using bench top surrogate spinal cord models, finite element computer models of device and spinal cord movement properties, as well as in-vivo device implantation and electrophysiology experiments performed in sheep. The results to date demonstrate the technical feasibility and electrophysiological efficacy of the HSCMS concept. Additional chronic implantation experimental animal studies are required before proceeding with a human pilot clinical study.

SATURDAY, OCTOBER 20

9:42-9:52 THE ADULT BAY AREA GLIOMA STUDY, A FOLLOW-UP

Mitchel S. Berger, MD, UCSF, Margaret Wrensch, PhD, UCSF. John Wienke, PhD, UCSF

The overarching goal of the ongoing Univ of California San Francisco Adult Glioma Study has been discovery and understanding of new etiologic and prognostic factors for glioma. With respect to etiology, we conducted among the first genome wide association studies which have identified inherited risk loci (SNPs) for glioma in 7 regions. Inherited variants in chromosome 8q24 and 11q23 increase risk for isocitrate dehydrogenase *(IDH)* mutated glioblastoma but not for *IDH* wildtype glioblastoma. We have identified a putative causative risk locus for oligodendroglial and *IDH* 1/2 mutated gliomas in the 8q24.21 region with odds ratio (OR) = 5.12, $p=1.1x10^{31}$ and OR= 4.77, $p=6.6\ x10^{22}$, respectively. Strong associations also were observed for *IDH1/2* mutated astrocytomas (grades 2-4) (OR =5.16-6.66; $p=10^{12}$ to 10^{8}, but not for *IDH112* wild-type astrocytomas. Regarding survival, we and others have consistently shown that patients have better survival if their tumors contain *IDH* mutations. Furthermore, in the first ever genome wide survival study for glioblastoma, we identified inherited SNPs associated with survival among GBM patients who received the current standard of care treatment (resection, radiation, and temozolomide). Tumor expression of the identified genes also was shown to be associated with poorer patient survival. Interestingly, the SNPs associated with survival are in different genes than the SNPs associated with the glioma risk. Future work aims to understand the functional significance of risk and prognostic loci, and to discover and validate additional genetic variants associated with risk and survival for patients with glioblastoma and grade II and Ill glioma.

SATURDAY, OCTOBER 20

10:17-10:27 PATIENT-SPECIFIC MODELING FOR PEDIATRIC CRANIOFACIAL RECONSTRUCTION

James Drake, MBBCh[*,**], *Nikoo Saber, PhD***, *Thomas Looi, MSc***, *Johnathan Berge, MD **, *John Phillips, MD*[*,**]

**Hospital for Sick Children Univ of Toronto; ** Centre for Image Guided Innovation and Therapeutic Intervention, Hospital for Sick Children, Toronto*

BACKGROUND: Patient-specific modeling facilitates preoperative evaluation and planning, intraoperative surgical technique, and post operative evaluation. It also provides a platform for education, training, and surgical skills assessment or certification. We have developed a technique for patient-specific modeling for anterior cranial vault reconstruction by creating a library of normalized age-matched cranial head shapes, fabricating a patient specific bandeau template for intraoperative use, and evaluating post operative results.

METHODS: A normative skull library for patients aged 8-12 months was created from 103 "normal" CTs, creating a series of 3D point clouds of normal head shape. An external cranial surface was subsequently passed through the point cloud averaged over the entire age range and its shape and size customized to fit the head circumference of individual patients undergoing anterior cranial vault reconstruction. The suprafrontal orbit anatomy was extracted to fabricate a bandeau template to guide intraoperative reshaping. The same virtual bandeau was used to evaluate postoperative results, by measuring the head shape error as an "area under the curve" between the virtual bandeau in preoperative and post operative CT scans.

RESULTS: The patient-specific normative head shape allowed for preoperative planning in 15 cases of anterior cranial vault synostosis, predominantly metopic or uni-coronal. Intraoperative use of the bandeau template facilitated objective orbital bandeau reconstruction. Post operative CT results were compared to 23 age-matched patients with metopic or uni-coronal synostosis where the bandeau was not used. Use of the bandeau resulted in a significant reduction in OR time, (218 vs 258 minutes, p .001) and a significant improvement in head shape (reduction of 69% vs 56%, p .02 in area under the curve).

CONCLUSIONS: Patient-specific modeling for anterior cranial vault reconstruction can enhance surgical planning, teaching, and improve outcomes, with reduced operating room times. It may also ultimately provide standardized objective outcomes for anterior cranial vault surgery.

SATURDAY, OCTOBER 20

10:29-10:39 LONG-TERM OUTCOME OF SELECTIVE DORSAL RHIZOTOMY FOR CEREBRAL PALSY SPASTICITY

A. Graham Fieggen MBChB MSc MD FCS(SA); *Nelleke G Langerak BSc MSc PhD; Anthony Figaji MBChB MMed PhD FCS (SA); Jonathan C Peter MBChB FRCS*

Division of Neurosurgery, Univ of Cape Town, Cape Town, South Africa

INTRODUCTION
Dorsal rhizotomy was found to be effective in treating spasticity a century ago but failed to gain wide acceptance due to a high rate of complications. Following modifications to the technique by Peacock in Cape Town in the early 1980's, Selective Dorsal Rhizotomy (SDR) has been found to be a safe and cost-effective treatment option for spasticity in cerebral palsy. As the procedure is typically performed on children, it is important to establish the long-term outcome, particularly the impact on quality of life.

METHODOLOGY
A cohort of 13 patients who have been followed up prospectively since 1985 was evaluated using 2D gait analysis and assessment of their functional status, compared to their preoperative data. In a further observational study, 32 out of 47 patients who had undergone SDR between 1981 and 1991 and were eligible for inclusion were assessed with respect to their current levels of activity and participation (Functional Mobility Scale and LIFE-H Questionnaire), complications such as spinal deformity and gait status using 3D gait analysis.

RESULTS
Improvements in gait pattern were maintained more than 20 years after surgery in the prospective cohort. This was accompanied by sustained improvements in functional status compared with preoperative data.

In the observational cohort, independent ambulation was possible for 5m in 84% and for 50m and 500m in 61%. Patients reported high levels of satisfaction with their outcome and 80% were able to accomplish all life habits. As was standard at the time, all patients had undergone at least a four-level laminectomy and although spinal deformity was more prevalent on long-term follow-up than it had been at 4 years postoperatively, no patient had required corrective surgery. 3D gait analysis confirmed that all walked with a mild crouch gait while 58% had improved in their GMFCS level and none had deteriorated.

CONCLUSIONS
SDR is effective in reducing spasticity in cerebral palsy and this is maintained for at least two decades after surgery. Objective improvements in gait pattern are sustained and patients experience a positive impact on their functional status and quality of life. Spinal complications appear to be infrequent, but this data needs to be compared with a matched group of patients who have not undergone SDR.

SATURDAY, OCTOBER 20

10:41-10:51 VOLUME-STAGED GAMMA KNIFE RADIOSURGERY FOR LARGE ARTERIOVENOUS MALFORMATIONS

Michael W. McDermott M.D.,[1,2] *Zachary A. Seymour M.D.,*[1] *David A. Larson M.D,*[1,2] *Nalin Gupta M.D.,*[2] *Michael Lawton M.D.,*[2] *William L. Young,*[2] *and Penny K. Sneed M.D.*[1]
Departments of Radiation Oncology[1] *and Neurosurgery*[2] *at Univ of California at San Francisco, San Francisco, California, USA.*

INTRODUCTION*:* Stereotactic radiosurgery (SRS) is a safe and efficacious option in selected patients with arteriovenous malformations (AVM), particularly small sized lesions where the three year obliteration rate for AVMs <10cc is 70-95%. Large AVMs remain difficult to treat. A rationale for volume stage SRS has been detailed previously as a way to potentially increase rates of obliteration and decrease complications though reports have been limited and rates of obliteration have varied between 33-74%. At Univ of California, San Francisco (UCSF), we have treated large AVMs with volume-staged radiosurgery for 20 years. In 2004, we changed our treatment paradigm, avoiding pre-SRS embolization, using smaller volumes (<7cc) and higher dose per stage (>15 Gy), and favoring salvage resection as soon as it was considered safe instead of waiting for complete obliteration in attempts to decrease interval from date of initial treatment to cure.

METHODS*:* All patients with planned volume-staged SRS for AVM were retrospectively reviewed. Post-SRS patients were followed with annual MRI and angiogram at 3yr after SRS. Response was evaluated volumetrically by a product of three-dimensions, with "no response" <25% reduction, "partial response" ≥25% reduction, "near obliteration" ≥ 75% reduction in nidus volume on MRI or angiogram. "Complete obliteration" classification was used only in cases where angiogram confirmed total obliteration. Other factors, such as SRS score, Spetzler-Martin Score, flair response and nidus architecture, were also evaluated. Patients were followed clinically to assess modified-Rankin Score (mRS).

RESULTS*:* A total of 69 patients were planned for volume-staged SRS and 62 completed all stages. 78% of patients had Spetzler-Martin grade 4-5 AVMs, and SRS score ranged from 1.82-7.95 (median, 3.46). For era 1 (1991 through April 2004), the median total target volume was 27.3 ml (range, 13.5-68.0 ml), treated volume 15.0 ml (7.1-38.7 ml) per stage, and dose 15.5 Gy (range, 12.0-19.0 Gy). For era 2 (May 2004 through 2009), the median total target volume was 18.9 ml (range, 8.6-65.9 ml), treated volume 6.8 ml (4.3-14.5 ml) per stage, and dose 17.0 Gy (range, 16.0-18.0 Gy). 14 patients had later salvage including surgery (5), SRS (2), SRS and surgery (5), embolization (1), or embolization and SRS (1). Eleven patients died 0.3-7.0 yr after SRS; the median follow-up was 3.9 yr in the remaining patients. Survival probability was 83% at 5 yr and 75% at 10 yr. 23 AVM hemorrhages occurred in 18 patients post-SRS resulting in 9 deaths; an additional patient died of an aneurysm hemorrhage and one died from seizure. For era 1, there were 4 complete obliterations, 5 near obliterations, 14 partial responses, 5 non-responses, and 10 patients without imaging follow-up. For era 2, there were 2 complete obliterations, 10 near obliterations, 10 partial responses, 3 non-responses, and 6 patients without imaging follow-up to date. Ten additional complete obliterations were achieved after salvage therapy, for an ultimate complete obliteration rate of 23% considering all 69 patients and 36% among patients with at least 1 yr of imaging follow-up. The 3-yr actuarial probability of at least partial response to staged SRS (not including salvage therapy) was 75% for era 1 vs. 95% for era 2. The 5-yr actuarial probability of cure including salvage therapy was 6% for era 1 vs 53% for era 2. The mean mRS increased from 1.3 preSRS to 2.3 postSRS.

CONCLUSION*:* Volume-staged SRS is a viable option for large AVMs. We recommend ~7-8 ml per stage and patients be assessed for surgery or repeat SRS in the likely event of residual nidus ~3-4 yr after completion of volume-staged SRS.

SATURDAY, OCTOBER 20

10:53-11:03 SUPERIORLY PROJECTING ANTERIOR COMMUNICATING ANEURYSMS: MICROSURGICAL TREATMENT WITH FENESTRATED CLIPS AND ADJUNCTIVE ICG ANGIOGRAPHY

David Chalif MD, FACS
Chief, Neurovascular Neurosurgery, North Shore Univ Hospital, Department of Neurosurgery, Hofstra-North Shore-LIJ School of Medicine

INTRODUCTION: Superiorly projecting Anterior Communicating Artery (ACoA) aneurysms pose a distinct microsurgical challenge due to frequent incorporation of proximal A2 vessels, association with perforators, and obscuration of the aneurysm fundus by the ipsilateral A2 segment. A variety of microsurgical clipping techniques are useful for these lesions. Aneurysm remnants may be left after clipping attempts with standard side-angled or curved clips. The most efficacious technique is the use of fenestrated clip(s) placed with blades parallel to the ACoA artery. Parallel and multiple fenestrated clips can achieve reconstruction of the proximal A2 segment. This clipping modality coupled with ICG angiography has been used effectively for treatment of these lesions. The technical approach to superiorly projecting ACoA aneurysms was reviewed for this study.

METHODS: A retrospective analysis was performed reviewing a single surgeon's experience using fenestrated clip(s) for both ruptured and unruptured superiorly directed ACoA aneurysms. A sub-set of this group was evaluated for efficacy of the use of fenestrated clips with adjunctive intra-operative ICG angiography.

RESULTS: 1389 aneurysms were treated by direct microsurgical clipping by a single surgeon over a 27-year period. Out of this series, 334 were at the ACoA. Of this cohort, 23 cases projected superiorly. Reconstruction strategy employed one or more fenestrated clips typically involving wide fenestrations and short blades. Structures incorporated into the clip fenestration included the proximal A2 segment, the distal ipsilateral A1 segment, the A1/A2 junction, the recurrent artery of Heubner, and an orbitofrontal branch. Adjunctive intra-operative ICG angiography in the latter half of the series demonstrated persistent filling leading to immediate clip adjustment in 2 cases. Aneurysm remnant, not visualized on ICG, was identified on post-operative angiography in one patient. In all other cases post-operative cerebral angiography confirmed complete aneurysm obliteration and patency of vascular structures within the fenestration.

CONCLUSION: In the era of advances in endovascular techniques – inclusive of stent-assisted coiling and flow diversion – superiorly projecting ACoA aneurysms frequently remain in the realm of microsurgical treatment. The combined use of fenestrated clips with adjunctive ICG angiography can lead to excellent reconstruction of the normal vascular angio-architecture of the region and preservation of perforators with good clinical outcomes

SATURDAY, OCTOBER 20

11:05-11:15 COMPARISON BETWEEN THE CLINICAL EFFECTS OF HYROXYAPATITE CEMENT AND TITANIUM MESH DURING CRANIOTOMIES IN THE RETROMASTOID APPROACH

Mohamed A Ragaee, MD[2] *, Khaled M. AbdelAziz, MD, PhD[1], Raymond F. Sekula, MD[3], Lynn H. Fletcher, RN[1], Daniel J. Cook, MS[1], Andrew M. Frederickson, BS[3], Mohab M. Nageeb, MD[5], Gregory D. Arnone, MD[4], Boyle C. Cheng, PhD 1, Peter J. Jannetta, MD 1/* *1Departments of*Neurosurgery, Allegheny General Hospital, Drexel College of Medicine, Pittsburgh, PA, - Department of Neurological Surgery, Assiut University Hospital, Assiut, Egypt, 3- University of Pittsburgh Medical Center Hamot, Erie Pennsylvania, 4- Department of Neurosurgery University of Illinois at Chicago, Chicoago Illinois*
5- Department of Neurosurgery, Minia University, Minia, Egypt

INTRODUCTION/HYPOTHESIS:
The Retromastioid craniotomy (RMC) is the principal neurosurgical approach for intradural posterior fossa pathology. Craniotomy for microvascular decompression of cranial nerves and the brainstem are proven therapies for a broad range of clinical conditions including trigeminal neuralgia, hemifacial spasm, disabling positional vertigo and vascular tinnitus. Following dural closure, cranioplasty is performed to minimize dural attachment to the suboccipital musculature, enhance cosmetic appearance and minimize the risk of postoperative cerebrospinal fistula.
We used the titanium cranial mesh and the hydroxyapatite cement as materials for cranioplasty after retromastoid craniotomy. This was a retrospective study that was comprised of 2 groups: Study group 1: hydroxyapatite cement (n=l50), Study group 2: titanium mesh (n=l50) All Subjects who underwent a retromastoid craniotomy and fulfilled the Inclusion criteria of the study and could be contacted were included in the study Patients were contacted and asked 4 questions answered by Yes or No in regards to the following areas:

An infection of the incision
Cerebrospinal fluid leak post operative
Incisional pain that required another surgical procedure
Patient satisfaction with appearance of surgical site

We also compared between the cost and the intraoperative length of time for both procedures.
RESULTS:
After analyzing the data from both groups statistically using Pearson's chi-squared test, statistically significant difference was not detected between the use of hydroxyapatite cement and the use of titanium mesh in cranioplasty for the retromastoid craniotomy patients. Also we found that the hydroxyapatite cement is generally more expensive and takes more time in the operative room than he titanium mesh
CONCLUSIONS:
We did a literature search and to the best of our knowledge we could not find any other research that was previously published that compared between the use of titanium mesh and hydroxyapatite cement for cranioplasty. No statistically significant difference was detected between the titanium mesh cranioplasty group and the hydroxyapatite cement cranioplasty group Using titanium mesh in cranioplasty gave the same results as hydroxyapatite cement cranioplasty. In terms of cost difference between the two , we found that hydroxyapatite cement is generally more expensive and usually increases the 0R time, which adds to the overall expense

SATURDAY, OCTOBER 20

11:17-11:27 SUPERIORLY PROJECTING ANTERIOR COMMUNICATING ANEURYSMS: MICROSURGICAL TREATMENT WITH FENESTRATED CLIPS AND ADJUNCTIVE ICG ANGIOGRAPHY

David Chalif MD, FACS
Chief, Neurovascular Neurosurgery, North Shore University Hospital, Department of Neurosurgery, Hofstra-North Shore-LIJ School of Medicine

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CONCLUSION: In the era of advances in endovascular techniques – inclusive of stent-assisted coiling and flow diversion – superiorly projecting ACoA aneurysms frequently remain in the realm of microsurgical treatment. The combined use of fenestrated clips with adjunctive ICG angiography can lead to excellent reconstruction of the normal vascular angio-architecture of the region and preservation of perforators with good clinical outcomes

SATURDAY, OCTOBER 20

11:29-11:39 EXOSOMES FROM GLIOMA-ASSOCIATED MESENCHYMAL STEM CELLS MODULATE THE PROLIFERATION OF GLIOMA STEM CELLS

Frederick F. Lang, MD, FACS, FAANS *– The University of Texas MD Anderson Cancer Center, Department of Neurosurgery, Houston, TX 77030, USA*
Javier Figueroa – The University of Texas MD Anderson Cancer Center, Department of Neurosurgery, Houston, TX 77030, USA
Anwar Hossain, PhD – The University of Texas MD Anderson Cancer Center, Department of Neurosurgery, Houston, TX 77030, USA

Current knowledge of glioblastoma multiforme (GBM) stems largely from work on Glioma Stem Cells (GSCs), which generate neuro-spheres in vitro and infiltrative tumors in vivo. However, the interactions of GSCs with the tumor niche have been largely ignored. In this context, we have recently isolated mesenchymal-like stem cells from the microenvironment of human gliomas and have shown that these so called Glioma Associated-human Mesenchymal Stem Cells (GA-hMSCs) can alter the growth of GSCs in vitro and in vivo. However, the mechanism underlying the communication between GA-hMSCs and GSCs is unknown. In this context, recent studies have suggested that nanosized vesicle, called exosomes, may contribute to cellular communication within the tumor niche. However, the contribution of exosomes in the communication between tumor-supporting GA-hMSCs and tumor-forming GSCs and has not been established, and poses an important objective in understanding GBMs. Here we show for the first time that exosomes can be isolated from patient-derived GA-hMSCs and that these exosomes harbor the known exosomal marker, CD-63. Additionally, exosomes from 3 different GA-MSC lines (GA-MSC7-6, GA-MSC230, and GA-MSC247) were found to contain various genetic material, including oncogenic microRNAs (e.g., miR-21 and miR-125b). Transfer of these exosomes was subsequently demonstrated in vitro when GSCs spontaneously absorbed GA-MSC exosomes labeled with a fluorescent membrane dye after 6 hour exposure. Moreover, we found that this in vitro delivery of exosomes isolated from GA-hMSCs increased the proliferation of GSCs by over 50% at 96 hours. Moreover, the assimilation of GA-hMSC-derived-exosomes was enough to alter the intracellular miRNA profile of GSCs, increasing the amount of oncogenic miRNAs in GSCs. Therefore, we conclude that exosomes released by GA-hMSCs may represent an alternative intra-tumoral communication mechanism for the exchange of miRNA, which could significantly impact the tumor microenvironment and enhance the aggressive nature of GBMs in vivo.

SATURDAY, OCTOBER 20

11:41-11:51 SENESCENCE-ASSOCIATED-GENE SIGNATURE IDENTIFIES GENES LINKED TO AGE, PROGNOSIS, AND PROGRESSION OF HUMAN GLIOMAS

***Steven Brem, MD**, Department of Neurosurgery, Perelman School of Medicine, Hospital of the University of Pennsylvania, Philadelphia, PA*

BACKGROUND: Senescence-associated genes (SAGs) are responsible for the senescence-associated secretory phenotype, linked in turn to cellular aging, the aging brain, and the pathogenesis of cancer.

OBJECTIVE: We hypothesized that senescence-associated genes are overexpressed in older patients, in higher grades of glioma, and portend a poor prognosis.

METHODS: Forty-seven gliomas were arrayed on a custom version of the Affymetrix HG-U133 + 2.0 GeneChip, for expression of fourteen senescence-associated genes: CCL2, CCL7, CDKN1A, COPG, CSF2RB, CXCL1, ICAM-1, IGFBP-3, IL-6, IL-8, SAA4, TNFRSF-11B, TNFSF-11 and TP53. A combined "senescence score" was generated using principal component analysis to measure the combined effect of the senescence associated gene signature.

RESULTS: An elevated senescence score correlated with older age ($r = 0.37$; $P = .01$) as well as a higher degree of malignancy, as determined by the WHO histological grade ($r = 0.49$; $P < .001$). There was a mild association with poor prognosis ($P = .06$). Gliosarcomas showed the highest scores. Six genes independently correlated with either age (IL-6, TNFRSF-11B, IGFBP-3, SAA4, and COPG), prognosis (IL-6, SAA4), or the grade of the glioma (IL-6, IL-8, ICAM-1, IGFBP-3, and COPG). The link between SAGs, age, and prognosis was a feature of gliomas, but absent in 4,415 surgical specimens of cancers of the breast, lung, colon, kidney, ovary, prostate, pancreas, bladder, or liver.

CONCLUSION: We report: 1) a novel molecular signature in human gliomas, based on cellular senescence, translating the concept of SAG to human cancer; 2) the senescence signature is composed of genes central to the pathogenesis of gliomas, defining a novel, aggressive subtype of glioma; 3) these genes provide prognostic biomarkers, as well as targets, for drug discovery and immunotherapy.

SATURDAY, OCTOBER 20

11:53-12:03 PROLONGED SURVIVAL IN PATIENTS UNDERGOING AGGRESSIVE SURGERY FOR IDH1-MUTANT MALIGNANT ASTROCYTOMA

Daniel P. Cahill MD, PhD[1]*, Fred G. Barker MD*[1]*, Dima Suki PhD*[2]*, Sujit Prabhu MD*[2]*, Jeff Weinberg MD*[2]*, Frederick Lang MD*[2]*, Ganesh Rao MD*[2]*, Ian McCutcheon MD*[2]*, Ray Sawaya MD*[2]
Massachusetts General Hospital, Boston, MA, [2]*MD Anderson Cancer Center, Houston, TX*

IDH1 mutations have recently been identified in most low-grade gliomas, and in a substantial fraction of high-grade astrocytic gliomas (AA and GBM), where it is associated with improved survival.

We scored 377 cases of high-grade astrocytoma for IDH1 mutation, and analyzed clinical factors associated with better survival and greater extent of resection.

In 33 IDH mutant GBMs that underwent gross total resection (GTR, <1.5cc residual enhancement), median survival was 50 months; 36% of patients were alive at 5 years after diagnosis. For 87 IDH-mutant AAs that underwent GTR, median survival was not reached (median followup >56mo in surviving patients).

Because few IDH mutant tumors received subtotal resections, we investigated IDH1 status as a predictor of resectability. In a multivariate model including Age, Tumor location, Eloquence, Histology, and IDH status, we identified Frontal tumor location (HR=2.6, p=0.028) and IDH1 mutation (HR=2.1, p=0.028) as independent predictors of GTR.

We developed a nomogram to predict IDH1 mutant status using clinical features and classical histology. Age ($p<0.001$), lack of MR enhancement ($p=0.08$), tumor volume ($p=0.02$), and AA diagnosis ($p<0.001$) were significant predictors of IDH mutation. The nomogram allows prediction of the chance of IDH mutation in malignant astrocytomas based on clinically available data. For example, a 30-yr-old patient presenting with a 6 cm enhancing glioblastoma has a 50% predicted chance of IDH1 mutation; a 50-yr-old patient presenting with the same 6 cm enhancing glioblastoma has a 7% chance of IDH mutation.

Taken together, these results suggest that the combination of IDH mutant status and aggressive surgical resection confer a better prognosis on this subset of glioblastoma patients than has been previously reported.

SATURDAY, OCTOBER 20

12.05-12:15 ENHANCED ANEURYSM FORMATION IN PRO-INFLAMMATORY, TRANSGENIC HAPTOGLOBIN 2-2 MICE

Rafael J. Tamargo MD, FACS*, Jacob Ruzevick BS, Christopher Jackson BA, Gustavo Pradilla MD, and Tomas Garzon-Muvdi MD*
All authors are affiliated with the Department of Neurosurgery, Johns Hopkins University School of Medicine, Baltimore, Maryland

INTRODUCTION: The pathophysiology of aneurysm formation is complex and thought to be due to a combination of genetic factors, vascular wall injury, and hemodynamic stress. The role of inflammation in general and of macrophages in particular regarding the evolution and growth of aneurysms remains controversial. The haptoglobin 2-2 (Hp2-2) genotype, which induces a constant pro-inflammatory state, is being increasingly linked to a variety of vascular pathologies. Hp is an abundant serum protein that scavenges extracorpuscular hemoglobin (Hgb) by forming a high affinity Hp-Hgb complex. The Hp-Hgb complex is then endocytosed peripherally by macrophages after binding to their CD163 surface receptor or centrally by parenchymal cells in the liver, where it is metabolized into bilirubin, which is less toxic. We investigated the role of inflammation and macrophages in the formation of aneurysms in a murine aneurysm model using transgenic, pro-inflammatory Hp2-2 mice, and wild-type Hp1-1 controls.

METHODOLOGY: Carotid artery aneurysms were induced in the common carotid artery (CCA) of wild-type Hp1-1 mice (n=31) and transgenic Hp2-2 mice (n=30) using elastase to degrade the arterial wall of the CCA and angiotensin II to induce hypertension. There were four experimental groups: (1) sham surgery (n=11); (2) angiotensin II only (n=10); (3) elastase only (n=20); and (4) elastase + angiotensin II (n=20). Aneurysm size was determined by measuring both the outer circumference and luminal circumference of the blood vessel. Cross sections of the CCA were acquired using a computerized image analysis system; the outer circumference and luminal circumference of the CCA were measured and averaged for each animal. Macrophages infiltrating the aneurysm wall were quantified immunohistochemically using a purified anti-mouse Mac-2 monoclonal antibody (CL8942AP, Cedarlane). Results were analyzed using a two-way ANOVA with a Bonferroni post-test.

RESULTS: Aneurysms in Hp-2-2 mice were significantly larger than those in Hp1-1 mice in the setting of vessel wall degradation with elastase and angiotensin II-induced hypertension (outer circumference 2658 ± 64 vs. 2467 ± 55 μm ± SEM, p=0.02, and luminal circumference 2118 ± 52 vs. 1921 ± 48 μm ± SEM, p=0.01). Furthermore, the number of macrophages infiltrating the aneurysm wall was significantly increased in Hp2-2 aneurysms as compared to Hp1-1 controls (43.3 ± 2.9 vs. 27.1 ± 2.1 macrophages ± SEM, p=0.0001).

CONCLUSIONS: In the presence of arterial wall injury and hypertension, aneurysmal infiltration with macrophages results in larger and potentially more dangerous aneurysms. These results suggests that inflammation and the Hp2-2 genotype may be involved in aneurysm formation and potential rupture.

SATURDAY, OCTOBER 20

12:17-12:27 A NEW METHOD FOR INTRAOPERATIVE FLUORESCENCE-GUIDED RESECTION OF HIGH-GRADE GLIOMAS

Aaron Cohen-Gadol, MD, MSc, *University of Indiana, Indianapolis, IN*

OBJECTIVE: Different fluorophores (fluorescent biomarkers) including 5-ALA have been recently examined for maximizing the extent of resection for high-grade gliomas. Since 5-ALA is not approved by FDA, regulatory barriers have limited its use. Herein, a new practical safe method for fluorescence-guided resection of such gliomas will be presented using an FDA approved agent (low-dose sodium fluorescein.)

METHODS: Following IRB approval, low-dose (300mg) sodium fluorescein was injected in 6 consecutive patients with presumed diagnosis of a high-grade glioma intravenously 10-20 minutes before resection of the tumor. A high definition filter (Yellow 560, Zeiss Meditec, Oberkochen, Germany) integrated onto the operating microscope was used to intensify and assess the degree of fluorescent signal between the tumor and normal surrounding brain. We conducted histopathological examination of the areas of maximal and minimal fluorescence to assess the authenticity of the fluorescent signal in demonstrating infiltrative glioma cells.

RESULTS: Upon injection of the fluorescein, the entire brain and vessels fluoresced immediately, however within minutes, the normal structures cleared fluorescein but all the tumors in all patients remained intensely stained with fluorescein and clearly demarcated from surrounding normal brain as confirmed by neuronavigation data. This low dose fluorescein fluorescence was not detectable by an unaided eye. Thirty histopathological sections were obtained randomly at tumor margins (defined as areas of major and minor fluorescence) among all tumors and assessed for presence (>50% vs <50% infiltrated) of glioma cells. For 26 sections, the degree of fluorescence corresponded correctly to the amount of tumor within the section. In four sections, although minor amount of fluorescence was present intraoperatively, more than 50% of each specimen contained viable tumor cells. Overall, presence of major fluorescence was approximately 100% sensitive and 90% specific for demonstrating tumors cells.
In one patient, the lack of fluorescence correctly confirmed the diagnosis of a non-neoplastic inflammatory lesion.
This method of fluorescence was easy to use and did not interfere with operating room workflow. Miniscule leakage of fluorescein in the blood and CSF within the surgical field did not interfere with tumor fluorescence. The fluorescent signal lasted for the entire intradural portion of each operation without any degradation in its intensity. No patient suffered from any adverse effect.

CONCLUSIONS: Intravenous low-dose fluorescein provides a readily available method for fluorescence-guided tumor resection. It can improve resection of gliomas with minimal risks. Further studies are necessary to establish the efficacy of this technique in affecting patients' survival.

Surgical videos of the above mentioned technique will be presented.

SATURDAY, OCTOBER 20

12:29-12:39 THE eCLIPS SELF EXPANDING ENDOLUMINAL DEVICE FOR THE TREATMENT OF BIFURCATION ANEURYSMS: PRELINIMARY ANIMAL MODEL STUDY RESULTS.

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BACKGROUND: The eCLIPs (Evasc Medical Systems Corp, Vancouver CA) is a self expanding endoluminal device designed to facilitate the treatment of wide neck bifurcation aneurysms and can function as either a flow diverter or coil retention device.

OBJECTIVE: To evaluate the delivery and technical use of the eCLIPs System and to examine the histology and pathology of the eCLIPs device in an animal model at 30 and 90 days follow up.

METHODS: 8 rabbits with surgically created bifurcation aneurysms were treated with the eCLIPs device and with detachable coils. Effectiveness of delivery and of treatment, progression of aneurysm occlusion and occurrence of complications were analyzed. Angiographic follow up was performed on all 8 animals at 30 days and 4 animals were sacrificed and tissues processed for scanning electron microscopy (SEM) and histology sections. Follow up at 90 days on the 4 remaining animals included angiography and evaluation of tissues with SEM and histology sections.

RESULTS: eCLIPs was successfully delivered and implanted across the necks of the bifurcation aneurysms in all 8 animals (100%). At implant the eCLIPs device demonstrated a reduction of contrast entering the aneurysm in all 8 animals (100%). Five devices were successfully crossed with a microcatheter and coils were placed within the aneurysms. Two aneurysms received 1 coil each and the remaining three animals received 4,6 and 12 coils respectively. All coils were successfully retained within the aneurysms by the eCLIPs devices at implant and at all follow-up evaluations. Three devices were left without coils to evaluate potential of using the eCLIPs device as a flow diverting therapy.

No complications occurred over the 30 day follow-up. No migration of devices was noted at 30 days. Follow up angiographs at 30 days show all vessels patent and demonstrated either reduced flow or occlusion of the aneurysms. SEM evaluations at 30 days on 3 eCLIPs devices demonstrated neointima formation with well-organized endothelium coverage of the device in the parent artery. Histology cross sections from one aneurysm indicated complete incorporation of the device with organized smooth muscle neointimal growth over the aneurysm neck. Incorporation of the device at the aneurysm neck and exclusion of the aneurysm was variable at 30 days ranging from complete to partial occlusion of the aneurysm necks. 90 day results were unavailable at the time of the abstract submission.

CONCLUSION: The eCLIPs device is a novel technology specifically designed to treat bifurcation aneurysms. The device, when used with coils, provides a scaffold for regrowth of the neointima over the neck of the aneurysm, an effective means of retaining coils and provides a modest degree of flow diversion away from the aneurysm. These studies suggest that the device is safe and effective in this model and recommends proceeding to initial human investigation.

SPECIAL GUESTS

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Franco DeMonte Houston, TX	Raymond Sawaya
Paul Gardner Philadelphia	Robert Friedlander
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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

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
The Fairmont Scottsdale Princess, Scottsdale, Arizona……..... October 19-22, 2011
The Chatham Bars Inn, Chatham, Massachusetts……. October 17-20, 2012

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
Robert A. Solomon…..2011
James T. Rutka…….. 2012

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
Jeffrey Bruce…….....2011
James Drake……...2012

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……..	2005-07
James Rutka…….. ..	2008-10
Mitchel S. Berger…….. ..	2011-

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	1996-98
L. Nelson Hopkins……..	1999-01
Ralph G. Dacey, Jr…….	2002-04
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Griffith Harsh…….	2008-10
Daniel Barrow……	2011-

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949-494-3422, fax 949-494-8947, eldonfoltz@gmail.com

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

ALLAN FRIEDMAN (Elizabeth Bullitt).....1994
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Box 3807
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919-684-3271, fax 919-681-7973, fried010@mc.duke.edu

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323-226-7421, fax 323-226-7833, giannott@usc.edu

PHILIP GORDY.....1968
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ROBERT GROSSMAN (Ellin) 1984
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The Methodist Hospital
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713-441-3810, fax 713-793-1004, rgrossman@tmhs.org

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Department of Neurological Surgery, Box 8057
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314-362-3567, fax 314-362-2107, grubbr@nsurg.wustl.edu

JOSEPH HAHN (Andrea)……1993
Neurosurgery/H18
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216-444-5802, fax 216-445-7100, hahnj@ccf.org

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

HAYNES LOUIS HARKEY, III (Alison).....2002
Department of Neurosurgery
Univ of Mississippi Medical Center
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DECEASED MEMBERS

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

JEAN BRIHAYE 1975 1999
Bruxelles, BELGIUM
(Senior Corresponding)

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

HOWARD BROWN 1939 1990
San Francisco, California
(Senior)

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

JUAN CARDENAS..... 1966 1996
Mexico City, MEXICO
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HARVEY CHENAULT……1949….....…….2006
Lexington, Kentucky
(Senior)

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

JUAN CARLOS CHRISTENSEN….1970 2003
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GALE CLARK..... 1970 ..... .1996
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(Senior)

WINCHELL McK. CRAIG…..1942 1960
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JAMES GALBRAITH..... 1947 1997
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HENRY GARRETSON…… 1973……..……2007
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Washington, D. C.
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